

Sudden sensorineural hearing loss as a vascular event: labyrinthine infarction, stroke risk, and the limits of the idiopathic label

Abstract

Most cases of sudden sensorineural hearing loss are classified as idiopathic, and the classification has consequences: it directs treatment towards corticosteroids, closes the diagnostic question, and returns the patient to primary care with a hearing problem rather than a vascular one. The anatomy invites scrutiny of that position. The labyrinthine artery is functionally an end artery, arising in most people from the anterior inferior cerebellar artery, supplying tissue with a high metabolic demand and limited collateral reserve, so a lesion below the resolution of routine imaging may produce permanent cochlear damage. Sudden deafness is a documented manifestation of anterior inferior cerebellar artery infarction, and in a minority of patients in small series it preceded the brainstem event by days to weeks. The epidemiological literature linking sudden hearing loss to subsequent stroke is more heterogeneous than it is sometimes presented to be: positive cohort studies derive largely from Asian administrative datasets restricted to hospitalised patients, whereas a Dutch primary care cohort found no significant excess risk overall. This narrative review examines the anatomical, clinical and epidemiological strands separately and argues that they support a modest and probably age-dependent association rather than a demonstrated causal relationship. We draw particular attention to a problem of reverse inference: evidence derived from patients with established infarction cannot be inverted into a diagnostic rule for the much larger and more heterogeneous population presenting with sudden hearing loss. We propose, explicitly as an expert-opinion framework rather than a validated prediction rule, a tiered set of features that may warrant emergency stroke assessment, vascular imaging, or routine vascular risk assessment. Current guidance does recognise focal neurological findings and non-idiopathic causes including stroke; the more specific gap we identify is the absence of a structured pathway for evaluating sudden hearing loss as a possible isolated manifestation or marker of posterior circulation ischaemia.

Keywords: sudden sensorineural hearing loss, labyrinthine infarction, anterior inferior cerebellar artery, posterior circulation stroke, internal auditory artery, sudden deafness

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Abbreviations: AICA, anterior inferior cerebellar artery; CT, computed tomography; DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery; iSSNHL, idiopathic sudden sensorineural hearing loss; MRI, magnetic resonance imaging; SSNHL, sudden sensorineural hearing loss; TIA, transient ischaemic attack; vHIT, video head impulse test.

Introduction

Sudden sensorineural hearing loss is conventionally defined as a loss of thirty decibels or more across at least three contiguous frequencies developing over seventy-two hours or less.¹ Reported annual incidence varies widely between series, in the region of five to twenty-seven cases per hundred thousand, and most authors who quote a figure add that the true incidence is probably higher, because patients whose hearing recovers quickly may never present.^{1,2} A small proportion of cases are attributable to retrocochlear pathology, a further minority to identifiable causes such as Ménière disease, autoimmune disease, infection or trauma, and the remainder — the large majority — are labelled idiopathic.¹

That label is a statement about the limits of investigation rather than about the underlying biology. Vascular occlusion of the cochlear supply has been proposed as the mechanism for at least a subset of these cases since the middle of the last century. It has not been established,

in large part because it is difficult to establish: no imaging modality in routine clinical use resolves the labyrinthine microcirculation, and histological material is rarely available at a useful interval after the event.

What has accumulated is indirect evidence from three separate directions — vascular anatomy, the clinical phenomenology of anterior inferior cerebellar artery infarction, and population-level cohort studies of stroke risk. Our purpose is to examine each strand on its own merits, because they are sometimes bundled together in a way that overstates the collective case, and to set out carefully what does and does not follow for practice. We should state at the outset that the clinical framework we propose in Section 8 is an expert-opinion synthesis. It has not been prospectively validated, and it should not be read as a diagnostic or prognostic rule.

Methods

This is a narrative review. It was not conducted as a systematic review: no PRISMA-compliant screening, duplicate independent selection, formal risk-of-bias appraisal or quantitative pooling was undertaken, and the evidence selected necessarily reflects the authors' judgement of clinical relevance. We make comparative judgements between bodies of evidence in what follows, and readers should weigh those judgements in the light of this non-systematic selection process, which we list among the limitations in Section 11.

