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Post-contrast 3D-FLAIR in idiopathic sudden sensorineural hearing loss

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Abstract

Purpose Our study investigated correlations between clinical characteristics, particularly hearing recovery, interval time between onset and three-dimensional fluid attenuation inversion recovery magnetic resonance imaging (3D-FLAIR MRI), and the signal intensity of post-contrast 3D-FLAIR MRI in patients with idiopathic sudden sensorineural hearing loss (SSNHL).

Methods The study enrolled 100 SSNHL patients. The signal intensities and asymmetry ratios of the inner ear structures, including the cochleae, vestibules and vestibulocochlear nerve, were evaluated and calculated. The relationships between the clinical characteristics and MRI findings were assessed.

Results After intravenous gadolinium (Gd) injection, 3D-FLAIR revealed high signal intensities in 65 patients. The corrected asymmetry ratios of cochlea correlated closely with interval time between onset and MRI. The asymmetry ratios of the inner ear structures were significantly lower in patients with final complete to partial hearing recovery. The corrected asymmetry ratios of the inner ear structures correlated with initial/final pure tone audiometry (PTA) and hearing recovery in the affected ear. Notably, it was shown that the corrected asymmetry ratios identified a poor prognosis for hearing recovery, with a sensitivity and specificity of 67.9% and 75.0% in the cochlea, 83.3% and 75.0% in the vestibule, and 52.4% and 81.2% in the vestibulocochlear nerve, respectively.

Conclusions Post-contrast 3D-FLAIR after intravenous Gd injection in SSNHL can be used to assess the permeability of the blood–labyrinth and blood–nerve barriers. The asymmetry ratios of the inner ear structures may identify patients with poor prognosis for hearing recovery. Signal characteristics are closely related to interval time between onset and MRI.

Keywords Sudden sensorineural hearing loss · Labyrinthine fluids · Magnetic resonance imaging · Signal detection analysis · 3D-FLAIR · Three-dimensional fluid attenuation inversion recovery

Introduction

Idiopathic sudden sensorineural hearing loss (SSNHL) is defined as a sensorineural hearing loss of at least 30 dB in three contiguous audiometric frequencies which occurs suddenly over a 3-day period [2, 15, 25]. In 80–90% of cases, SSNHL pathology is undetermined [3, 8, 26]. The hypothesised pathologies include viral infection, vascular compromise, trauma, disruption of the cochlear membranes, inner ear anomalies and autoimmunity [3, 4, 8, 11].

Over the past decade, the three-dimensional fluid attenuation inversion recovery (3D-FLAIR) sequence, a heavily-weighted T2 image sequence, allows for multiplanar reconstruction, isotropic voxels and higher signal-to-noise ratios. As 3D-FLAIR suppresses artifacts from the cerebrospinal fluid and the venous sinus blood flow [6, 9, 19], it has been used to detect inflammatory brain disorders, vascular disorders, tumours and other diseases such as multiple sclerosis [10, 16, 28]. Since 3D-FLAIR is more sensitive than T1 and T2 [1, 18, 21, 24, 27], it can detect alterations in the composition of the inner ear fluid, notably the endolymphatic hydrops of patients with Meniere's disease. Recent reports have suggested that high-intensity signals may be

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observed in the affected ears of some SSNHL patients [23, 27]. Gadolinium (Gd) enhancement of the inner ear has been observed for up to 4 h after intravenous Gd injection [12, 22]. Researchers have speculated that high-intensity signals in post-contrast 3D-FLAIR may indicate the breakdown of the blood–labyrinth barrier and/or the blood–nerve barrier [13, 21]. The presence of such abnormalities in 3D-FLAIR is thought to indicate a poor prognosis for SSNHL patients [14, 27]. However, the correlations between therapies, aetiology and 3D-FLAIR have not been clearly reported.

The purpose of our study was to report the clinical implications of post-contrast 3D-FLAIR in SSNHL patients and to explore the hearing outcomes and the observed abnormalities through qualitative and semi-quantitative approaches.

Materials and methods

Patients

This retrospective clinical study included 100 SSNHL patients, who visited Our outpatient department from January 2017 to August 2018 and underwent contrast-enhanced 3T temporal magnetic resonance imaging (MRI). The medical history of the study patients was collected, including patient age, sex, which ear was affected, the average baseline hearing level, the severity of the hearing loss and the presence or absence of vertigo and tinnitus. All patients underwent pure tone audiometry (PTA) and an intravenous Gd injection MRI. The criteria used to define SSNHL were as follows: the presence of a sensorineural hearing loss of 30 dB or more over at least three contiguous audiometric frequencies developed over a period of a few hours to 3 days without an obvious cause. The exclusion criteria were as follows: (1) hearing loss which had been diagnosed as an inner ear disease, such as Meniere's disease, vestibular migraines or vestibular schwannoma; (2) progressive hearing loss; (3) previous ear-related diseases, including chronic otitis media, trauma, tinnitus and other diseases; and (4) any symptoms and/or diseases in the contralateral ear.

