

Acute vertigo and sensorineural hearing loss from infarction of the vestibulocochlear nerve

A case report

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Abstract

Rationale: Acute unilateral audiovestibulopathy is a common neurotological syndrome. Differential diagnoses of acute unilateral audiovestibulopathy include viral infection, vascular insults, and tumors. Regarding vascular causes, ischemic stroke in the anterior inferior cerebellar artery (AICA) territory is known to be the leading cause of acute audiovestibular loss. Previous reports of AICA infarction with audiovestibulopathy failed to demonstrate magnetic resonance imaging (MRI)-positive vestibulocochlear infarctions. Only 1 report demonstrated acute infarction involving the vestibulocochlear nerve on diffusion weighted imaging (DWI)-MRI.

Patient concerns: A 67 year old man complained of sudden left hearing loss and vertigo. The patient showed left horizontal gaze-evoked nystagmus (GEN) and the head impulse test (HIT) was positive on the left side. Videonystagmography revealed spontaneous rebound nystagmus toward the right side; head-shaking nystagmus toward the right side. The patient presented with left caloric paresis (20.1%). Pure tone audiometry (PTA) revealed severe sensorineural hearing loss on the left side.

Diagnosis: MRI of temporal bone showed multifocal acute infarctions in the left inferior cerebellum. Moreover, images revealed tiny infarctions along the left vestibulocochlear nerve and the cochlea, implying acute vestibulocochlear nerve and labyrinthine infarction. There was no evidence of steno-occlusion of major cerebral vessels on MR angiography.

Interventions: Immediate stroke management was done.

Outcomes: Neurological symptoms gradually improved after 3 to 5 days.

Lessons: We present a case illustrating a rare but significant finding of vestibulocochlear nerve infarction revealed by DWI-MRI. Prompt imaging protocol enabled the detection of significant findings in this patient with acute unilateral audiovestibulopathy. Clinicians should be aware of the vestibulocochlear nerve and labyrinth on MRI in patients with cerebellar stroke.

Abbreviations: AICA = anterior inferior cerebellar artery, DWI = diffusion weighted imaging, EPI = echo planar imaging, FLAIR = fluid attenuated inversion recovery, GEN = gaze-evoked nystagmus, HIT = head impulse test, IAA = internal auditory artery, MRI = magnetic resonance imaging, PTA = pure tone audiometry.

Keywords: labyrinth diseases, stroke, vestibulocochlear nerve diseases

1. Introduction

Acute onset of peripheral vertigo and hearing loss is a common neurotological complaint. This clinical syndrome is thought to be

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The authors report no conflicts of interest.

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the result of an inflammatory process occurring in the inner ear, presumably with a viral etiology.^[1] However, acute ischemic stroke involving the internal auditory artery (IAA), a branch of the anterior inferior cerebellar artery (AICA), can also result in ischemic damage to the inner ear structures, leading to acute onset audiovestibulopathy.^[2,3] As there is no reliable imaging or diagnostic modality to distinguish labyrinthitis from labyrinthine infarction, diagnosing acute unilateral audiovestibulopathy accompanied by vertigo and unilateral deafness remains a diagnostic challenge.

Current advances in magnetic resonance imaging (MRI) techniques, including 3-dimensional (3D)-isotropic fluid attenuated inversion recovery (FLAIR) sequences, suggest a specific etiopathogenesis in patients with sudden sensorineural hearing loss and vertigo – vascular or inflammatory.^[4] However, routine diffusion weighted imaging (DWI) usually fails to detect small ischemic foci in the inner ear and the vestibulocochlear nerve due to insufficient spatial resolution.

Here, we present an interesting case of a patient with sudden audiovestibular loss, with DWI-MRI-depicted acute infarction involving the vestibulocochlear nerve and the inner ear.