PubMed/MEDLINE, Embase and the Cochrane Library were searched from inception to July 2026, supplemented by Google Scholar for forward and backward citation tracking. The major search concepts were combined in various permutations: sudden sensorineural hearing loss, sudden deafness, labyrinthine infarction, internal auditory artery, anterior inferior cerebellar artery, posterior circulation stroke, vertebrobasilar ischaemia, cochlear ischaemia, stroke risk, and vestibular function testing. The reference lists of all retrieved articles, of relevant clinical practice guidelines, and of previous reviews on the vascular hypothesis were hand-searched.

Sources were selected for inclusion if they reported: original cohort or case-control data on the association between sudden hearing loss and cerebrovascular disease; clinical series of audiovestibular dysfunction in angiographically or radiologically confirmed posterior circulation infarction; anatomical studies of the labyrinthine artery; systematic reviews or meta-analyses on any of these questions; or current clinical practice guidelines. Where multiple studies addressed the same question, priority was given to the most recent evidence synthesis, to prospective over retrospective designs, and to studies whose case definition of sudden hearing loss was clinical rather than derived from administrative coding. Case reports were included only where they documented a mechanism or a clinical sequence not otherwise represented in the literature, and are identified as such in the text. Non-English-language reports were excluded, which we acknowledge as a limitation.

The anatomy of the inner ear blood supply

The inner ear is supplied by the labyrinthine artery, also called the internal auditory artery, which divides into the common cochlear artery and the anterior vestibular artery and thence into vessels supplying the cochlea, the utricle and the semicircular canals.

Its origin is variable, and the frequently quoted figure of approximately eighty-five per cent origin from the anterior inferior cerebellar artery is less secure than its repetition suggests. A systematic review and meta-analysis of thirty-three anatomical studies comprising 3,778 arteries found the anterior inferior cerebellar artery to be the origin in 75.4% of cases (95% confidence interval 62.7–86.4), with the remainder arising from the basilar artery, from a common trunk shared with the anterior inferior cerebellar artery, or occasionally from the superior or posterior inferior cerebellar arteries; the proportion varied between populations.³ A recent cadaveric microdissection series reported origin from the anterior inferior cerebellar artery in 65.6%, from a shared common trunk in 21.1% and directly from the basilar artery in 13.3%, together with considerable variability in number and course.⁴ The clinically relevant point is not a precise percentage but that the labyrinthine supply arises, in the great majority of individuals, from a vessel that also supplies the lateral pons and middle cerebellar peduncle.

Two further properties matter. The labyrinthine artery has limited collateral supply, so occlusion tends to produce infarction rather than the graded ischaemia that better-collateralised territories tolerate. And the cochlea has a high metabolic requirement, driven by the ion transport needed to maintain the endocochlear potential, with hair cells correspondingly intolerant of interrupted perfusion.

The volume of tissue involved is very small, and here the clinically important statement requires care. It is not that labyrinthine infarction is invisible on MRI in any absolute sense; dedicated inner ear sequences, including contrast-enhanced three-dimensional fluid-attenuated inversion recovery, can demonstrate labyrinthine signal change in some patients, and this is an active area of investigation.⁵

The accurate statement is narrower: routine brain MRI, including standard diffusion-weighted imaging, may fail to demonstrate an isolated labyrinthine vascular lesion, and a normal study of that kind therefore does not exclude a vascular cause. It excludes a vestibular schwannoma and a brainstem or cerebellar infarct of appreciable size. It provides limited information about the cochlear supply.

The same anatomy has been invoked to explain a clinical asymmetry. Viral inner ear disease tends to produce isolated deficits — vestibular neuritis without hearing loss, or sudden deafness without vertigo — whereas a vascular lesion of the labyrinthine artery would not be expected to respect the distinction between cochlear and vestibular divisions, since both share a supply. Combined simultaneous loss of auditory and vestibular function has accordingly been proposed as more suggestive of a vascular mechanism.^{6,7} We would note that this reasoning is based on observed patterns in confirmed infarction rather than on prospective discrimination in undifferentiated patients.