Data collection and definitions

The basic information and clinical history of all the patients were recorded (Table 1). The PTA was recorded at first and every subsequent visit to our hospital, and 3T MRI scans were taken as soon as possible. Except for patients with hypertension and diabetes, all patients were given intravenous dexamethasone 10 mg/day for five consecutive days and intratympanic dexamethasone 5 mg/day for five consecutive days, followed by a tapered oral therapy. Hyperbaric oxygen, vasodilatation and other etiological treatments were administered at the same time.

Audiologic evaluation

Hearing levels were evaluated using an audiometer (Madsen Astera^2, Otometrics, Taastrup, Denmark) in a sound-insulated chamber. The average hearing level was calculated as the mean of the hearing levels measured at 250, 500, 1000, 2000 and 4000 Hz. If the patient did not respond to the maximum sound level produced by the audiometer, the threshold was defined as 5 dB above the maximum sound level or 115 dB. The severity of the initial hearing loss was classified as mild (26–40 dB), moderate (41–55 dB), moderately severe (56–70 dB), severe (71–90 dB) or profound (> 90 dB). The degree of recovery was evaluated according to Siegel's criteria (Table 2).

MRI and image analysis

All MRI scans were performed in a 3T MRI (MAGNETOM Prisma, Siemens Healthineers, Erlangen, Germany) with a 64-channel head/neck receive-array sensitivity-encoding head coil with a phase array wrist coil after an intravenous bolus of 0.2 mmol/kg of meglumine gadopentetate (Gadopentetate Dimeglumine Injection, Beijing, China) injection. The MRI was taken 4 h after the injection. All patients underwent an MRI of the temporal bones using the following sequence: T1, T2, 3D-FLAIR and T2-SPACE.

All the MRI images were double-blind evaluated by two experienced radiologists. The radiologists were blinded to the patient's medical histories and to the other radiologist. The evaluating criteria were as follows: (1) None (no signal in the affected ear); (2) General (The signal of the affected ear was similar to that of the contralateral inner ear and not higher than that of the contralateral inner ear); (3) Slightly higher signal (The signal of the affected ear was slightly higher than that of the contralateral inner ear but not higher than the cerebellum); (4) Significantly higher signal (The signal of the affected side was significantly higher than that of the cerebellum and the contralateral inner ear). The patients who were assessed as having the general condition were classified as MRI−, while the others were classified as MRI+. If any assessment dispute arose between the readers, a consensus was achieved by discussion.

One experienced radiologist evaluated the signal intensity on an eFilm Workstation 4.1.1 (eFilm Workstation, Chicago, USA) with an ImageJ (ImageJ, USA) on both sides. First, the T2-space images were used for anatomic reference. Second, multiple manual polygonal regions of interest (Fig. 1) were marked in the 3D-FLAIR. The region of interest (ROI) of the cochlea, the vestibule and

Table 1 Patient characteristics

	MRI+ group, $n = 65$	MRI- group, $n = 35$	P value
Age, mean (SD)	42.55 ± 12.975	43.11 ± 13.519	0.840
Sex (female/male)	35/30	19/16	0.966
Ear (right/left)	31/34	15/20	0.644
Associated symptoms			
Vertigo	40	12	0.009
Tinnitus	59	32	> 0.1
Ear fullness	42	21	0.648
Initial hearing loss			
Mild	1	4	
Moderate	3	3	0.002
Moderately severe	2	5	
Severe	15	7	
Profound	44	16	
Initial PTA (SD)	95.98 ± 19.671	79.64 ± 24.417	< 0.001
Final PTA (SD)	78.23 ± 22.926	51.00 ± 23.083	< 0.001
ΔPTA[a]	18.43	28.99	0.007
Period from onset to MRI (days)	11.00	18.00	0.780
Hearing recovery (Siegel's criteria)			
Complete recovery	2	7	
Partial recovery	2	6	< 0.001
Slight improvement	12	8	
No improvement	33	13	
Non-serviceable ear	18	1	

PTA pure tone audiometry, *SD* standard deviation, *MRI* magnetic resonance imaging, *MRI− group* the signal of the affected ear was similar to that of the contralateral inner ear and not higher than that of the contralateral inner ear, *MRI+ group* the signal of the affected ear was higher than that of the contralateral inner ear

[a] ΔPTA means difference of final PTA and initial PTA

Table 2 Siegel's criteria for hearing recovery

	Characteristic
Complete recovery	Final hearing better than 25 dB
Partial recovery	More than 15 dB gain, final hearing 26–45 dB
Slight recovery	More than 15 dB gain, final hearing 46–70 dB
No improvement	Less than 15 dB gain, final hearing 71–90 dB
Non-serviceable ear	Final hearing poorer than 91 dB

the vestibulocochlear nerve were positioned to cover as many of the anatomical structures as possible. A 25 mm^2 circular ROI in the cerebellar hemisphere was taken on both sides on the same axis and levels to measure the cerebellar signal intensity. The average signal intensity value was measured three times and designated as the signal intensity for each ear.