2. Case report

A 67-year-old male patient was admitted to our otology clinic with acute spontaneous vertigo accompanied by left hearing loss

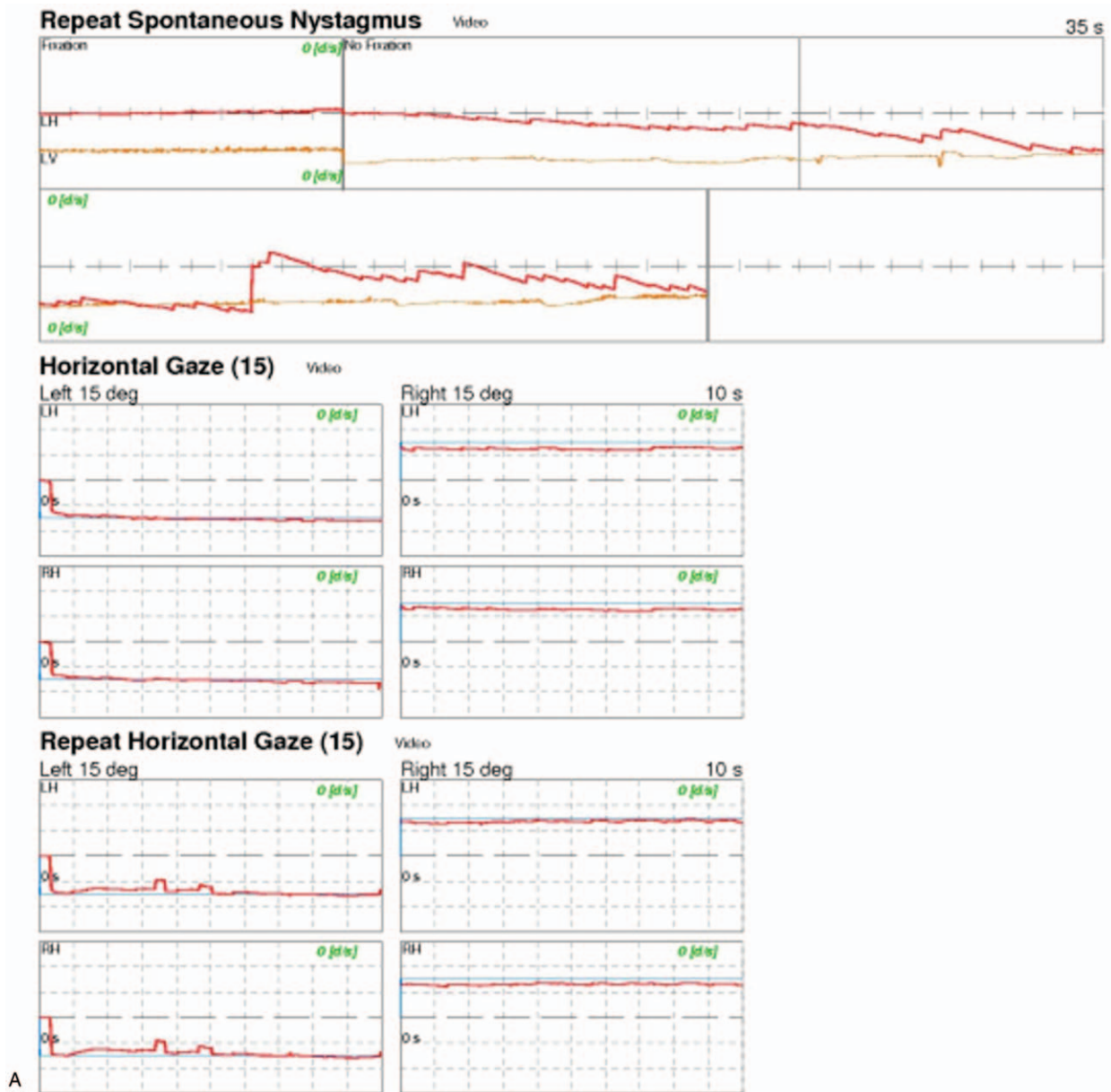


Figure 1. MRI and audiovestibular findings for a 67-year-old male patient with AICA territory infarction and acute left-sided audiovestibular loss. A. Videonystagmography examination presented spontaneous nystagmus beating and head shaking nystagmus toward the right side. B. HIT was positive on the left side. C. Bithermal caloric tests reveal left caloric paresis (20.1%). D. PTA shows severe sensorineural hearing loss on the left side. E. Subjective visual vertical and subjective visual horizontal are tilted counter-clockwise. F–H. Axial DWI – MRI demonstrates acute infarction in the left middle cerebellar peduncle (F, arrowhead) also involving the left vestibulocochlear nerve and cochlea (G, axial DWI and H, coronal DWI; arrows). AICA=anterior inferior cerebellar artery, DWI=diffusion weighted imaging, HIT=head impulse test, MRI=cmagnetic resonance imaging, PTA=pure tone audiometry.

