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HEAD AND NECK

Detection of intralabyrinthine abnormalities using post-contrast delayed 3D-FLAIR MRI sequences in patients with acute vestibular syndrome

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Abstract

Purpose 3D-FLAIR sequences with delayed acquisition after contrast medium injection have demonstrated new insights into blood-labyrinthine barrier (BLB) abnormalities in various diseases. The aim of this study was to assess the BLB in patients referred with unilateral acute vestibular syndrome (UAVS).

Materials and methods In this retrospective multicenter imaging study, we performed 3D-FLAIR and steady-state free precession (SSFP) sequences 4 h after contrast medium administration in 26 healthy volunteers and in 30 patients with UAVS. Two radiologists, blinded to the clinical data, independently assessed the asymmetrical enhancement of the labyrinthine structures and the vestibular nerve on 3D-FLAIR sequences, and the signal of the labyrinthine structures on SSFP sequences. Inter-reader agreement tests were performed.

Results An asymmetrical enhancement of the semicircular canals was observed in 26 out of 30 ears (86.6%, $p < 0.001$) and never observed in healthy subjects. An asymmetrical enhancement of the vestibular nerve was never observed in either patients or healthy subjects. An asymmetrical enhancement of the cochlea was observed on the 3D-FLAIR sequence in 6 out of 30 ears only in the patients' group (20%, $p = 0.03$) and always associated with an enhancement of at least one semicircular canal. A low signal on SSFP sequences was observed only in 11 out of 30 symptomatic ears (36.7%, $p < 0.001$), involving the utricle in 7 ears and the superior semicircular canal in 4 ears.

Conclusion Patients with typical UAVS presented with semicircular canal enhancement on MRI, while an asymmetrical enhancement of the vestibular nerve was not displayed.

Trial registration NCT02529475

Key Points

- *Patients with typical vestibular neuronitis presented with semicircular canal enhancement on MRI in 87% of cases.*
- *An enhancement of the vestibular nerve was never displayed.*

Keywords Acute vestibular syndrome · MRI · 3D-FLAIR · Inner ear

Abbreviations

BLB	Blood-labyrinthine barrier
MD	Menière's disease
SSFP	Steady-state free precession
UAVS	Unilateral acute vestibular syndrome

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Introduction

Unilateral acute vestibular syndrome (UAVS), also known as vestibular neuritis, is the third most common cause of peripheral vertigo after benign paroxysmal positional vertigo and Menière's disease [1]. UAVS is caused by a sudden loss of the vestibular function causing a rotatory vertigo for several days without any auditory- or neurological-associated symptoms.

The origin of UAVS remains controversial and three theories have been hypothesized [2]: (1) infectious, by reactivation of neurotropic virus on the vestibular nerve; (2) vascular, by a reduced perfusion of the vestibular organ; and (3) immunological, by a mediated immune inflammation of the vestibular nerve. The infectious theory is the most commonly accepted. However, a study performed on 60 patients with vestibular neuritis never displayed an enhancement of the vestibular nerve on T1-weighted images with enhanced MRI [3]. It was suggested that the lack of enhancement was due to the poor prominent circumneural arteriovenous structures of the vestibular nerve.

Recently, Byun et al [4] performed 3D-FLAIR sequences 4 h after a double-dose intravenous administration of gadolinium. They demonstrated an enhancement of the vestibular nerve in 69% of cases and of the vestibule and semicircular canals in 41% of the cases. The authors suggested that the initial vestibular nerve inflammation caused the distribution of gadolinium in the perilymphatic space of the vestibule. 3D-FLAIR sequences enabled the visualization of subtle compositional changes of the inner ear fluids in various diseases tied to hemorrhage or increased protein levels [5]. It has been demonstrated that an impairment of the blood-labyrinthine barrier (BLB) in the cochlea, as evaluated with 3D-FLAIR sequences performed 4 h after an intravenous administration, is observed in various inner ear diseases such as Menière's disease, otosclerosis, and perilymphatic fistula [6, 7].

Recently, a temporal bone histopathological study assessed the BLB in the vestibular organ and found a dysfunction in the microvasculature of vestibular end organs in Menière's disease patients [8].

The aim of this study was to assess whether an impairment of the vestibular BLB, as evaluated by 3D-FLAIR sequences, can be displayed in UAVS patients.

Methods

Patients

This was a multicenter, parallel group, retrospective imaging study (IRB E2018-46) from three university hospitals. The imaging data coming from the healthy volunteers' cohort was registered with the ClinicalTrials.gov registry (identifier: NCT02529475). Twenty-six healthy volunteers, with no history or symptoms of inner ear, brain, or psychiatric disorders, were consecutively included in this study. Twenty of the 26 healthy subjects have been previously reported [9]. Patients were consecutively included between May 2017 and April 2018.

Inclusion criteria were (1) acute vertigo for at least 24 h, (2) spontaneous horizontal unidirectional nystagmus at the time of physical examination, (3) complete or severe canal paresis using Jongkee's formula, and (4) absence of auditory or neurological symptoms.

Exclusion criteria were (1) clinical evidence of central nervous system involvement, (2) unilateral or asymmetric hearing loss, (3) recurrence of vertigo, and (4) patients with a history of intratympanic therapy or a previous UAVS episode.

Audiometric tests and bithermal caloric tests

The pure-tone average hearing levels of air and bone conduction were evaluated.