The signal intensity ratio was defined as the signal intensity of the cochlea, vestibule and vestibulocochlear nerve divided by that of the ipsilateral cerebellum. The asymmetry ratio was defined as the signal intensity ratio of the affected side divided by that of the contralateral side in the same structure. A correction factor (the signal intensity of the ipsilateral cerebellum in the affected side divided by the signal intensity of the ipsilateral cerebellum in the contralateral side) was used to eliminate any differences attributed to signal interference. The asymmetry ratio after correction was defined as the asymmetry ratio divided by correction factor. The other radiologist used the same method to measure the images of the first ten consecutive patients to calculate interobserver reliability.

Statistical analysis

The audiologic findings and the characteristics of the MRI− and MRI + groups were compared using the independent sample *t* test, the Mann–Whitney *U* test and the Pearson Chi square test. The Spearman rank correlation coefficient was used to quantify the relationships between the patient characteristics and the signal intensity. Receiver operating characteristic (ROC) curves were calculated for the sensitivity and specificity analyses of the asymmetry ratios to hearing recovery prognosis. The interobserver reliability and retest reliability of the asymmetry ratios were assessed by calculating intraclass correlation coefficients (ICCs). The results were considered statistically significant at *P* values of less than 0.05. All statistical analyses were performed using SPSS version 21 (SPSS, IBM, USA).

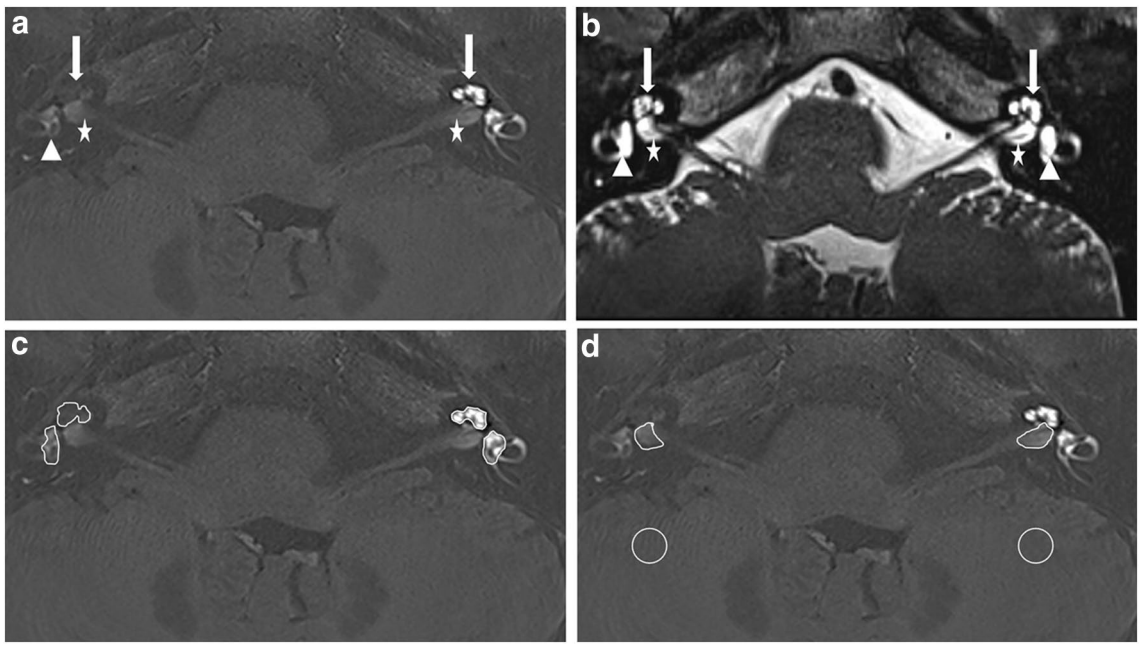

Fig. 1 3D-FLAIR MRI after intravenous gadopentetate dimeglumine injection in a 31-year-old female with right SSNHL. The affected ear had high signal intensity compared with the unaffected ear. The arrows indicate the cochlea, the triangles indicate the vestibule and the asterisks indicate the vestibulocochlear nerve. **a** Axial 3D-FLAIR image shows increased signal intensity in the right ear. **b** Axial 3D T2-SPACE image helps define the boundary of the cochlea, the vestibule and the vestibulocochlear nerve. **c** Regions of interest were designated to measure the signal intensity of the cochlea, the vestibule in axial 3D-FLAIR image. **d** Regions of interest were designated to measure the signal intensity of the vestibulocochlear nerve; 25 mm^2 circular ROIs in the cerebellar hemisphere were taken to measure the cerebellar signal intensity. *3D-FLAIR* three-dimensional fluid attenuation inversion recovery, *SSNHL* sudden sensorineural hearing loss, *3D T2-SPACE* three-dimensional T2-weighted high-sampling-efficiency technique

Results

The characteristics of the 100 SSNHL patients are summarised in Table 1. Among the 100 patients (male:female = 46:54, mean age 42.75 ± 13.102 years, age range 12–69 years). The results of clinical and MRI findings are listed in Table 1. Abnormal signals in the affected ear were found in 65 patients (33 with significantly higher signals, 32 with slightly higher signals). Three patients had endolymphatic hydrops in the cochlea, one patient had hydrops in the vestibule, two had hydrops in both cochlea and vestibule.