Sudden deafness in anterior inferior cerebellar artery infarction

The clearest clinical evidence comes from series of patients with confirmed infarction. In an early study of twelve patients with anterior inferior cerebellar artery territory infarction, the middle cerebellar peduncle was the most commonly affected structure, involved in eleven; audiological testing confirmed sensorineural hearing loss in eleven patients, with a predominantly cochlear pattern in six, retrocochlear in one and combined in four; and electronystagmography showed absent caloric responses in ten.⁸ A subsequent prospective series of eighty-two consecutive patients with anterior inferior cerebellar artery territory infarction found that eighty of eighty-two had acute prolonged vertigo with vestibular dysfunction of peripheral, central or combined origin, and concluded that labyrinthine dysfunction of vascular cause characteristically produces combined auditory and vestibular loss, unlike a viral cause.⁷ These observations invert the common clinical reflex in which hearing loss is taken as evidence against a central cause.

Cases have been described in which sudden deafness with vertigo was the sole manifestation of anterior inferior cerebellar artery infarction, without any brainstem or cerebellar sign detectable on examination.⁹ Others have documented sudden deafness as a manifestation of stroke with an initially normal diffusion-weighted MRI,¹⁰ isolated labyrinthine infarction preceding a subsequent visible infarct in the same territory,¹¹ bilateral sudden deafness as a prodrome of anterior inferior cerebellar artery infarction,¹² and sudden hearing loss with vertigo following cerebellar hypoperfusion from vertebral artery dissection in the absence of established infarction.¹³ These are case reports and small series, and they establish that such presentations occur; they do not establish how often.

Taken together, these observations describe a spectrum. At one end is the patient with obvious brainstem signs in whom the hearing loss is a secondary feature. At the other is the patient with isolated sudden deafness, a normal routine scan and no neurological signs, who is indistinguishable at presentation from idiopathic sudden sensorineural hearing loss and who will be managed as such. How large that second group is remains unknown.

Prodromal audiovestibular symptoms, and the problem of reverse inference

Among the most frequently cited observations in this literature is one concerning timing. In the twelve-patient series described above,

four patients experienced vertigo, hearing loss or tinnitus as an isolated manifestation between one day and two months before the infarct declared itself, the episodes often lasting minutes.⁸ Similar antecedent audiovestibular disturbance has been reported in subsequent work from the same group.⁶

This observation should be handled carefully, and we would place two qualifications on it. First, four of twelve is a small numerator over a small denominator; the resulting proportion is highly imprecise and should not be treated as a stable estimate of the frequency of prodromal symptoms before anterior inferior cerebellar artery infarction. Second, and more fundamentally, the cohort consisted of patients selected because they had already sustained an infarct.

That second point deserves to be stated explicitly, because it is the principal methodological weakness in the case for a vascular evaluation pathway. Observing that a proportion of patients with confirmed posterior circulation infarction reported preceding hearing loss, tinnitus or vertigo tells us about the antecedents of stroke in a stroke population. It does not tell us what proportion of patients presenting with those same symptoms will go on to have an infarct. The two quantities are different, and the second — which is the one a clinician facing an undifferentiated patient actually needs — depends on the prevalence of posterior circulation ischaemia among all patients with such symptoms, which is not known. Reversing the inference without that denominator would substantially overestimate risk.

An analogy is often drawn here, and it is worth examining rather than simply deploying. Transient monocular visual loss prompts urgent vascular assessment because the ophthalmic artery is a branch of the internal carotid; by the same anatomical logic, transient audiovestibular symptoms might be expected to warrant similar attention, the labyrinthine artery being a branch of the anterior inferior cerebellar artery. Population-based work has established that transient isolated brainstem symptoms frequently precede posterior circulation stroke and carry substantial short-term risk.¹⁴ The anatomical parallel is genuine. What is missing, and what distinguishes the two situations in practice, is that the predictive value of transient monocular visual loss has been quantified in prospective cohorts, whereas the predictive value of isolated transient audiovestibular symptoms in unselected patients has not. The difference in current practice reflects that difference in evidence, not merely inherited convention. Establishing the missing quantity is, in our view, the single most useful piece of research the field could undertake.