Bithermal caloric tests were performed by irrigating the ears alternatively with 150 to 250 ml of cold and hot water (30 °C and 44 °C, respectively). The induced nystagmus was recorded with video-oculography (Ulmer VNG, v. 4.15.0.275, SYNAPSYS®). Asymmetry of vestibular function was calculated using Jongkees' formula. Canal paresis was considered to be abnormal when ≥ 20%.

Imaging

Healthy volunteers underwent post-contrast delayed MRI on 3-T Siemens Skyra® and 3-T Philips Achieva® TX MRI while patients' examinations were carried out on 3-T General Electric Discovery®, 3-T Siemens Skyra®, and 3-T Philips Achieva® TX MRI scanners. Surface coil (FLEX), 64-channel head coil, and 32-channel SENSE head coil were respectively used.

All patients underwent an MRI scan after a single intravenous dose of gadobutrol (Gd-DO3A-butrol, Gadovist® 0.1 mmol/kg, 1 mmol/ml) that provided a high contrast in the labyrinth [9]. We performed the 3D-FLAIR and the steady-state free precession (SSFP) sequences 4 h after contrast administration with parameters summarized in Tables 1 and 2. No pre-contrast or immediate post-contrast imaging was performed.

All patients received oral corticosteroids (methylprednisone 1 mg/kg per day for 7 days) as part of their treatment following the clinical examination and before MRI scan acquisition.

Assessment of the vestibular BLB

Images for each subject were evaluated independently with Osirix MD® by two readers who were blinded to the clinical

Table 1 3D-FLAIR sequences parameters

Sequences	Vendor	Repetition time (ms)	Echo time (ms)	Inversion time (ms)	Section thickness (mm)	Flip angle (degree)	Nex	ELT	Acceleration factor	Matrix size	Scan time (min)
Cube	General Electric	9000	130	2376	0.5	Variable	2	145	2	288 × 288	7′30
Brainview	Phillips	7600	345	2300	0.4	Variable	4	101	2.5	252 × 252	9′
SPACE	Siemens	10,000	323	2500	0.4	Constant (165°)	2	125	2	320 × 280	7′50

data (ME and JH, senior radiologists with certificates of added qualification in head and neck imaging).

A visual assessment was performed based on the symmetrical enhancement of the labyrinthine structures (semicircular canals and cochlea) and of the vestibular nerve on the 3D-FLAIR sequence. We did not consider to be pathological a slight bilateral increased enhancement of the common crus and the posterior semicircular canal (Fig. 1), as they are constantly visible in the control group. For the purpose of the study, the asymmetrical enhancement of the labyrinthine structures and the vestibular nerve, evaluated by the most experienced radiologist, was taken as an abnormal finding.

We also performed a visual assessment based on the low signal of the labyrinth on SSFP sequences. Recently, it has been demonstrated that the vestibular endolymphatic space could be evaluated on 3-T MRI with SSFP sequences on a coronal plane going through the lateral and superior semicircular canal ampullae. The utricular macula appears as a linear area of low signal intensity at the level of the lateral semicircular canal and is outlined by hyperintense endolymph, which corresponds to the utricle superiorly and the saccule inferiorly [10, 11].

Statistical analysis

Data were analyzed using R software v 3.4.2 (The R Foundation for Statistical Computing).

The inter-reader agreement in detecting asymmetrical enhancement of the semicircular canals, cochlea, and vestibular nerve from the 3D-FLAIR sequence was estimated using Cohen's kappa coefficient (k). We considered a k value greater than 0.80 to indicate a very good agreement and less than 0.2 as a very poor agreement [12]. Continuous data are presented as the mean with a standard deviation. Between-group comparisons were analyzed with Student's t test for continuous data to assess the mean age difference between the groups. Sensitivity and specificity were estimated by taking the clinical examination as the gold standard and were estimated by the exact Clopper-Pearson interval.

We report categorical data as frequency and percentages. We set the significance threshold (p values) at 0.05.

Results

Population

Twenty-six healthy volunteers (13 women) with a mean age of 59.7 ± 10.1 years and 30 UAVS patients (16 women) with a mean age of 51.4 ± 15.2 years were included in this study. Patients presented with UAVS, 9 on their right and 21 on their left side.

There was a significant difference in the mean ages of the healthy subjects and the patient groups ($p = 0.044$).

The delay between inner ear symptoms and MR imaging was 24.7 ± 15.3 days ranging from 1 to 60 days. The mean canal paresis on caloric tests was 84.6% ± 23.1%. The right side was involved in 10 of these patients and the left ear in 20 of these patients.