and tinnitus. His medical history was irrelevant. The patient was able to stand unaided. Videonystagmography revealed spontaneous rebound nystagmus toward the right side (Grade II); head-shaking nystagmus toward the right side. The patient showed full ocular motility; subtle left horizontal gaze-evoked nystagmus (GEN) was noted (Fig. 1A). The HIT was positive on the left side (Fig. 1B). The patient presented with left caloric paresis (20.1%) (Fig. 1C) and bilateral sensorineural hearing loss with pure tone audiometry (PTA); severe sensorineural hearing loss on the left side (PTA: right, 37dB; left, 80 dB) (Fig. 1D). The subjective visual vertical and horizontal were tilted counter-clockwise

toward the left side (Fig. 1E). Vestibular evoked myogenic potentials revealed diminished response on the left.

Temporal bone MRI was performed to exclude structural lesions in the posterior fossa. Axial images of 1.4-mm-thick EPI (echo planar imaging) DWI [TR (repetition time)=10,000 ms, TE (echo time)=72 ms; matrix=128×128; field of view (FOV)=240 mm; b=0 and 1,000 s mm⁻²] showed multifocal hyperintensities in the left inferior cerebellum, suggesting acute infarction of AICA territory (Fig. 1F). Moreover, these images revealed multifocal infarctions along the left vestibulocochlear nerve and the cochlea, implying acute vestibulocochlear nerve