The average initial hearing levels were 95.98 ± 19.671 dB in the MRI + group and 79.64 ± 24.417 dB in the MRI− group (independent sample *T* test, $P < 0.001$). In the MRI + group, 59 of 65 patients were classified as having severe to profound hearing loss, compared with 23 of 35 patients in the MRI− group, a statistically significant difference (Chi square test, $P = 0.002$). The overall hearing improvement of the MRI + group was significantly less than that of the MRI− group (78.23 ± 22.926 dB versus 51.00 ± 23.083 dB, independent sample *T* test, $P < 0.001$). The rate of complete or partial recovery in the MRI− group was significantly higher than that of the MRI + group (37.14% versus 6.15%, Chi square test, $P < 0.001$). Except for the vestibular symptoms, we found no statistically significant differences in age, sex, tinnitus or aural fullness between the MRI− and MRI + groups.

In severe to profound hearing loss patients, there was no significant difference in the initial PTA between the MRI + group and the MRI− group (100.38 ± 14.231 versus 94.63 ± 12.644, $P = 0.94$ 81), but the patients in MRI + group were significantly worse in final PTA (81.58 ± 20.548 versus 58.22 ± 22.144, $P < 0.001$) and PTA enhancement (19.38 ± 17.099 versus 36.46 ± 18.798, $P < 0.001$). 3 of 59 patients showed MRI + recovered to complete or partial recovery, while 6 of 23 patients of MRI− recovered to complete or partial recovery ($P = 0.019$ after the Chi square test correction). In the mild to moderately severe hearing loss patients, MRI + group show low complete or partial recovery rate than MRI− group (1/6 versus 6/12). Because of the small sample, we did not find a significant difference in signal between the two groups (Chi square test, $P_{\text{fisher}} = 0.184$).

The eFilm and ImageJ analyses were performed to evaluate the MRI signal intensity. The affected ears had significant signal intensity enhancement in the cochlea compared with those areas of the contralateral ear (Mann–Whitney *U* test, $P_{\text{cochlea}} = 0.003$). The asymmetry ratios of the inner ear structures after correction were significantly lower in the patients with final complete to partial hearing recovery (Mann–Whitney *U* test, $P_{\text{cochlea}} = 0.001$, $P_{\text{vestibule}} < 0.001$, $P_{\text{vestibulocochlear nerve}} = 0.048$). The Spearman correlation demonstrated that the corrected asymmetry ratios of the inner ear structures were significantly correlated with initial hearing loss level, hearing recovery and aural fullness (Table 3). Vestibular symptoms also correlated with the corrected asymmetry ratios of the cochlea and the vestibulocochlear nerve. The ROC curves were plotted to analyse whether high signal intensity indicated a poor prognosis for hearing recovery (Fig. 2). According to the Youden index method, the corrected asymmetry ratios identified patients with slight recovery, no improvement or non-serviceable ear with the following sensitivities and specificities in the specified structures: 67.9% and 75.0% in the cochlea (cutoff asymmetry ratio, 1.099; area under curve [AUC], 0.757, $P = 0.01$); 83.3% and 75.0% in the vestibule (cutoff asymmetry ratio, 1.056; AUC 0.802, $P < 0.001$);

Table 3 Correlations between patient characteristics and signal intensities

	cAR_C	cAR_V	cAR_{INC}
Vertigo			
P value	0.031	0.196	0.001
r_s	0.288	0.096	0.250
Tinnitus			
P value	0.090	0.204	0.795
r_s	0.229	0.094	0.019
Ear fullness			
P value	0.219	0.005	0.009
r_s	0.167	0.208	−0.193
Initial hearing loss[a]			
P value	< 0.001	0.031	0.182
r_s	0.300	0.159	0.099
Initial PTA			
P value	< 0.001	< 0.001	0.008
r_s	0.423	0.278	0.196
Final PTA			
P value	< 0.001	< 0.001	0.002
r_s	0.443	0.467	0.223
Hearing recovery[b]			
P value	< 0.001	< 0.001	0.014
r_s	0.302	0.498	0.180
Period from onset to MRI			
P value	< 0.001	0.390	0.080
r_s	−0.376	−0.064	−0.130

PTA pure tone audiometry, cAR_C corrected asymmetry ratio of the cochlea, cAR_V corrected asymmetry ratio of the vestibule, cAR_{INC} corrected asymmetry ratio of the vestibulocochlear nerve, *MRI* magnetic resonance imaging, r_s Spearman rank correlation coefficient