MRI data (Table 3)

In healthy volunteers (Fig. 2), no asymmetrical enhancement of the labyrinthine structures or of the vestibular nerve was found. The rate of semicircular canal and vestibular nerve asymmetrical enhancement was estimated by the Clopper-Pearson method at 0% (IC95 0%; 6.4%). A low signal of the

Table 2 High-resolution SSFP sequence parameters

Sequences	Vendor	Repetition time (ms)	Echo time (ms)	Section thickness (mm)	Flip angle (degree)	Number of excitations	Acceleration factor	Matrix size	Scan time (min)
FIESTA	General Electric	6.4	2.6	0.3	60	1	2	484 × 484	4′20
bFFE	Philips	5.3	2.2	0.45	25	4	1	424 × 426	5′24
CISS	Siemens	6.6	3.1	0.4	57	1	1	320 × 320	5′35

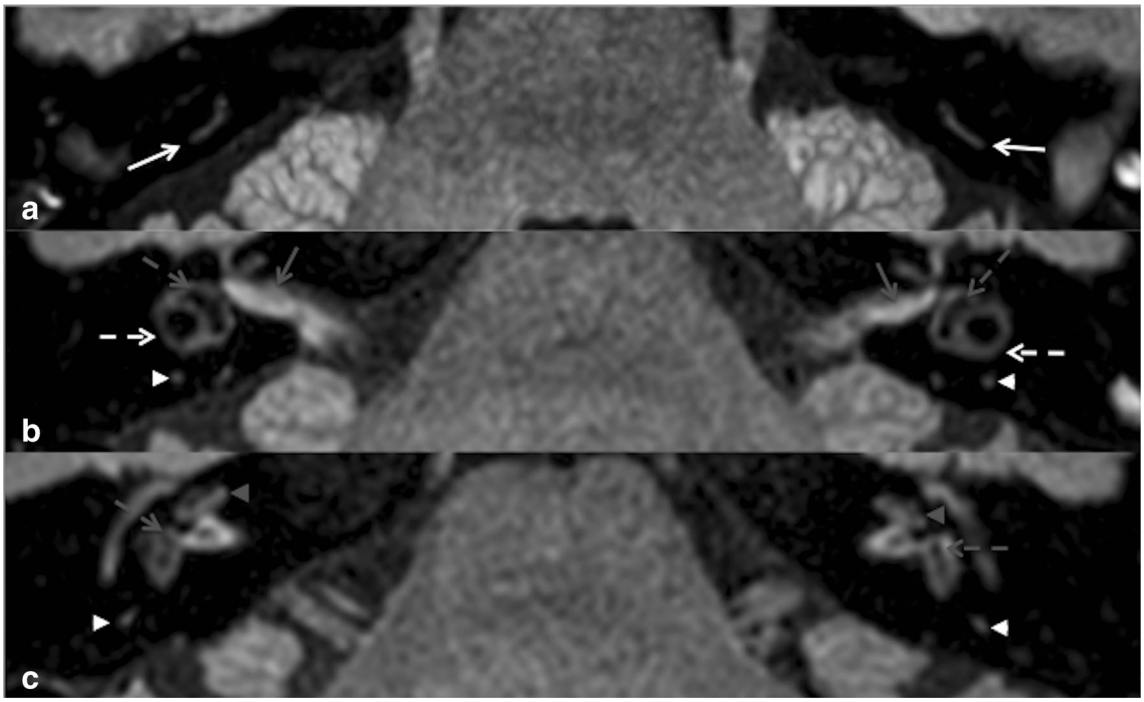

Fig. 1 A 71-year-old female (healthy subject). **a** Axial 3D-FLAIR at the level of the superior semicircular canals showed a slight increased enhancement of both posterior semicircular canals (white arrow) considered as not pathological. **b** Axial 3D-FLAIR at the level of the utricle (gray dotted arrow) demonstrated a symmetrical enhancement of the superior vestibular nerves (gray arrow) and of the lateral and posterior semicircular canals (white arrowhead). **c** Axial 3D-FLAIR at the level of the saccule (gray dotted arrow) demonstrated a symmetrical enhancement of the posterior semicircular canal (white arrowhead) and cochlea (gray arrowhead)

labyrinth on the SSFP sequence for each patient was never displayed.

In patients, an asymmetrical enhancement of the vestibular nerve was never seen on the delayed 3D-FLAIR sequences. A marked enhancement of the semicircular canals (Fig. 3) was observed on the 3D-FLAIR sequence in 26 out of 30 ears (90%, IC95 73.5%; 97.9%, $p < 0.0001$), involving the superior canal in 18 ears, the lateral canal in 17 ears, and the posterior canal in 5 ears. Two semicircular canals were both involved in 5 ears (superior and lateral in 4 ears, lateral and posterior in 1 ear) and all three semicircular canals were involved in 3 ears. The specificity was estimated by the Clopper-Pearson method at 100% (IC95 93.6%; 100%). The inter-reader agreement was 0.86 for the asymmetrical enhancement of the semicircular canals.

An asymmetrical enhancement of the cochlea (Fig. 3) was observed on the 3D-FLAIR sequence in 6 out of 30 ears (20%, IC95 8.5%; 37%, $p = 0.03$) and always associated with an enhancement of at least one semicircular canal. The inter-reader agreement was 0.77 for the asymmetrical enhancement of the cochlea.

A low signal on SSFP sequence was observed in 11 out of 30 ears (36.7%, IC95 (19.9%; 56.1%), $p < 0.001$) involving the utricle above the utricular macula in 7 ears and the superior semicircular canal in 4 ears (Fig. 4). It was always associated with a marked labyrinthine enhancement on the 3D-FLAIR sequence. The specificity was estimated by the Clopper-Pearson method at 100% (IC95 93.6%; 100%).

The inter-reader agreement was 0.52 for the intralabyrinthine low signal on SSFP sequence.