The epidemiological evidence

The study most often cited in support of a vascular mechanism examined 1,423 patients hospitalised with sudden hearing loss in Taiwan against 5,692 patients who had undergone appendectomy, followed for five years.¹⁵ Stroke occurred in 12.7 per cent of the hearing loss cohort and 7.8 per cent of controls, giving an adjusted hazard ratio of 1.64 (95% confidence interval 1.31 to 2.07). The authors concluded that sudden hearing loss may be an early warning sign of impending stroke.

Several subsequent cohorts, predominantly Korean and Taiwanese, reported associations in the same direction,^{16,17} and meta-analyses have summarised the pooled adjusted risk at around 1.3 to 1.4.^{18,19} At this point the association is sometimes treated as settled.

It is not. A cohort study drawn from eighty-four Dutch general practices, comprising 480 patients aged fifty and over with idiopathic sudden sensorineural hearing loss matched 1:4 to 1,911 controls, found a hazard ratio of 1.25 (95% CI 0.50 to 3.27) for cerebrovascular

accident alone and 0.92 (95% CI 0.50 to 1.71) for cerebrovascular accident and transient ischaemic attack combined.²⁰ Neither estimate approached significance, and both confidence intervals are wide.

The discrepancy is explicable, and the explanation is instructive. The positive studies drew on national insurance databases and included only patients admitted to hospital with sudden hearing loss. Admission for this condition selects for severity, for comorbidity and for the kind of patient in whom clinicians are already concerned — which is to say, for vascular risk. Those patients then enter a period of intensified medical contact during which any cerebrovascular event is more likely to be detected and coded. A general practice cohort, capturing a fuller spectrum including patients whose hearing recovers rapidly, contains a materially different population. The case definition also differs: administrative coding of sudden hearing loss is less reliable than a clinical diagnosis made at the point of presentation.

The Dutch investigators did observe a higher hazard among patients aged sixty and over. We would urge caution in how this is read. The relevant subgroup estimate is imprecise, derived from a modest number of events in a post hoc analysis, and age sixty should not be presented as a validated clinical decision threshold. What the finding is consistent with — rather than what it demonstrates — is a vascular contribution concentrated among patients who already have vascular disease.

The parsimonious interpretation, and the one we favour, is that sudden sensorineural hearing loss and stroke share risk factors — hypertension, diabetes, dyslipidaemia, smoking, atrial fibrillation — and that in some older patients with those risk factors the hearing loss may represent a genuine ischaemic event. That is a considerably more modest claim than the proposition that sudden deafness predicts stroke, but it is the claim the data support.

The association has also been reported in the opposite direction. A Taiwanese cohort of 11,115 stroke patients and 33,345 matched controls found an adjusted hazard ratio of 1.71 for subsequent sudden hearing loss, rising to 5.65 within the first year.²¹ A bidirectional association of this kind is more readily explained by shared vasculopathy and shared ascertainment than by either condition causing the other.

Current guidance and the gap we identify

The 2019 update to the American Academy of Otolaryngology–Head and Neck Surgery clinical practice guideline on sudden hearing loss is a carefully constructed document.¹ It distinguishes conductive from sensorineural loss, requires audiometry within fourteen days, recommends strongly against routine head computed tomography in the initial evaluation, recommends strongly against routine laboratory testing, and recommends strongly against the routine use of antivirals, thrombolytics, vasodilators or vasoactive substances. Retrocochlear pathology is to be excluded by MRI, auditory brainstem response or audiometric follow-up.

Each of these positions is defensible on the evidence considered. Routine head computed tomography offers little, since it provides no high-resolution view of the internal auditory canal and rarely alters early management. Routine laboratory screening has a low yield. Trials of vasoactive agents were not encouraging.