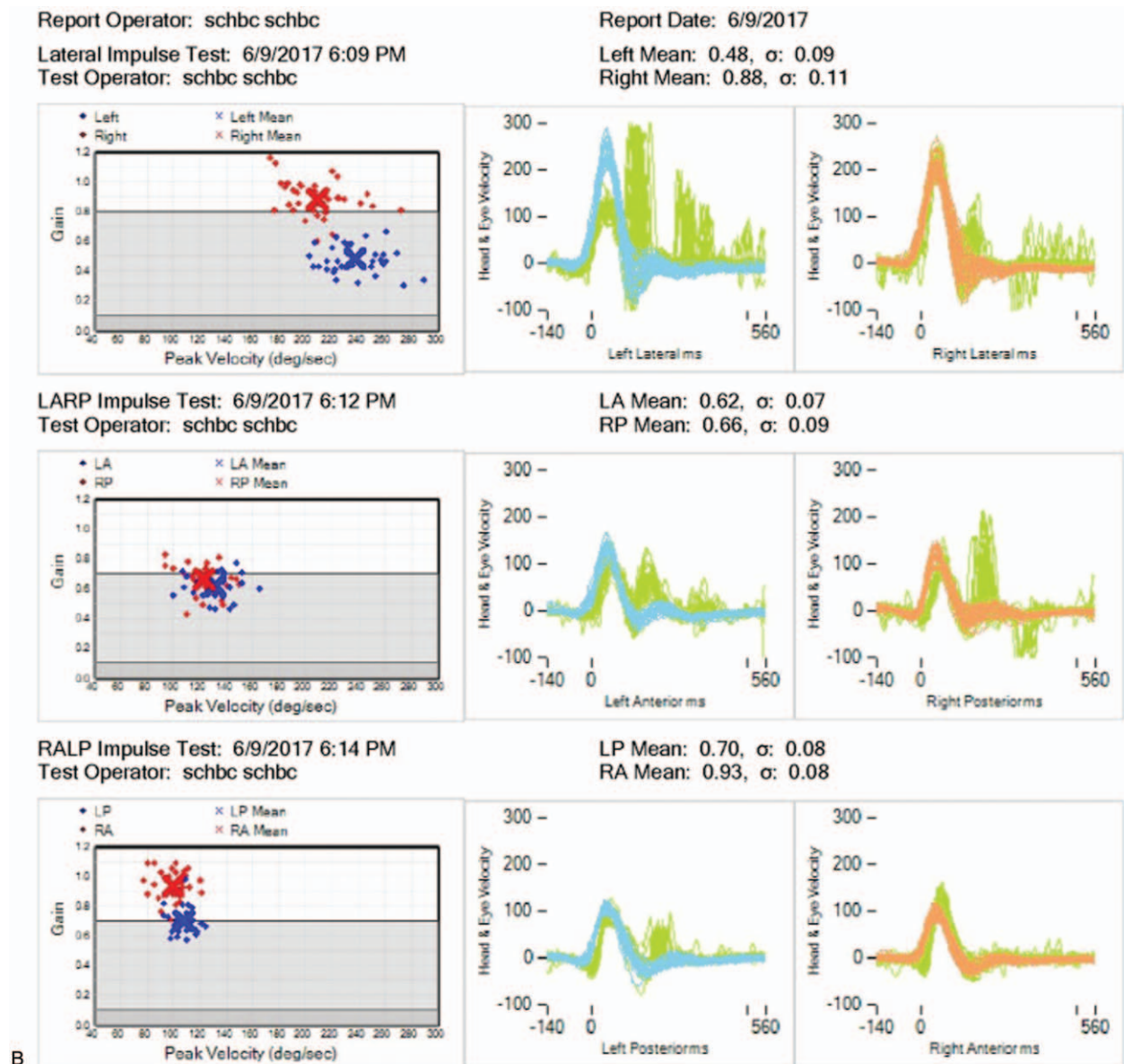


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and labyrinthine infarction (Fig. 1G, H). There was no abnormal hyperintensity on T1 images or T2 FLAIR (1-mm thickness) to suggest labyrinthine inflammation or hemorrhage. There was no evidence of steno-occlusion of major cerebral vessels on MR angiography.

Immediate acute stroke management including aspirin, clopidogrel were administered with an impression of the left AICA territory infarction. Neurological symptoms gradually improved after 3 to 5 days. The patient was discharged without any sequelae.

3. Discussion

Acute unilateral audiovestibulopathy is a common neurotological dysfunction. Many etiologies including infection, vascular insults, Ménière's disease, trauma, and tumors should be

included in the differential diagnosis.^[5] MRI is performed to patients with this clinical syndrome, to identify brain parenchymal lesions and retrocochlear abnormalities. However, the etiology remains unknown in the majority of cases. In previous studies, MRI using 3D FLAIR showed labyrinthine abnormalities in patients with acute audiovestibulopathy, by detecting the presence of methemoglobin and increased protein in the labyrinth.^[4] Regarding vascular causes, ischemic stroke in the AICA territory is known to be the leading. The inner ear structure and the vestibulocochlear nerve are supplied by the IAA arising from the AICA. Diseases involving IAA usually affect both the cochlea and the vestibular labyrinth, resulting in audiovestibulopathy. Thus, differentiating acute infarction from acute labyrinthitis is important to prevent the spread of infarction.^[3]

Patients with AICA infarction may present with both central and peripheral vestibulopathies.^[3] Negative HIT,