[a]Initial hearing loss: initial hearing loss level

[b]Hearing recovery: hearing recovery level according to the Siegel's criteria

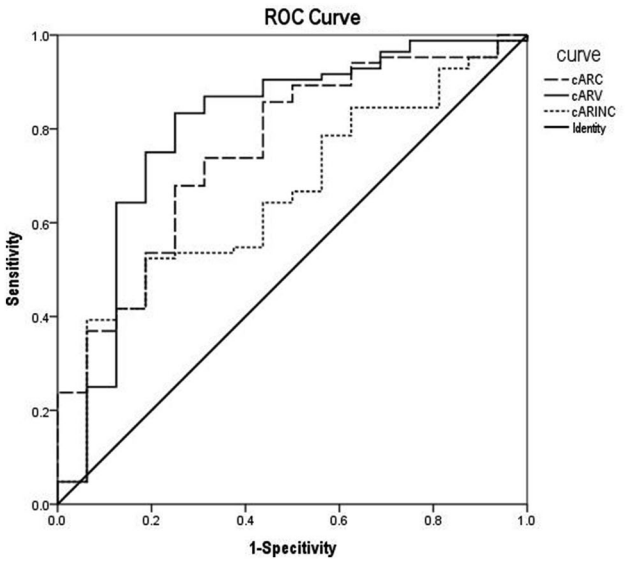

Fig. 2 ROC Curve Analysis of the Asymmetry Ratios of the Post-contrast 3D FLAIR for Identifying the Hearing Recovery Prognosis of Patients with Sudden Hearing Loss. *cARC* corrected asymmetry ratio of the cochlea, *cARV* corrected asymmetry ratio of the vestibule, *cARINC* corrected asymmetry ratio of the vestibulocochlear nerve, *ROC* receiver operating characteristic curve, *3D-FLAIR* three-dimensional fluid attenuation inversion recovery, *CR* complete recovery, *PR* partial recovery, *SR* slight recovery, *NI* no improvement, *NS* non-serviceable ear

and 52.4% and 81.2% in the vestibulocochlear nerve (cut-off asymmetry ratio, 1.040; AUC 0.656, $P = 0.039$). The ICC of the corrected asymmetry ratio measured between both reviewers was a good degree of reliability for the cochlea (ICC 0.915), the vestibule (ICC 0.965) and the vestibulocochlear nerve (ICC 0.625).

The intravenous meglumine gadopentetate 3D-FLAIR MRI was performed on all patients within 60 days after the onset of the hearing loss. There are 69.05% (58/84) patients showed high signal (28 with significantly higher signals, 30 with slightly higher signals) within 30 days after the onset of the hearing loss, while 43.75% (7/16) patients display abnormal (5 with significantly higher signals, 2 with slightly higher signals) within 31–60 days. Although we did not find a significant difference in signal between the two groups (Chi square test, $P = 0.052$), it is noteworthy that the corrected asymmetry ratios of the cochlea showed a close correlation with interval time between onset and MRI ($P = 0.003$, $r_s = -0.299$).

Discussion

In recent studies, abnormal signals in pre/post-contrast 3D-FLAIR MRIs have been detected in the affected ears of patients with sudden deafness, and high signal intensities in the affected ear have been inversely correlated with initial hearing levels and hearing improvement [20, 27]. A recent study conducted using intravenous Gd 3D-FLAIR in patients with SSNHL [1, 22] found that the signal intensities of the cochlea, the vestibule and the vestibulocochlear nerve in the affected ear were significantly higher than in those of the healthy side. It has been suggested that this difference may indicate destruction of the blood–labyrinth barrier and/or the blood–nerve barrier [1]. Our study found no absent signals in the inner ears of all patients. The 65.00% (65/100) Gd enhancement finding in the affected ear differs from the studies of Stefano et al. [1] (34.78%, 8/23), Lee et al. [13] (13.27%, 15/113) and Liao et al. [14]. (59.30%, 32/54). This difference appears to reflect the different MRI diagnostic criteria and measurement methods, and the dose of gadolinium and interval time of delayed intravenous gadolinium-enhanced 3D-FLAIR MR imaging were different in various studies.

It is noteworthy that "no signal" in the affected side of the inner ear was not observed in our study, indicating that gadolinium could enter the inner ear through blood vessels without significant obstruction of the inner ear vessels. We speculate that the proportion of vascular embolism in the cause of sudden deafness is lower than expected. But this may be related to the period from onset to MRI, because MR imaging in the acute phase of vascular obstruction is different from that in the chronic phase.

According to our study, the affected side showed slightly high signals rather than significantly high signals in patients on whom MRI was performed over 50 days after the onset of hearing loss, and the corrected asymmetry ratios of the cochlea were inversely correlated closely with the interval time between onset and MRI. We also found that some patients who displayed significantly high signals within 50 days after the onset of hearing loss became slightly high signals after a few months (Fig. 3). We hypothesise that the blood–labyrinth barrier can be gradually repaired over time, so we cannot observe a significantly high signal. But the changes of endolymphatic fluid composition caused by the blood–labyrinth barrier breakdown need a long time to recover, so we can still observe a slightly high signal in the affected inner ear for a period of time. This supports our hypothesis that vascular obstruction may not be the main cause of sudden deafness.