Discussion

We showed that 86% of patients referred with UAVS presented vestibular BLB impairment on delayed 3D-FLAIR sequences, while no patient presented with asymmetrical enhancement of the vestibular nerve. We also showed that 36% of patients presented with a low signal on SSFP sequences, involving the utricule and the superior semicircular canal (Fig. 5).

Physiopathology of UAVS

Since its first description by Ruttin in 1909 [13], the infectious hypothesis is the most accepted, based on the presence of Herpes simplex virus type 1 (HSV-1) in human vestibular ganglia. The association with preceding respiratory tract infections is present in nearly half of patients with UAVS [14]. Inoculation of HSV-1 into the auricle of mice managed to reproduce vestibular neuritis symptoms, strengthening the infection hypothesis [15].

Table 3 Clinical and radiological characteristics in patients with UAVS

Patients	Age	Sex	Side	Delay MRI (days)	Caloric test (%)	MRI			
						FLAIR			SSFP
						Vestibular nerve	Semicircular canal	Cochlea	Vestibule
1	63	M	L	27	58	0	1	0	0
2	57	M	L	40	100	0	1	0	0
3	50	F	R	23	30	0	0	0	0
4	41	M	L	2	100	0	1	1	1
5	24	M	L	10	100	0	1	1	0
6	33	M	L	20	100	0	0	0	1
7	37	M	R	28	100	0	1	1	1
8	59	F	L	13	100	0	1	0	0
9	48	F	L	20	100	0	1	1	0
10	44	F	R	30	100	0	1	0	0
11	70	M	L	16	94	0	1	0	1
12	68	F	L	24	100	0	0	0	0
13	37	F	L	21	34	0	1	0	0
14	66	M	R	28	94	0	1	0	1
15	32	F	R	15	100	0	1	0	0
16	58	M	R	40	100	0	1	0	0
17	46	M	L	48	100	0	1	0	1
18	79	F	L	7	100	0	1	1	0
19	31	M	L	60	84	0	1	0	1
20	73	F	L	1	62	0	1	0	1
21	58	M	L	3	100	0	1	0	0
22	66	F	L	1	100	0	1	0	0
23	55	F	L	30	80	0	1	0	0
24	59	F	R	40	87	0	1	0	1
25	33	F	R	52	63	0	0	0	0
26	40	M	R	45	67	0	1	0	1
27	48	F	L	20	100	0	1	1	0
28	62	H	L	32	54	0	1	0	0
29	31	F	L	24	32	0	1	0	0
30	75	F	L	23	100	0	1	0	1

FLAIR (0, symmetrical enhancement; 1, asymmetrical enhancement). SSFP (0, normal; 1, low signal)

However, in 1956, Lindsay and Hemenway [16] described cases of UAVS caused by an occlusion of the anterior vestibular artery. A pro-inflammatory state in patients with UAVS has lately been reported, leading to thrombotic events and a reduced perfusion of the vestibular organ causing the vestibular loss function [2]. Various authors also refute the infectious hypothesis because some studies found no evidence of seasonality and a significantly higher prevalence of cardiovascular risk factors, in comparison with the general population [17, 18]. Similarly, Chuang et al [19] showed a significantly higher incidence of vertebral artery hypoplasia in patients with UAVS, along with an association between vertebral artery hypoplasia and ipsilateral vestibular neuritis, mostly on the left side.

In our study, the superior and the lateral semicircular canals were more involved than the posterior semicircular canal. It has been demonstrated that transitory supply disorders in the inner ear are more likely to damage the superior vestibular organ for two reasons [20]: (1) the absence of collateral supply of the terminal arterial branches of the superior vestibular organ and (2) the narrow bony channel of the vestibular nerve and anterior vestibular artery that are more susceptible to entrapment. On the contrary, the inferior vestibular nerve that supplied