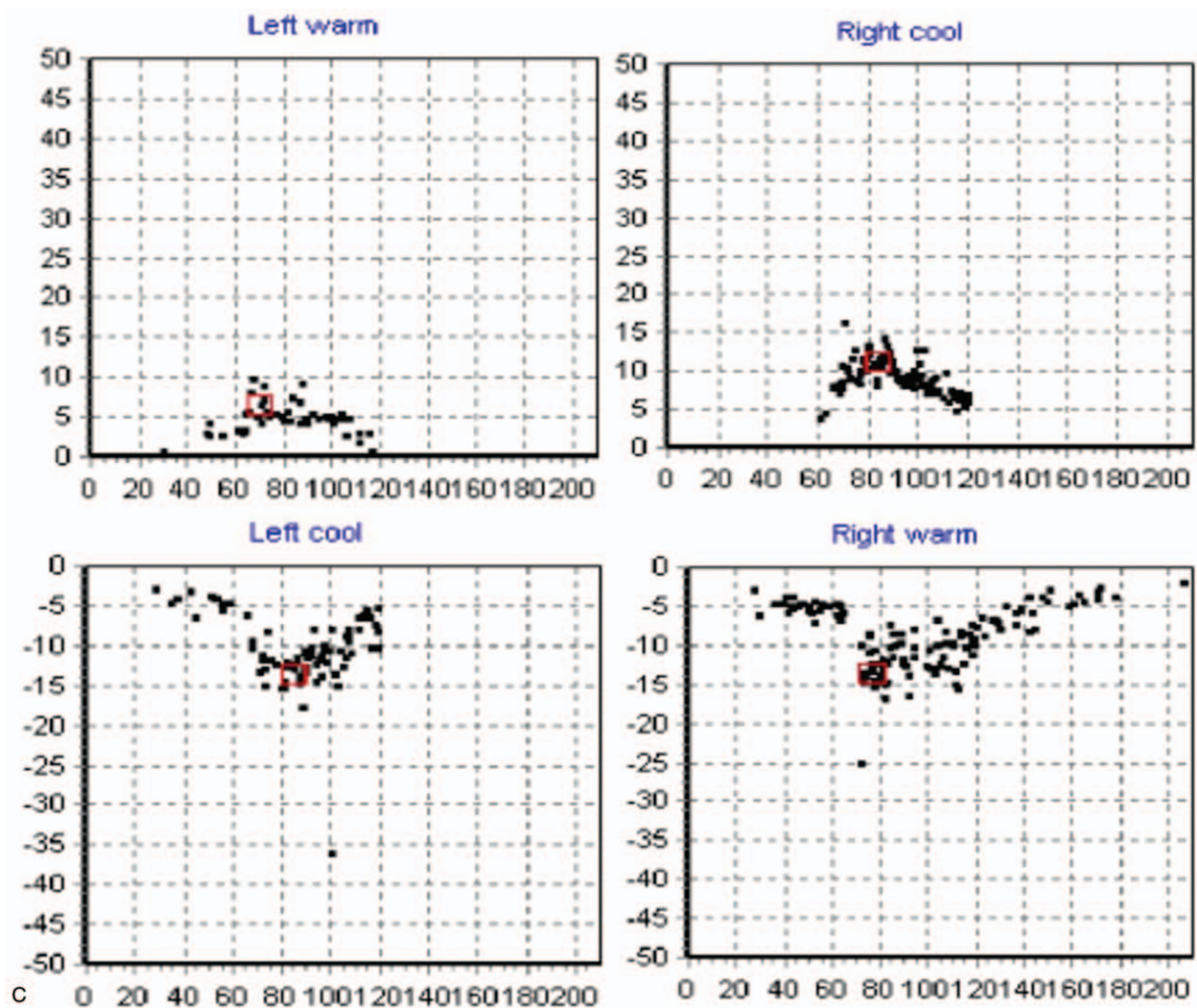


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direction-changing nystagmus, and skew deviation are known to be reliable signs for distinguishing stroke in patients with acute vertigo. However, diagnosis is challenging because peripheral signs often overshadow central signs. These signs may also be seen in patients with stroke involving vestibular nucleus or cerebellar flocculus.^[6] Given that clinical findings do not provide a conclusive diagnosis, infarction remains a presumptive diagnosis. Thus, radiologic findings of vestibulocochlear infarction have more value.

Generally, infarctions involving the brain parenchyma can be easily detected by routine DWI. However, conventional MRI may provide insufficient spatial resolution, where adequate resolution is crucial for depiction of cranial nerves. Specifically, EPI-DWI has limitations in the evaluation of the labyrinth due to its vulnerability to susceptibility artifacts arising from the adjacent temporal bone. Because delayed treatment of IAA infarction can result in permanent audiovestibular dysfunction, timely diagnosis is important. In this patient, thin-section DWI images allowed prompt and early diagnosis.

Previous reports of AICA territory infarction with audio-vestibulopathy^[2,7] failed to demonstrate MRI-positive vestibulo-

locochlear infarction. Only one report demonstrated acute infarction involving the vestibulocochlear nerve using DWI-MRI.^[8] In our case, thin-section MRI showed infarctions of the IAA territory. Also, negative thin-section T2 FLAIR images supported the diagnosis by excluding labyrinthitis. To our knowledge, our case is the second radiology report to visualize acute infarction of the vestibulocochlear nerve and labyrinth.