According to Gao's meta-analysis [5], high signal intensities in 3D-FLAIR indicate a poor hearing prognosis. Our study found that fewer patients in the post-contrast 3D-FLAIR high signal intensity group recovered to complete or partial recovery (Chi square test, $P < 0.001$). The enhancement in post-contrast 3D-FLAIR may be associated with damage to the blood–labyrinth barrier and/or the blood–nerve barrier, which may be caused by bleeding or inflammation. This abnormal signal indicates previous or

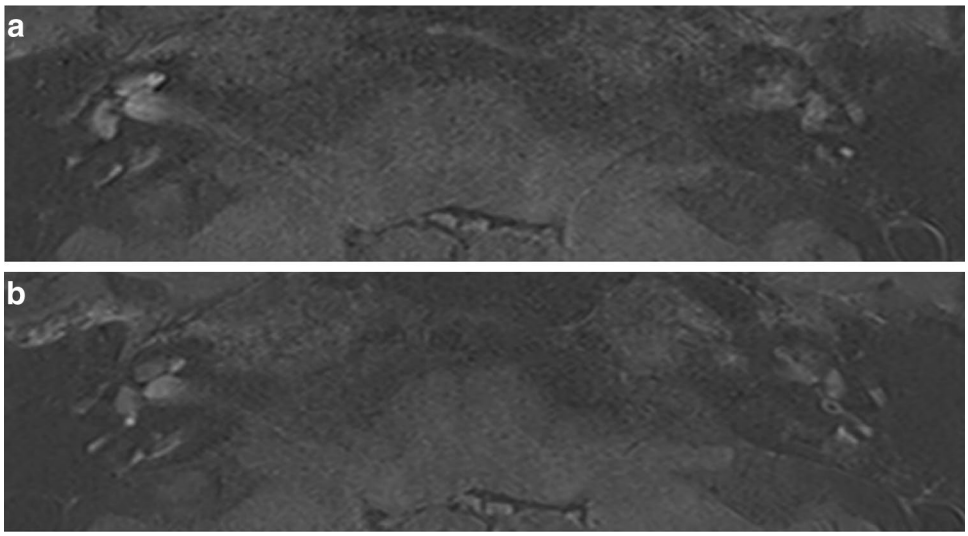

Fig. 3 Abnormal MRI findings in a 66-year-old woman with right SSNHL. **a** Axial 3D-FLAIR image in 5th days after the onset of hearing loss shows significantly higher signals in the inner ear structures than the contralateral inner ear. **b** Axial 3D-FLAIR image in 95th days after the onset of hearing loss shows slightly higher signals in the inner ear structures. The hearing recovery level is no improvement

present endolymphatic damage, which is more likely to cause vertigo.

A previous study reported that the ratio of the signal intensities of the inner ear and the cerebellar hemisphere may be a prognostic indicator for evaluating the disruption of the blood–labyrinthine barrier in sudden deafness [22]. Liao et al. [14] found that 3D-FLAIR was more sensitive than 3D FIESTA-C and post-contrast 3D FSPGR in detecting signal intensity asymmetry in patients with unilateral SSNHL. Although Liao's study speculated that a greater difference between pre-contrast and post-contrast 3D-FLAIR indicated a lower chance of hearing recovery, no statistical significance was provided ($P = 0.055$). Notably, we found overall hearing improvement were worse in patients who showed high signal intensity than the MRI negative patients, especially in severe to profound hearing loss patients. Moreover, the signal intensity ratios of the inner ear structures in the affected ear were higher than in the contralateral inner ear. The corrected asymmetry ratios of the inner ear structures were all significantly correlated with initial and final PTA. We propose that the signal intensity might be related to the permeabilities of the blood–labyrinth or the blood–nerve barriers. Serious inner ear damage leads to greater permeability of these barriers, which would cause higher signal intensities and a poor prognosis for hearing loss recovery. We used ROC curves and the Youden index to determine the optimal correction ratio for each structure. These ratios can be used as prognostic factors for treatment. We conclude that higher asymmetry ratios in the inner ear, including the ratios of the cochlea, the vestibule and the vestibulocochlear nerve, indicate a poor prognosis.

Despite the research which has been conducted over the past decades, the aetiology of sudden deafness is still unknown. Some studies have suggested that viral infection and vascular factors are the primary aetiologies. Consequently, clinical doctors often use steroids and vasodilators to treat the disease immediately when SSNHL attacks. However, these two treatments are contradictory. Systemic steroids can induce a prothrombotic state due to the glucocorticoid effect. They can also increase synthesis of the von Willebrand factor and plasminogen activator inhibitor-1 [7, 17]. To provide a clear basis for the treatment and prognosis of sudden deafness, it is critical to determine the relationship between hearing loss aetiology and the anomalies observed in MRIs. It is difficult but preferable to perform MRI before the masking effect of steroids.