It would be inaccurate to say that the guideline neglects the brain. It explicitly separates idiopathic from non-idiopathic sudden hearing loss, and it identifies focal neurological findings as indicating a cause requiring separate evaluation, of which stroke is one. The gap we wish to identify is narrower and, we think, more useful to state precisely. Current guidance does not set out a structured pathway for evaluating

sudden sensorineural hearing loss as a possible isolated manifestation or marker of posterior circulation ischaemia — that is, for the patient in whom the hearing loss is not accompanied by an obvious focal deficit but in whom a vascular mechanism may nonetheless be operating. The guideline is organised around the question of what improves hearing outcome. The question of what the episode implies for cerebrovascular risk is a different one, and it is not addressed.

One consequence is worth drawing out. The strong recommendation against thrombolytics and vasodilators concerns hearing recovery. It is not a statement about secondary prevention in a patient in whom a cerebrovascular event has been diagnosed on independent grounds, and it should not be read as one.

A proposed framework for identifying patients who warrant vascular evaluation

We are not proposing that sudden sensorineural hearing loss be reclassified as a cerebrovascular disorder. Most of it is not, and the

harms of overinvestigation — unnecessary antithrombotic exposure, incidental findings, anxiety and cost — are real.

(Table 1) sets out features that may warrant vascular evaluation, stratified by the level of clinical urgency and by the strength of the supporting evidence. We wish to be explicit about its status. **This is a proposed clinical framework derived from expert opinion and from the observational literature reviewed above. It is not a validated prediction rule.** None of the features listed has been prospectively evaluated for sensitivity, specificity or predictive value in an unselected population presenting with sudden hearing loss. The tiers should be read as a structured way of organising clinical suspicion, not as thresholds with established operating characteristics. Features in Tier A derive from findings that are recognised in existing guidance as indicating a non-idiopathic cause. Features in Tier B rest largely on case reports and small series. Tier C reflects general cardiovascular risk management that would be appropriate irrespective of the mechanism of the hearing loss.

Table 1 Proposed framework for identifying patients with sudden sensorineural hearing loss who may warrant evaluation for posterior circulation ischaemia

Tier	Clinical feature	Rationale and evidence base	Proposed action
A. Features warranting emergency stroke assessment	Accompanying vertigo with nystagmus showing central features (direction-changing, pure vertical or pure torsional), or a normal head impulse test in a patient with an acute vestibular syndrome	Raises concern that the lesion may lie central to the vestibular nerve rather than within the labyrinth. Supported by bedside diagnostic accuracy studies in acute vestibular syndrome, although these were not conducted in SSNHL populations specifically; the pattern increases suspicion of a central cause but does not by itself establish one, and should be interpreted alongside the wider clinical assessment	Urgent MRI with diffusion-weighted sequences; same-day discussion with stroke services
	Any brainstem or cerebellar sign, however slight — facial numbness, dysmetria, Horner syndrome, gaze palsy, dysarthria, limb ataxia	AICA territory infarction commonly involves the middle cerebellar peduncle and lateral pons. Recognised in existing SSNHL guidance as indicating a non-idiopathic cause	Manage as acute stroke; urgent imaging and admission
B. Features warranting vascular imaging or specialist neurological evaluation	Recent neck pain, headache or trauma preceding the hearing loss	Raises the possibility of vertebral artery dissection, which has been reported to produce hearing loss through hypoperfusion without established infarction. Case-level evidence only	MRI with vessel imaging; avoid vigorous head impulse testing and cervical manipulation pending results
	Bilateral simultaneous sudden hearing loss	Rarely idiopathic; described in case reports as a prodrome of AICA or basilar territory ischaemia, and also seen in autoimmune, infective and neoplastic disease	MRI including vessel imaging; broaden the differential substantially
	Preceding transient episodes of vertigo, tinnitus or hearing disturbance in the preceding weeks	Observed in a minority of patients in small AICA infarction series, but the predictive value of these symptoms in unselected patients presenting with SSNHL has not been established prospectively (see Section 5)	Prompt neurological assessment and vascular risk evaluation. Whether such patients should be managed as having had a posterior circulation transient ischaemic attack is at present a matter of clinical judgement rather than established practice
C. Features supporting routine vascular risk assessment	Older age together with hypertension, diabetes, atrial fibrillation, smoking or established vascular disease	The epidemiological association between SSNHL and subsequent stroke appears most consistent in older patients, but the relevant subgroup estimates are imprecise and no validated age threshold exists	Assessment and management of modifiable vascular risk factors in the usual way, whether or not hearing recovers. This is good general practice and does not depend on accepting a vascular mechanism for the hearing loss

This is an expert-opinion framework, not a validated prediction rule; see Section 8 and the limitations in Section 11.