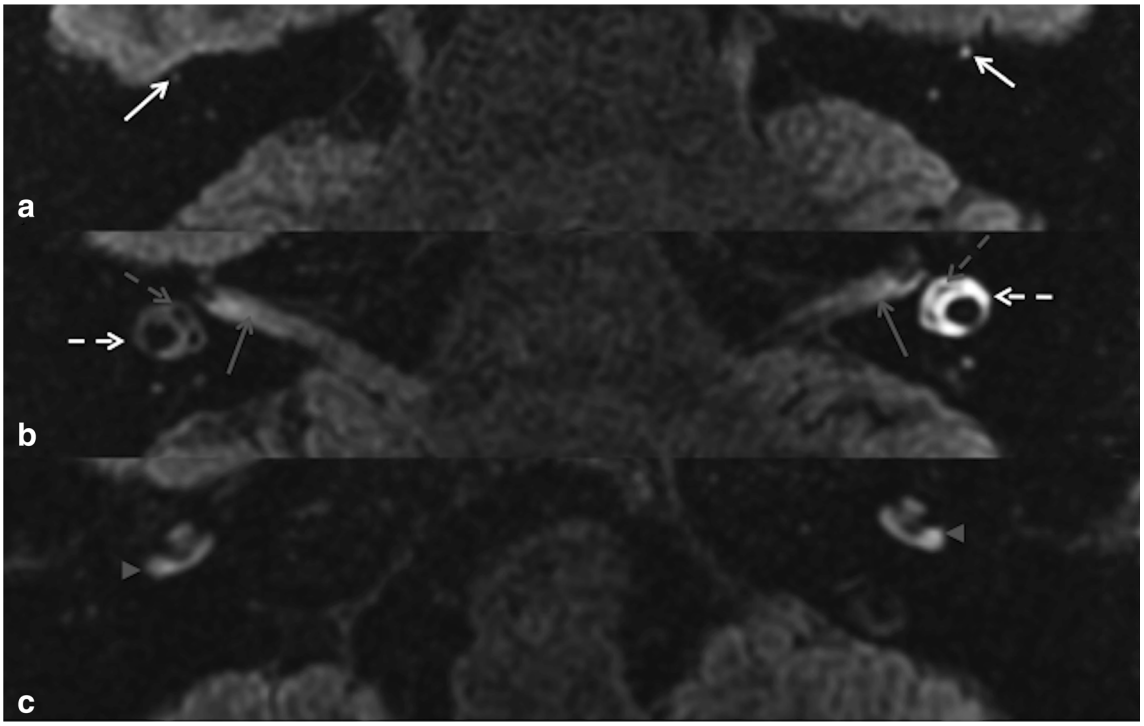

Fig. 2 A 48-year-old female with left acute vestibular syndrome. **a** Axial 3D-FLAIR at the level of the superior semicircular canals (white arrow) showed a slight increased enhancement of the left superior semicircular canal. **b** Axial 3D-FLAIR at the level of the utricle (gray dotted arrow) demonstrated an increased enhancement of the left lateral semicircular canals (white dotted arrow). There is a symmetrical enhancement of both superior vestibular nerves (gray arrow). **c** Axial 3D-FLAIR at the level of the cochlea showed a symmetrical enhancement of both cochlea (gray arrowhead)

the saccule and the posterior semicircular canal is rarely (3%) involved [21].

Detection of labyrinthine abnormalities with MRI in UAVS patients

3D-FLAIR sequences with variable flip-angle refocusing have the advantage of increasing echo train length, enabling a shorter acquisition time without blurring. These sequences are more sensitive than conventional FLAIR sequences to detect T1-shortening [22]. 3D-FLAIR sequences also enable the assessment of the cochlear BLB in various otological diseases 4 h after the intravenous administration of gadolinium, yet the assessment of the vestibular BLB has never been previously showed after a single-dose intravenous administration of gadolinium. Byun et al [4] assessed the vestibular BLB permeability after a double-dose administration of Gd-diethylenetriamine penta-acid that presents closely similar physiochemical properties with gadoterate meglumine. It has been reported that a single dose of gadobutrol is superior to a double-dose of gadoterate meglumine in inner MRI exploration because the concentration of gadobutrol is twice as high and because the longitudinal relaxivity (r1) is higher [9].

Byun et al found an asymmetrical enhancement of the vestibular nerve in 69% of patients and of the labyrinth in 41% of patients and have suggested that UAVS is caused by the vestibular nerve inflammation spreading to the perilymph of the vestibule and the semicircular canals. Here, none of our patients presented an asymmetrical enhancement of the vestibular nerve, yet the average delay between the onset of the crisis and MR imaging was 24.7 days, while MRI was performed within a week from the onset in the study of Byun et al. In addition, since our assessment was qualitative, we cannot exclude that both nerves were actually slightly enhanced after the injection; we only reported the visual absence of any such signs (asymmetrical enhancement or evidence of strong nerve enhancement) in our cohort. The spontaneous high signal of nervous structures on non-enhanced 3D-FLAIR can make the diagnosis of local enhancement difficult. We would like to mention that five of our patients underwent MRI within the week after the onset and showed no evidence of an asymmetrical enhancement of the vestibular nerve, although they all presented with a semicircular asymmetrical enhancement.

Interestingly, six patients presented with high signal intensity of the cochlea with normal hearing thresholds. Like Byun et al, we believed that this cochlear high signal could be explained by the diffusion of gadolinium in the perilymphatic space, yet we could not exclude the possibility of a concurrent inner ear inflammation [4]. We could mention that only four of our patients presented no asymmetrical enhancement of the