There were a few limitations to our study. The interval between the onset of hearing loss and the MRI scans did not show significant differences between signals because of the small sample. Additional studies are needed to evaluate the relationship between interval and signal intensity. This research concentrated primarily on the connection between post-contrast 3D-FLAIR and clinical findings. More research and animal experiments are needed in aetiology. We used a new method to measure signal intensity and to calculate the asymmetry ratios, and the ICC values are in an acceptable range, which is superior to that used in the previous research. This novel method provides guidance for the future study of SSNHL aetiology and therapy and should be further explored.

Conclusion

This is a unique report using new methods of measurement and calculation to clearly demonstrate a correlation between sudden deafness and high signal intensities in post-contrast 3D-FLAIR after intravenous double-dose Gd injection. We conclude that a higher asymmetry ratio in the cochleae, the vestibule and the vestibulocochlear nerve correlates with

a poorer hearing loss prognosis. Signal characteristics are closely related to interval time between onset and MRI. We hypothesise that vascular obstruction may not be the main cause of sudden deafness.

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Compliance with ethical standards

Conflict of interest No potential conflicts of interest relevant to this article are reported.

References

1. Berrettini S, Seccia V, Fortunato S, Forli F, Bruschini L, Piaggi P, Canapicchi R (2013) Analysis of the 3-dimensional fluid-attenuated inversion-recovery (3D-FLAIR) sequence in idiopathic sudden sensorineural hearing loss. JAMA Otolaryngol Head Neck Surg 139(5):456–464. https://doi.org/10.1001/jamaoto.2013.2659
2. Biavati MJ, Gross JD, Wilson WR, Dina TS (1994) Magnetic resonance imaging evidence of a focal pontine ischemia in sudden hearing loss and seventh nerve paralysis. Am J Otol 15(2):250–253
3. Chau JK, Lin JR, Atashband S, Irvine RA, Westerberg BD (2010) Systematic review of the evidence for the etiology of adult sudden sensorineural hearing loss. Laryngoscope 120(5):1011–1021. https://doi.org/10.1002/lary.20873
4. Eisenman D, Arts HA (2000) Effectiveness of treatment for sudden sensorineural hearing loss. Arch Otolaryngol Head Neck Surg 126(9):1161–1164
5. Gao Z, Chi FL (2014) The clinical value of three-dimensional fluid-attenuated inversion recovery magnetic resonance imaging in patients with idiopathic sudden sensorineural hearing loss: a meta-analysis. Otol Neurotol 35(10):1730–1735. https://doi.org/10.1097/MAO.0000000000000611
6. Hajnal JV, Bryant DJ, Kasuboski L, Pattany PM, De Coene B, Lewis PD, Pennock JM, Oatridge A, Young IR, Bydder GM (1992) Use of fluid attenuated inversion recovery (FLAIR) pulse sequences in MRI of the brain. J Comput Assist Tomogr 16(6):841–844
7. Huang LQ, Whitworth JA, Chesterman CN (1995) Effects of cyclosporin A and dexamethasone on haemostatic and vasoactive functions of vascular endothelial cells. Blood Coagul Fibrinolysis 6(5):438–445
8. Hughes GB, Freedman MA, Haberkamp TJ, Guay ME (1996) Sudden sensorineural hearing loss. Otolaryngol Clin N Am 29(3):393–405
9. Jack CJ, Rydberg CH, Krecke KN, Trenerry MR, Parisi JE, Rydberg JN, Cascino GD, Riederer SJ (1996) Mesial temporal sclerosis: diagnosis with fluid-attenuated inversion-recovery versus spin-echo. MR imaging Radiol 199(2):367–373. https://doi.org/10.1148/radiology.199.2.8668780
10. Jin EH, Liang YT, Ma DQ (1999) Diagnostic evaluation of subacute and chronic subarachnoid hemorrhage with MRI using turbo fluid attenuated inversion recovery pulse sequence. Chin J Radiol(6):384. https://doi.org/10.3760/j.issn:1005-1201.1999.06.008
11. Kim C, Sohn JH, Choi HC (2012) Vertebrobasilar angulation and its association with sudden sensorineural hearing loss. Med Hypotheses 79(2):202–203. https://doi.org/10.1016/j.mehy.2012.04.035
12. Kim TY, Park DW, Lee YJ, Lee JY, Lee SH, Chung JH, Lee S (2015) Comparison of inner ear contrast enhancement among patients with unilateral inner ear symptoms in MR images obtained 10 minutes and 4 hours after gadolinium injection. AJNR Am J Neuroradiol 36(12):2367–2372. https://doi.org/10.3174/ajnr.A4439
13. Lee HY, Jung SY, Park MS, Yeo SG, Lee SY, Lee SK (2012) Feasibility of three-dimensional fluid-attenuated inversion recovery magnetic resonance imaging as a prognostic factor in patients with sudden hearing loss. Eur Arch Otorhinolaryngol 269(8):1885–1891. https://doi.org/10.1007/s00405-011-1834-1
14. Liao WH, Wu HM, Wu HY, Tu TY, Shiao AS, Castillo M, Hung SC (2016) Revisiting the relationship of three-dimensional fluid attenuation inversion recovery imaging and hearing outcomes in adults with idiopathic unilateral sudden sensorineural hearing loss. Eur J Radiol 85(12):2188–2194. https://doi.org/10.1016/j.ejrad.2016.10.005
15. Loughran S (2000) Management of sudden sensorineural hearing loss: a consultant survey. J Laryngol Otol 114(11):837–839
16. Moayer R, Ishiyama GP, Karnezis S, Sepahdari AR, Ishiyama A (2018) High resolution three-dimensional delayed contrast MRI detects endolymphatic hydrops in patients with vertigo and vestibular schwannoma. Otol Neurotol 39(1):e39–e44. https://doi.org/10.1097/MAO.0000000000001627
17. Morange PE, Aubert J, Peiretti F, Lijnen HR, Vague P, Verdier M, Negrel R, Juhan-Vague I, Alessi MC (1999) Glucocorticoids and insulin promote plasminogen activator inhibitor 1 production by human adipose tissue. Diabetes 48(4):890–895
18. Naganawa S, Koshikawa T, Nakamura T, Kawai H, Fukatsu H, Ishigaki T, Komada T, Maruyama K, Takizawa O (2004) Comparison of flow artifacts between 2D-FLAIR and 3D-FLAIR sequences at 3 T. Eur Radiol 14(10):1901–1908. https://doi.org/10.1007/s00330-004-2372-7
19. Rydberg JN, Hammond CA, Grimm RC, Erickson BJ, Jack CJ, Huston JR, Riederer SJ (1994) Initial clinical experience in MR imaging of the brain with a fast fluid-attenuated inversion-recovery pulse sequence. Radiology 193(1):173–180. https://doi.org/10.1148/radiology.193.1.8090888
20. Ryu IS, Yoon TH, Ahn JH, Kang WS, Choi BS, Lee JH, Shim MJ (2011) Three-dimensional fluid-attenuated inversion recovery magnetic resonance imaging in sudden sensorineural hearing loss: correlations with audiologic and vestibular testing. Otol Neurotol 32(8):1205–1209. https://doi.org/10.1097/MAO.0b013e31822e969f
21. Sugiura M, Naganawa S, Teranishi M, Nakashima T (2006) Three-dimensional fluid-attenuated inversion recovery magnetic resonance imaging findings in patients with sudden hearing loss. Laryngoscope 116(8):1451–1454. https://doi.org/10.1097/01.mlg.0000228005.78187.23
22. Tagaya M, Teranishi M, Naganawa S, Iwata T, Yoshida T, Otake H, Nakata S, Sone M, Nakashima T (2010) 3 T magnetic resonance imaging obtained 4 hours after intravenous gadolinium injection in patients with sudden deafness. Acta Otolaryngol 130(6):665–669. https://doi.org/10.3109/00016480903384176
23. Tanigawa T, Shibata R, Tanaka H, Gosho M, Katahira N, Horibe Y, Nakao Y, Ueda H (2015) Usefulness of three-dimensional fluid-attenuated inversion recovery magnetic resonance imaging to detect inner-ear abnormalities in patients with sudden sensorineural hearing loss. J Laryngol Otol 129(1):11–15. https://doi.org/10.1017/S0022215114003028