There are a few limitations in this case which should be discussed. Follow up MR imaging was performed with usual DWI protocol in 5 mm thickness, not in 1.4-mm-thickness. The imaging progress of the DWI high spots in the left vestibulocochlear nerve was not clearly visualized. However, repeated vestibular function tests revealed normalization of vestibular dysfunction which might reflect the resolution of the ischemic lesions. Second, direct visualization of IAA occlusion was not possible, since digital subtraction angiography was not performed in this patient. However, typical distribution of infarcted area which showed diffusion restriction in the AICA and IAA territory indirectly reflects the vascular occlusion of small distal branches.

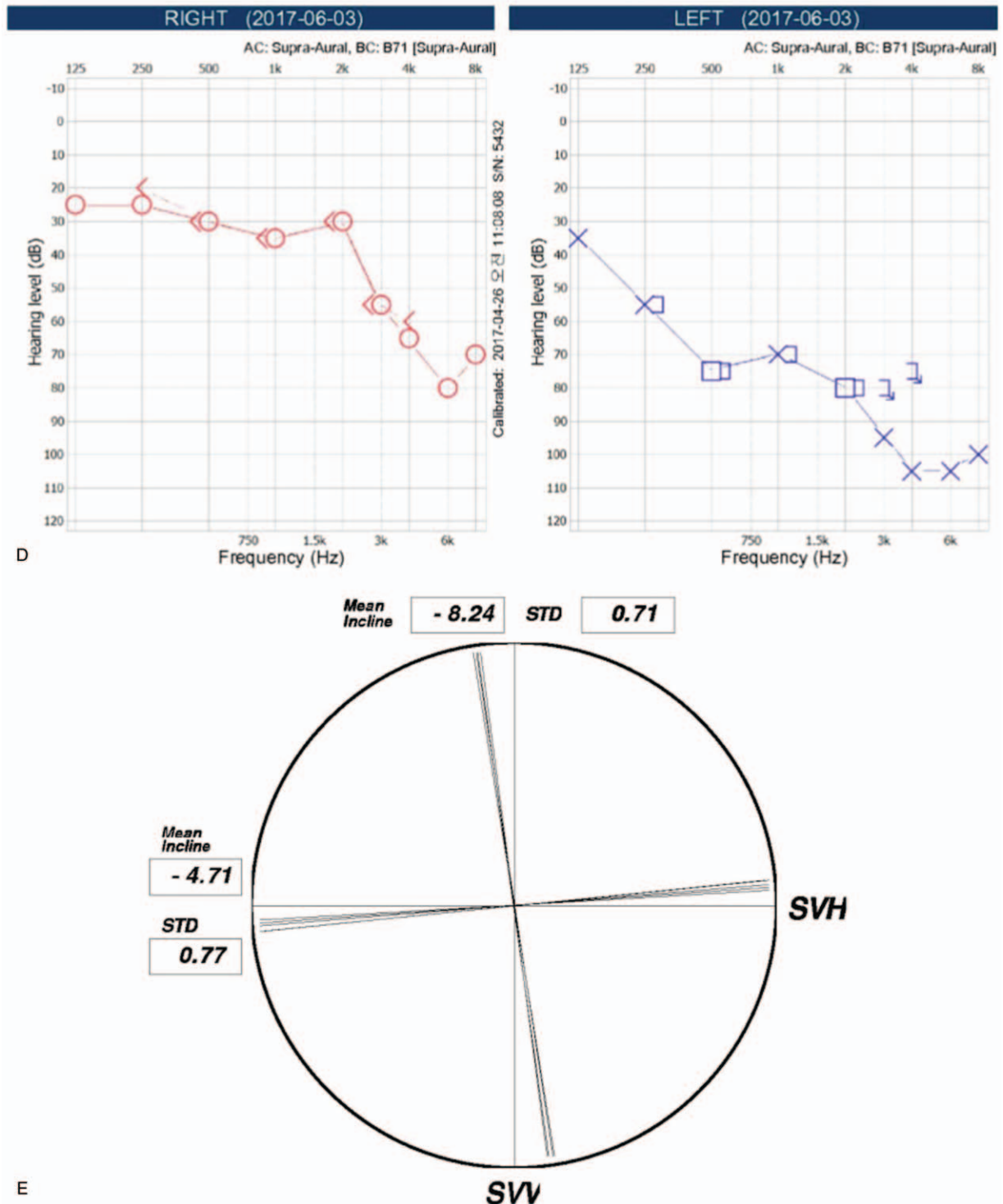