Two entries deserve expansion. The first is the accompanying vestibular examination. Where sudden hearing loss occurs with vertigo, the bedside oculomotor findings carry the significance they carry in any acute vestibular syndrome, and a normal head impulse test alongside deafness is a combination that warrants attention.²² Video head impulse testing has been reported to show differing patterns of semicircular canal involvement between sudden hearing loss with vertigo and vestibular neuritis, with posterior canal impairment predominating in the former and horizontal canal impairment in the latter.²³ It should be said clearly that this comparison was between two clinically defined groups and does not establish that the test distinguishes vascular from inflammatory sudden hearing loss; whether it can do so is among the open questions listed in Section 10.

The second is the preceding history, which is easily lost. Patients do not spontaneously report brief episodes of tinnitus or unsteadiness weeks earlier, because they do not connect them to the present complaint. The question has to be asked directly, and about the preceding two months rather than the preceding few days. Eliciting such a history should prompt neurological assessment; as set out in Section 5, whether it should trigger the full pathway applied to a transient ischaemic attack is not something the current evidence settles.

Investigation and management

Imaging

Where a vascular cause is being considered, the appropriate study is MRI of the brain with diffusion-weighted sequences together with fine slices through the internal auditory meatus, rather than routine head computed tomography. Dedicated inner ear sequences, including contrast-enhanced three-dimensional fluid-attenuated inversion recovery, may demonstrate labyrinthine signal change, although the specificity of this finding is not established and comparable changes occur in inflammatory and haemorrhagic conditions.⁵

Vessel imaging should be added where dissection is a possibility, where there are brainstem signs, or where the history suggests preceding transient events. Beyond imaging, the investigation is that of any suspected posterior circulation event: blood pressure, glycaemic status, lipids, and rhythm assessment adequate to detect paroxysmal atrial fibrillation, which means prolonged monitoring rather than a single electrocardiogram where suspicion is substantial.

The caveat from Section 3 applies here too: a normal routine MRI does not exclude labyrinthine infarction, and a clinician who ordered the scan because of concerning clinical features should not treat a normal report as settling the question.

The therapeutic question

Corticosteroids, systemic or intratympanic, remain the mainstay of treatment for idiopathic sudden sensorineural hearing loss. The evidence is imperfect, but the treatment is time-critical and the argument for early administration is reasonable.

If the mechanism in a given patient is ischaemic, steroids do not address it. The interventions that would alter that patient's cerebrovascular trajectory are antithrombotic therapy, blood pressure control and investigation for an embolic source. No trial has addressed the combined management of such patients, and none is likely to, because the population cannot at present be identified prospectively with confidence.

It is important to separate two things that are easily conflated. Treating the hearing loss according to established guidance while *urgently evaluating* for suspected cerebrovascular disease is reasonable, carries little additional risk, and is what we would advocate where the features in Table 1 are present. Initiating stroke-specific *secondary prevention* is a different matter. Antithrombotic therapy requires a level of diagnostic confidence that the evidence reviewed here does not supply in the isolated case, and it carries bleeding risk and contraindications that must be assessed individually. That decision belongs with a clinician who has assessed the patient for a cerebrovascular diagnosis on its own merits — typically a stroke physician or neurologist — and should not follow automatically from the presence of sudden hearing loss, however suggestive the accompanying features.

Tier C in Table 1 is deliberately different in kind. Assessing and managing modifiable vascular risk factors in an older patient with hypertension or diabetes is appropriate general practice and does not require accepting any particular mechanism for the hearing loss.

The counterargument should be stated fairly. Over-attributing sudden deafness to vascular disease risks a distinct set of errors: unnecessary antiplatelet therapy in patients at bleeding risk, diagnostic anchoring that delays recognition of autoimmune or neoplastic causes, and medicalisation of a condition that in many patients resolves.