24. Tanigawa T, Tanaka H, Sato T, Nakao Y, Katahira N, Tsuchiya Y, Nonoyama H, Ueda H (2010) 3D-FLAIR MRI findings in patients with low-tone sudden deafness. Acta Otolaryngol 130(12):1324–1328. https://doi.org/10.3109/00016489.2010.496461
25. Whitaker S (1980) Idiopathic sudden hearing loss. Am J Otol 1(3):180–183
26. Wilson WR, Byl FM, Laird N (1980) The efficacy of steroids in the treatment of idiopathic sudden hearing loss. A double-blind clinical study. Arch Otolaryngol 106(12):772–776
27. Yoshida T, Sugiura M, Naganawa S, Teranishi M, Nakata S, Nakashima T (2008) Three-dimensional fluid-attenuated inversion recovery magnetic resonance imaging findings and prognosis in sudden sensorineural hearing loss. Laryngoscope 118(8):1433–1437. https://doi.org/10.1097/MLG.0b013e318172ef85
28. Zivadinov R, Ramasamy DP, Hagemeier J, Kolb C, Bergsland N, Schweser F, Dwyer MG, Weinstock-Guttman B, Hojnacki D (2018) Evaluation of leptomeningeal contrast enhancement using pre-and postcontrast subtraction 3D-FLAIR imaging in multiple sclerosis. AJNR Am J Neuroradiol 39(4):642–647. https://doi.org/10.3174/ajnr.A5541