Prompt Contrast Enhancement of Cerebrospinal Fluid Space in the Fundus of the Internal Auditory Canal: Observations in Patients with Meningeal Diseases on 3D-FLAIR Images at 3 Tesla

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We speculated that meningeal pathologies might facilitate the permeability of cranial nerves at the fundus of the internal auditory canal (IAC), causing prompt enhancement after administration of Gd-DTPA. Using a 3D-fluid-attenuated inversion recovery (FLAIR) sequence, we evaluated the enhancement of the cerebrospinal fluid (CSF) space in the IAC fundus 10 min after Gd-DTPA administration in patients with meningeal diseases. Twenty patients (aged 22 to 79 years) were divided into 2 groups, a group with meningeal disease comprising 9 patients with meningeal abnormalities (6, tumor dissemination; 3, infection) and a control group of 11 patients with unilateral IAC pathology whose healthy sides were included as controls. Six of the 9 patients in the group with meningeal disease showed bilateral enhancement; one showed unilateral enhancement. None of the control group showed enhancement in the healthy side. One patient with Ramsay-Hunt syndrome showed only ipsilateral enhancement. Enhancement in the IAC fundus was frequently observed in patients with meningeal disease, even just 10 min after administration of contrast agent. This enhancement in the IAC fundus was never visible on T1-weighted 3D-FLASH images.

Keywords: magnetic resonance, contrast enhancement, internal auditory canal, FLAIR, 3D

Introduction

The clinical utility of the fluid-attenuated inversion recovery (FLAIR) sequence has been reported,1,2 and combining FLAIR with fast spin echo has further improved FLAIR’s clinical utility.3–8 Compared to 2D-FLAIR, a 3D-FLAIR sequence using a nonselective inversion pulse allows thinner slices while avoiding cerebrospinal fluid (CSF) motion artifacts.9,10 FLAIR is sensitive to abnormalities in the subarachnoid space3 and is able to detect subtle contrast enhancement in CSF space after intravenous administration of Gd-DTPA under various disease conditions.11–13

Contrast enhancement has been observed on 3D-FLAIR images at 3T in the basal turn of the cochlea 4 hours after intravenous Gd-DTPA administration in healthy subjects.14 In that study, none of the subjects showed visible enhancement at 10 min after Gd-DTPA administration. It was speculated that the permeability to Gd-DTPA in the cochlea was quite small.14

Although the permeability of the peripheral part of the nerve in the fundus of the internal auditory canal (IAC) is unknown, we had a patient with meningitis carcinomatosa who showed strong enhancement at 10 min after Gd-DTPA administration. We speculated that meningeal pathologies might facilitate the permeability of the cranial nerve at the fundus of the IAC, resulting in prompt enhancement even 10 min after Gd-DTPA administration. The purpose of this study was to evaluate the enhancement of CSF space in the IAC fundus 10 min after Gd-DTPA administration using a 3D-FLAIR sequence at 3T in patients with various
meningeal diseases.

Materials and Methods

Patients

Twenty patients (13 men, 7 women, aged 22 to 79 years, mean 52.1 years) with intracranial diseases were assigned to 2 groups, one comprising consecutive patients with meningeal abnormalities diagnosed by CSF analysis (meningeal disease group; 9 patients) and the second comprising patients with unilateral pathologies around the IAC or labyrinth (11 patients). The contralateral (healthy) sides of this group of patients were included as the control group.

The meningeal disease group consisted of 6 patients with meningeal dissemination (three from lung cancer, one from esophageal cancer, one from germinoma, and one from astrocytoma) and 3 patients with infectious meningitis (two with cryptococcus meningitis and one with presumed viral meningitis). The control group consisted of one patient with unilateral Ramsay-Hunt syndrome, two with Bell's palsy, seven with unilateral sudden sensorineural hearing loss, and one with subacute infarction in the right side of the pons. Written informed consent was obtained from all subjects. This study was approved by the institutional review board of our university hospital.