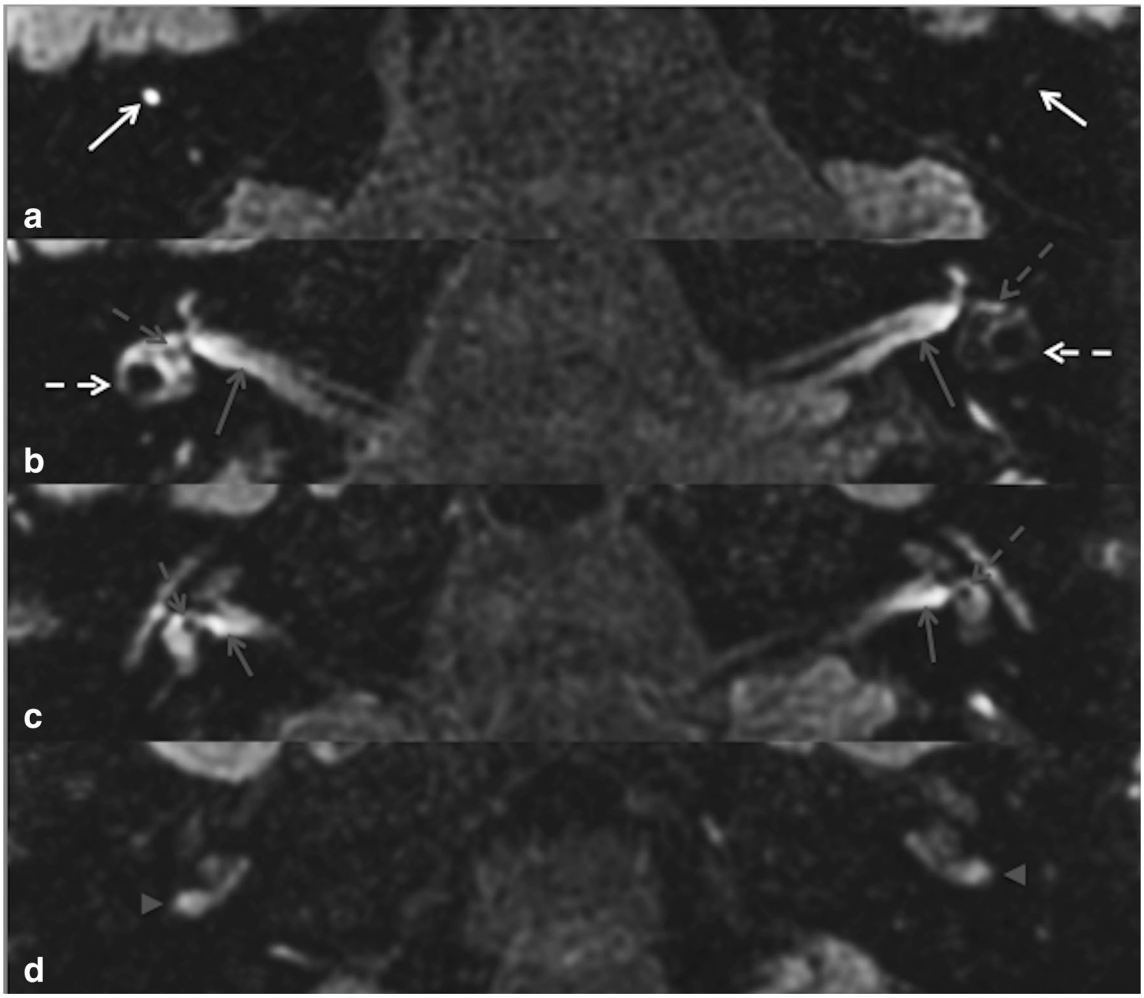

Fig. 3 A 66-year-old male with right acute vestibular syndrome. **a** Axial 3D-FLAIR at the level of the superior semicircular canals (white arrow) showed a bright enhancement of the right superior semicircular canal. **b** Axial 3D-FLAIR at the level of the utricle (gray dotted arrow) demonstrated a slight increased enhancement of the right lateral semicircular canal (white dotted arrow) in comparison to the left side. There is a symmetrical enhancement of both superior vestibular nerves (gray arrow). **c** Axial 3D-FLAIR at the level of the saccule (gray dotted arrow) demonstrated a slight increased enhancement of the vestibular perilymph surrounding the saccule in comparison to the left side. There is a symmetrical enhancement of both inferior vestibular nerves (gray arrow). **d** Axial 3D-FLAIR at the level of the cochlea (gray arrowhead) showed a symmetrical enhancement of both cochlea

labyrinthine structures on 3D-FLAIR sequence, although an MRI was performed between 20 and 50 days in these patients. We hypothesized that these four patients had a less severe BLB impairment, although canal paresis was profound in two of these four patients.

Interestingly, 23% of our patients showed a low signal on SSFP sequences involving the utricle. It has been reported that 10 to 15% of patients had benign paroxysmal positional vertigo (BPPV) secondary to UAVS. In these cases, BPPV was potentially caused by damage to the utricle that could detach otoconia entering the posterior semicircular canal duct [23]. We raised the hypothesis that patients with a low signal involving the utricle on SSFP sequences could develop BPPV, also known as Lindsay-Hemenway syndrome.

Potential therapeutic implications

UAVS is a common benign disease with an incidence of 3.5 cases per 100,000 people [24] and affects patients between 30 and 60 years old. Nearly half of these patients experienced chronic dizziness and unsteadiness [25], which have a significant effect on quality of life and work. The treatment is based on corticosteroids and/or antiviral therapy because animal models demonstrated an enhancement of the vestibular compensation in response to the activation of glucocorticoid receptors [26]. As in our study, it has been supposed that UAVS is an intralabyrinthine pathology, rather than a pathology involving the nerve. The authors have stated that an intravenous administration of corticosteroids is not sufficient [27], and they have suggested that an intratympanic administration of