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Treatment for acute vestibulocochlear ischemia includes immediate anticoagulant therapy to reduce the autonomic symptoms associated with affected inner ear lesion.^[9] This prevents further progression of infarction, especially to other AICA regions. The long-term outcome for adequately treated infarction is good (81%), which is higher than the spontaneous recovery rate (40%–69%).^[10]

4. Conclusions

Vestibulocochlear nerve infarction revealed by DWI-MRI is rare, although it is clinically significant. Prompt MRI enables the detection of significant findings in patients with acute audio-vestibulopathy. Thus, MRI with thin-section DWI, focused on the temporal bone, should be performed in patients with

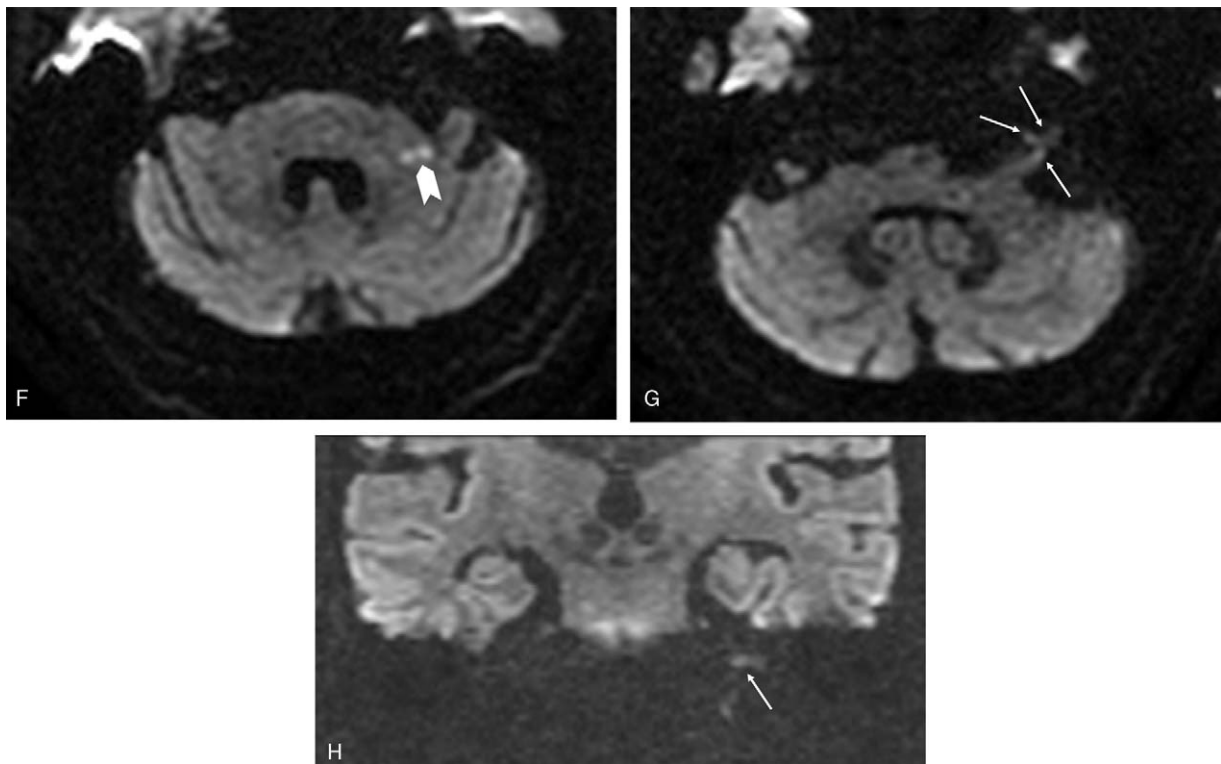


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unilateral audiovestibulopathy. Clinicians should be aware of the vestibulocochlear nerve and labyrinth on MRI in patients with cerebellar stroke.

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