MR imaging

All scans were performed on a 3T MR imager (Trio, Siemens; Erlangen, Germany) using a receive-only, 8-channel phased array. Both T1-weighted 3D-FLASH (fast low-angle shot) and 3D-FLAIR (fluid-attenuated inversion recovery) images were acquired before and after intravenous administration of a single dose of Gadolinium-diethylene-triamine pentaacetic acid-bis(methylamide) (Gd-DTPA-BMA; Omniscan®, Daichi Pharmaceutical Co., Ltd., Tokyo, Japan) at 0.1 mmol/kg. Imaging with 3D-CISS (constructive interference in the steady state) sequence was performed only before the administration of contrast material to provide an anatomical reference for CSF space. The parameters for T1-weighted 3D-FLASH were: repetition time (TR) of 6.93 ms with water-selective excitation, echo time (TE) of 3.29 ms, flip angle (FA) of 8 degrees with RF spoiling, matrix size of 256×256, 96 axial 0.8-mm slice thickness with a 16-cm square field of view (FOV). The number of excitations (NEX) was two, giving a total scan time of 3 min 26 s. The center of k-space was acquired one second after initiation of the sequence.

The parameters for 3D-CISS were TR = 5.91 ms, TE = 2.96 ms, FA = 50 degrees, matrix size = 256×256, 72 axial 0.8-mm slice thickness with a 14-cm square FOV, and 25% phase oversampling. NEX was one, and the scan time was 3 min 12 s.

Parameters for 3D-FLAIR were TR = 9000 ms; effective TE = 638 ms; turbo spin-echo refocusing pulse train with varying FA (average of 151 degrees); echo spacing of 3.64 ms and 171 echoes in the echo-train; matrix size = 384×384; and acceleration factor of two using the parallel imaging technique.15 Forty-eight axial 0.8-mm-thick slices were used to cover the labyrinth with a 25.6-cm square FOV, giving a voxel size of 0.7×0.7×0.8 mm. The inversion pulse was nonselective, excitation pulse was slab selective, and readout bandwidth was 592 Hz/pixel. NEX was 2, giving a total scan time of 5 min 26 s. The features of this variable FA sequence have been reported elsewhere.10,16,17 It allows the use of very long echo-train lengths in the range of approximately 150 to 220 echoes while maintaining contrast similar to that of conventional 2D fast-FLAIR with no significant blurring, even with relatively long effective echo times.

3D-FLAIR and -FLASH images were obtained before and after the administration of a single dose of Gd-DTPA-BMA (0.1 mmol/kg). After contrast administration, 3D-FLASH was scanned first followed by contrast-enhanced 3D-FLAIR initiated 7 min after Gd administration such that the middle of the 3D-FLAIR sequence would occur approximately 10 min after Gd administration.

Image evaluation

Images were evaluated independently by 2 radiologists; if there were any discrepancies between the two, consensus was obtained through discussion. Contrast enhancement of CSF space in the fundus of the IAC was taken to be positive if all 3 of the following conditions were fulfilled: (1) There was no mass other than the cranial nerves in the CSF space of the IAC fundus on 3D-CISS and pre-and post-contrast-enhanced 3D-FLASH images. (2) The intensity of CSF space of the IAC fundus was lower than that of the cranial nerves on post-contrast 3D-FLAIR images. (3) The intensity of CSF space of the IAC fundus was higher than that of the cranial nerves on post-contrast 3D-FLAIR images.

Results

In the group with meningeal disease (9 patients), six showed bilateral enhancement (three with lung cancer and one each with esophageal cancer, astrocytoma, and cryptococcus); one patient showed unilateral enhancement (presumed viral meningi-
Fig. 1. A 54-year-old woman with Ramsay-Hunt syndrome on the left side. No mass lesion was seen in the cerebrospinal fluid (CSF) space at the internal auditory canal (IAC) fundus between the cochlear nerve and the inferior vestibular nerve on this 3D-CISS (constructive interference in the steady state; arrow, a). No area of abnormal signal was seen on pre-contrast 3D-FLAIR (fluid-attenuated inversion recovery; b) and 3D T₁-weighted images (c). Enhancement was found on post-contrast 3D-FLAIR images (arrow, d); however, no enhancement was visible on post-contrast 3D-T₁-weighted images (arrow, e). In the control side (normal right side), no enhancement was observed.