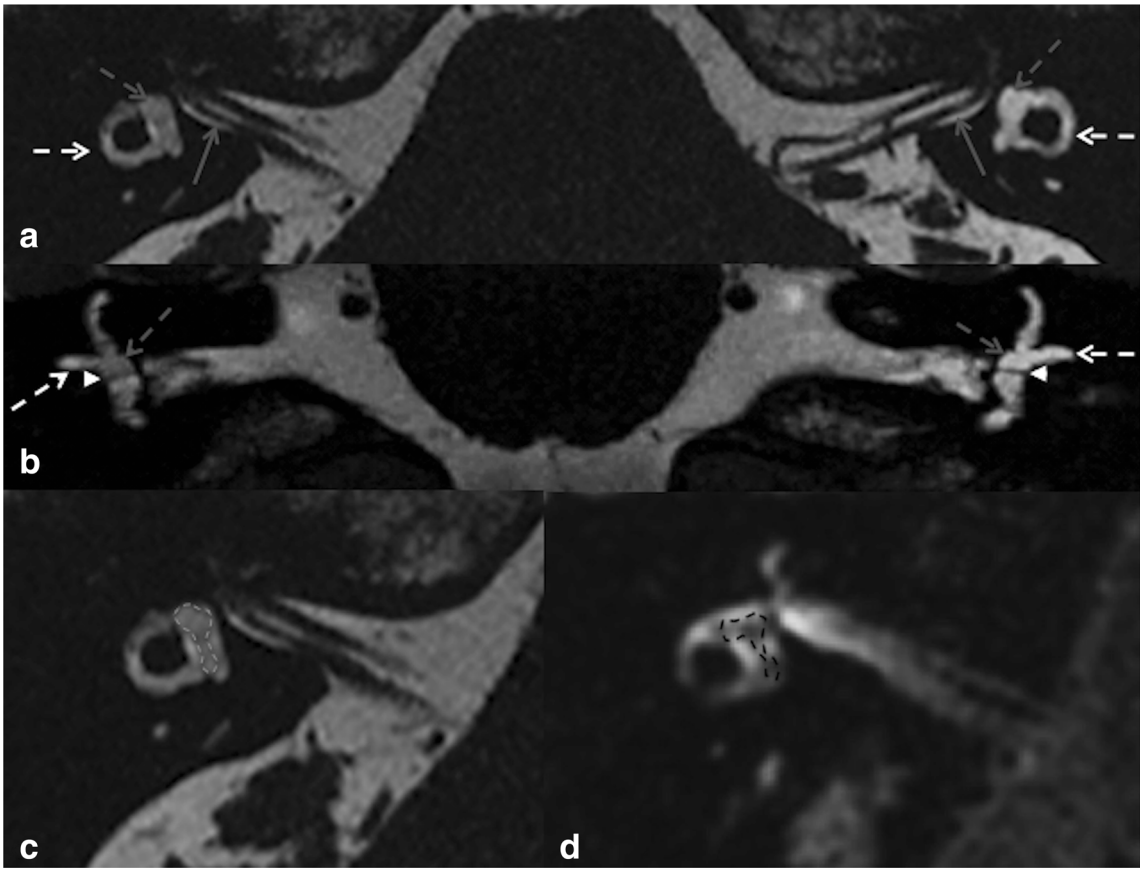

Fig. 4 A 66-year-old male with right acute vestibular syndrome (the same patient as in Fig. 3). **a** Axial SSFP sequence at the level of the lateral semicircular canals (white dotted arrow) and the superior vestibular nerves (gray arrow) demonstrated a low signal involving the right utricle (gray dotted arrow). **b** Coronal SSFP sequence at the level of the superior and lateral semicircular canals (white dotted arrow) showed a low signal involving the right utricle (gray dotted arrow) located above the utricular macula (white arrowhead). **c** Axial SSFP sequence and **d** axial 3D-FLAIR at the same level through the utricle. The low-signal area on the 3D FLAIR sequence (**d**) corresponds to the low-signal area on T2-weighted sequence

corticosteroids would be more useful, reaching a higher level of concentration in the inner ear such as is already performed in patients with sudden sensorineural hearing loss [28]. As UAVS seems to be an intralabyrinthine pathology that associates a marked enhancement and a low signal on SSFP sequence, a follow-up should be performed due to the high risk of semicircular canal fibrosis as it is observed in labyrinthitis at a later stage [29].

Sometimes, the distinction between the vestibular form of Menière's disease (vertigo without cochlear symptoms) and UAVS can be a challenge to the clinicians. MRI could enable a better management of those patients showing a vestibular BLB impairment in cases of UAVS on one hand, and endolymphatic hydrops in cases of Menière's disease on the other hand. Furthermore, the absence of endolymphatic hydrops or BLB impairment on MRI could refute a peripheral origin and suggests a central origin. Since infarctions involving the vestibular nucleus or the cerebellum [30] can mimic UAVS, MRI is usually performed to assess the brain, mostly the vestibular central pathways. Rarely, peripheral vestibulopathy may progress, involving the territory supplied by the anterior inferior cerebellar artery [31].

Limitations of this study

The main problem we encountered was the impossibility to perform a quantitative assessment based on the region of interest method [7, 8], since the signal intensity ratio was different between each MRI vendor. Yet, data inclusions coming from different machines add value and expand this protocol.

Although rare, inferior vestibular neuritis could not be included because of the design of this study, since the caloric tests only assess the lateral semicircular canal. Further studies, including a video head impulse test in the diagnosis of UAVS, would allow including inferior vestibular neuritis.