Research priorities

The most valuable study would be a prospective cohort of unselected patients with sudden sensorineural hearing loss, recruited from both primary and secondary care to avoid the severity bias that distorts the existing literature, undergoing protocolised MRI with dedicated inner ear sequences, vestibular function testing and prolonged cardiac monitoring, with follow-up for cerebrovascular events over at least five years. Such a study would establish what proportion of apparently idiopathic cases have a demonstrable vascular substrate, and would supply the denominator missing from the reverse inference described in Section 5.

Four narrower questions are tractable. Whether dedicated inner ear MRI sequences can identify labyrinthine ischaemia with adequate specificity. Whether vestibular test patterns, including video head impulse testing, can separate vascular from inflammatory disease rather than merely separating clinically defined syndromes. What the short-term stroke risk actually is among patients presenting with isolated transient audiovestibular symptoms — a question that could be approached through prospective registry data. And whether the tiered framework proposed in Table 1 performs better than clinical judgement alone, which would require prospective evaluation before it could be recommended for adoption.

Limitations

Several limitations constrain the conclusions of this review.

The evidence selection was narrative and non-systematic. No duplicate independent screening, formal risk-of-bias assessment or quantitative synthesis was performed, and relevant studies may have been missed; the exclusion of non-English-language reports may have introduced further selection bias.

Several of the phenotypic observations on which the argument rests derive from small clinical series and individual case reports, often from a small number of investigative groups, with the attendant risks of publication bias and of unstable frequency estimates.

The epidemiological literature is heterogeneous in case definition, population, healthcare setting and analytical approach, and the administrative cohort studies that dominate it are subject to selection, severity and surveillance biases that cannot be fully adjusted for.

Imaging cannot at present reliably demonstrate isolated labyrinthine infarction in routine practice, so the central pathological claim is supported by inference rather than by direct visualisation in most patients.

There are no validated diagnostic criteria for vascular sudden sensorineural hearing loss, and consequently no reference standard against which any proposed clinical feature could be evaluated.

Finally, the clinical pathway proposed in Table 1 has not been prospectively validated. Its sensitivity, specificity and effect on clinical outcomes are unknown, and it should be regarded as a hypothesis for evaluation rather than a recommendation for adoption.

Conclusion

The vascular hypothesis in sudden sensorineural hearing loss is neither established nor dismissible. The anatomy is suggestive: an end artery with limited collateral reserve supplying metabolically demanding tissue, arising in most people from a vessel that also supplies the lateral pons and middle cerebellar peduncle. The clinical evidence from infarction series shows that sudden deafness can be the presenting, and occasionally the only, manifestation of anterior inferior cerebellar artery ischaemia. The epidemiological evidence is the weakest strand, distorted by the selection effects inherent in hospital-based administrative datasets, and probably indicates a modest association concentrated among older patients with existing vascular disease.

What follows for practice is correspondingly modest. The idiopathic label should be understood as provisional. The history should extend back two months and should ask specifically about transient episodes. The assessment should include vestibular and oculomotor examination, not only an audiogram. Patients with focal neurological signs or central oculomotor findings require urgent stroke assessment. And in the older patient with vascular risk factors, an episode of sudden deafness is a reasonable occasion to review those risk factors, whether or not hearing recovers and whether or not the scan is normal — a step that is justifiable on general grounds and does not depend on resolving the mechanistic question.

What does not yet follow is a change in the treatment of the individual patient with isolated sudden hearing loss. Establishing whether such a change is warranted requires the prospective data that the field currently lacks.

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Ethical approval and informed consent

Not applicable. This is a narrative review of previously published literature. It involved no human participants, no patient-identifiable data and no animal subjects, and institutional review board approval and informed consent were therefore not required.

Author contributions

Recorded using the CRediT taxonomy. AD: Conceptualisation; writing — original draft; writing — review and editing; visualisation. SK, VK, GG: Conceptualisation; methodology; supervision; writing — review and editing; resources. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Data availability

No new data were generated or analysed in the preparation of this review. All sources cited are publicly available.

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