Discussion

It has been reported that FLAIR imaging can detect a concentration of Gd 4 times lower than that detectable by T₁-weighted imaging. In a normal dog, Gd concentration in CSF measured one hour after intravenous Gd administration was 0.007 mmol/L, well higher than the detection threshold (0.000061 mmol/L) of FLAIR. However, the local Gd concentration of the IAC in CSF cannot be measured easily. From the results of the present study at least, 10 min after contrast injection, Gd concentration in the IAC fundus of most patients with meningeal disease was higher than in other CSF space, such as the CP angle cistern or the prepontine cistern, where the signal on post-contrast 3D-FLAIR remained as low as that on pre-contrast 3D-FLAIR.

CSF motion artifacts can cause high signal in CSF space on 2D-FLAIR images. However, 3D-FLAIR sequence is insensitive to CSF motion artifacts, so any signal increase after Gd administration can be attributed to Gd leakage into CSF space.

Contrast enhancement in CSF space has been reported in various conditions, such as stroke, tumor, postoperative state, and familial amyloid poly-
Fig. 2. A 61-year-old man with advanced esophageal cancer. This post-contrast-enhanced 3D T₁-weighted image (a) showed enhancement of the subarachnoid space (short arrow), but enhancement in the cerebrospinal fluid (CSF) space of the internal auditory canal (IAC) fundus was barely visible (long arrow). On post-contrast 3D-FLAIR (fluid-attenuated inversion recovery; b), these enhancements were more clearly visualized (short and long arrows). The cochlear fluid signal on the left side was also enhanced.

neuropathy. All previously reported enhancements were observed more than one hour after the administration of Gd. Enhancement in CSF space after only 10 min has not been reported.

In the present study, contrast enhancement in the CSF space of the IAC fundus was observed in seven of 9 patients just 10 min after intravenous Gd injection in the patients with meningeal disease. One patient with germinoma and one with criptococcus meningitis who did not show enhancement had already received successful treatment, and their disease was not aggressive at the time of MR examination.

The present study differed with those previous in scan timing and in having more patients with meningeal disease and thinner 3D-FLAIR slices to evaluate minute areas in the IAC and in using a field strength of 3T.

A higher magnetic field provides increased sensitivity to Gd and can provide a higher signal-to-noise (SNR) ratio. This higher SNR enables the use of thinner slices in FLAIR imaging, such as the 0.8-mm thickness in the present study.

It has been reported that CSF in the fundus of the IAC in healthy human subjects was enhanced 4 hours after Gd injection. In that study, imaging after 2 hours of Gd injection showed slight Gd leakage around the cochlear and vestibular nerve bundles in the fundus of the IAC; thus, we speculate that the CSF enhancement in the IAC fundus in the present study can be attributed to prompt leakage of Gd from cranial nerve bundles as the result of a pathological condition such as meningitis or Ramsay-Hunt syndrome.

In tuberculous meningitis, an increased concentration of vascular endothelial growth factor (VEGF) in the CSF stimulates disruption of the blood-brain barrier. In the cases of meningitis reported here, we speculate that a chemical mediator, such as VEGF, that was brought onto by the meningeal disease, might have disrupted the blood-nerve barrier in the fundus of the IAC.

However, we cannot conclude that enhancement in the IAC fundus was caused only by Gd leakage into CSF induced by a chemical mediator; it might also result from the presence of microscopic meningeal foci around the cranial nerve because they are invisible on post-contrast 3D-FLASH T₁-weighted images.

In the case of Ramsay-Hunt syndrome, active inflammation in response to viral infection, causing facial nerve palsy, hearing loss, and vertigo, might have influenced the permeability of the nerve.

The clinical relevance of this enhancement in the IAC fundus still requires further investigation, especially to confirm whether it can be used to monitor the progression of meningeal disease during therapy.

References
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