Another limitation is that MRI was obtained without pre-contrast 3D-FLAIR sequences that can be related to proteinaceous contents as observed in intralabyrinthine hemorrhage. However, pre-contrast FLAIR hyper-intensity has never been described in UAVS; thus, we suggested that the BLB impairment in patients with UAVS was not influenced by pre-

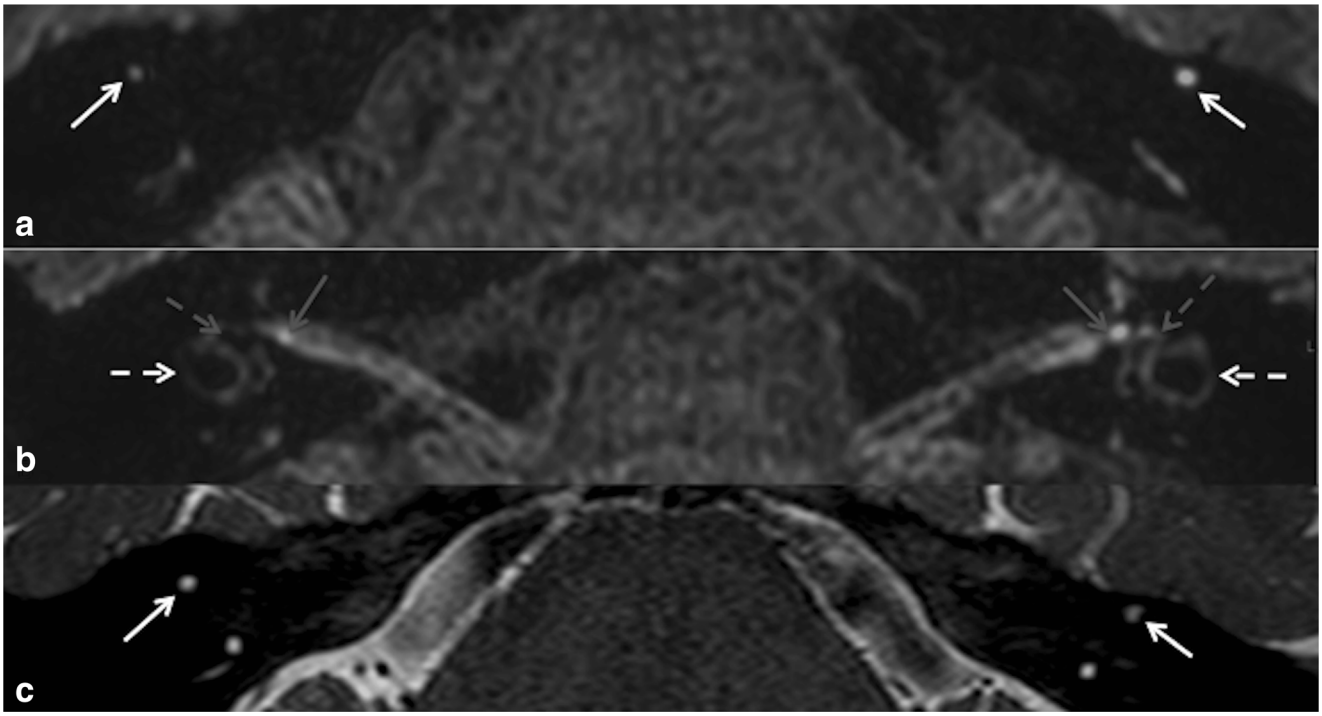

Fig. 5 A 75-year-old male with left acute vestibular syndrome. **a** Axial 3D-FLAIR at the level of the superior semicircular canals (white arrow) showed a bright enhancement of the left superior semicircular canal. **b** Axial 3D-FLAIR at the level of the utricle (gray dotted arrow) and the lateral semicircular canal (white dotted arrow) demonstrated a symmetrical increased enhancement of the superior vestibular nerves (gray arrow). **c** Axial SSFP sequence showed a low signal involving the left superior semicircular canal (white arrow)

contrast asymmetries [4]. In addition, the intensity of the signal of the inner ear structures appears typical of contrast medium effect.

More importantly, all patients received systemic corticosteroids before their MRI that could have modified the vestibular BLB permeability on MRI. Indeed, it is well known that corticosteroids have a positive effect on the labyrinthine blood flow [32]. Nevertheless, in most studies, patients with sensorineural hearing loss underwent corticosteroids before MRI, and an asymmetric high signal in the affected cochlea was still observed in nearly 80% of these patients [33]. We do not believe that the asymmetric nerve enhancement disappeared because of the treatment, since we observed an asymmetric labyrinthine enhancement. Most of our patients underwent MRI within 30 days after the onset of symptoms, yet it has been reported that high-intensity signals disappear 90–150 days after the onset of idiopathic sudden sensorineural hearing loss [34]. Our findings are preliminary and discordant with other sparse comparable data, which need to be addressed in further studies.

Our study showed asymmetrical findings in the labyrinths between sides, whereas symmetrical findings were found in extralabyrinthine structures between sides. We demonstrated that 86% of patients presented with an asymmetrical enhancement of the semicircular canals, as evaluated with delayed post-contrast 3D-FLAIR.

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Compliance with ethical standards

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Conflict of interest The authors of this manuscript declare no relationships with any companies, whose products or services may be related to the subject matter of the article.

Statistics and biometry One of the authors has significant statistical expertise.

Informed consent Written informed consent was waived by the Institutional Review Board.

Ethical approval Institutional Review Board approval was obtained.

Study subjects or cohorts overlap Some study subjects or cohorts have been previously reported in Attyé et al, Eur Radiol 2017, https://doi.org/10.1007/s00330-016-4701-z.

Methodology
- retrospective
- case-control study
- multicenter study

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