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ORIGINAL RESEARCH

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Enhanced fallopian canal as a potential marker for temporal bone vasculitis

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Abstract

Objectives: This study aimed to test the hypothesis that contrast-enhanced 3D MRI with gradient-echo sequences (CE-3D-GRE) can detect signs of vasculitis in the fallopian canal, which may cause otologic involvement, in four patients with antineutrophil cytoplasmic antibody-associated vasculitis (AAV).

Methods: CE-3D-GRE acquired at 3.0 Tesla was performed on four patients diagnosed with granulomatosis with polyangiitis or eosinophilic granulomatosis with polyangiitis, at onset or relapse of the disease, and in remission. Clinical correlations between otologic symptoms and radiological findings were examined for each patient. Furthermore, signal intensity of the mastoid segment of the fallopian canal was compared between the ears with active disease (n = 3) and those in remission or without vasculitis (n = 3).

Results: Intense enhancement in the tympanic and mastoid segments of the fallopian canal was associated with development of external otitis, otitis media, and sensorineural hearing loss, and was unrelated to the presence of facial paresis. Maximal intensity projection images visualized the close relationship between the enhanced fallopian canal and middle ear inflammation. The findings were absent in remission. Signal intensity of the mastoid segment of the fallopian canal was higher in ears with active disease than in normal ears ($P < .001$) and decreased to normal levels during remission ($P = .597$).

Conclusion: CE-3D-GRE can demonstrate vasculitis in the temporal bone, reflecting disease activity and the severity of otologic manifestations, including cochlear involvement, in AAV patients. Intense enhancement of the fallopian canal on CE-3D-GRE can be a potential marker for vasculitis of the temporal bone.

Level of Evidence: 5.

KEYWORDS
3D MRI, eosinophilic granulomatosis with polyangiitis, granulomatosis with polyangiitis, otologic involvement, the fallopian canal

1 | INTRODUCTION

Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) is a necrotizing vasculitis that predominantly affects small-size vessels of any organ.[1] Among AAV, granulomatosis with polyangiitis (GPA) and eosinophilic granulomatosis with polyangiitis (EGPA) frequently affect the ear to develop various otologic manifestations, including external otitis, otitis media, sensorineural hearing loss (SNHL), and facial paresis.[2,3] Moreover, otologic manifestations may occasionally be the first and only signs before progression to multiple systemic involvement.[4] Hence, early diagnosis of otologic involvement is important to prevent irreversible hearing loss and other long-term complications caused by systemic AAV.[5]

Despite the unique clinical features of AAV, there are several issues with management of otologic involvement. First, frequent otologic manifestations of AAV are commonly seen in other inflammatory ear diseases.[3,6] Next, the use of imaging has been limited to detecting otomastoiditis and granulomatous lesions secondary to vasculitis due to the small size of vessels affected.[6] Lastly, biopsy specimens from the ear never provide histological confirmation of AAV, except for those taken from the mastoid cavity via a surgical approach.[7,8] Given the lack of clinicopathologic correlation, the diagnosis and monitoring of otologic involvement of AAV is occasionally difficult, especially in ear-limited variants or ANCA-negative AAV.

Temporal bone pathology of GPA shows evidence of vasculitis in the fallopian canal with and without the clinical presentation of facial paresis.[9] Furthermore, the stylomastoid artery (SMA), one of the main arteries in the fallopian canal, supplies the posterior half of the external ear canal, tympanic cavity, mastoid, and facial nerve,[10] which are in line with frequent sites affected in otologic involvement of GPA and EGPA. These facts indicate that the fallopian canal is one of the predilection sites of vasculitis in the temporal bone. In the present study, using contrast-enhanced, 3D T1-weighted, magnetic resonance imaging (MRI) with gradient-echo sequences (CE-3D-GRE),[11,12] we tested the hypothesis that this imaging protocol could be utilized to detect signs of vasculitis in the fallopian canal, which may cause otologic involvement in AAV patients. We further discussed the possible pathophysiology of otologic manifestations in relation to vascular anatomy of the temporal bone.

2 | MATERIALS AND METHODS

The current study was conducted with Institutional Review Board approval from Tokyo Medical and Dental University for Clinical Research (M2018-136). All patients included in this report were provided informed consent for publication.

2.1 | Subjects

We prospectively performed CE-3D-GRE at the onset or at relapse of the disease, and during remission, on four AAV patients who presented with otologic manifestations as major initial symptoms. Diagnosis for GPA was made based on the European Medicines Agency (EMEA) vasculitis classification algorithm of 2007 as it requires characteristic symptoms of the respiratory tract suggestive of GPA, including otologic involvement, and positive serology for ANCA, and does not rely on histological confirmation for diagnosis.[13] Diagnosis of EGPA was made in accordance with the 1990 American College of Rheumatology (ACR) criteria.[14] Further, we recruited three patients with acoustic schwannoma who underwent CE-3D-GRE during the same period as age- and gender-matched control subjects (median age 55 [range: 50-58] years; two males and one female).

2.2 | Scanning protocol of CE-3D-GRE

A dedicated 8-channel head coil was used on a 3.0 Tesla MRI scanner, with the 3D fast-spoiled gradient-echo sequence (SPGR) (Signa HDxt; GE Healthcare, Waukesha, Wisconsin) or with the 3D fast-field echo sequence (FFE) with fat-suppression (Ingenia CX; Philips Healthcare, Best, the Netherlands), depending on the method in the institute where the imaging was performed. The parameters for SPGR were as follows: TR/TE = 7.4/2.8 ms; flip angle, 20°; field of view, 20 × 20 cm; matrix size, 288 × 256; section thickness, 1.5 mm; slice spacing, 1.5 mm; and acquisition time, 110 seconds. The parameters for FFE were as follows: TR/TE = 16.0/4.6 ms; flip angle, 20°; field of view, 22 × 22 cm; matrix size, 448 × 448; section thickness, 1.0 mm; slice spacing, 0.5 mm; and acquisition time, 218 seconds. Contrast-enhanced images were acquired immediately after an intravenous bolus injection of 0.1 mL/kg gadobutrol (Gadovist 1.0; Bayer Schering Pharma, Berlin, Germany) or 0.2 mL/kg meglumine gadoterate (Magnescope; Guerbet, Tokyo, Japan).

2.3 | Signal intensity assessment of the facial nerve

Multiplanner reconstruction and maximum intensity projection (MIP) images were obtained using RadiAnt DICOM Viewer (Medixant, Poznan, Poland). Anatomical segments of the facial nerve were identified in accordance with the reported method,[15] and the signal intensity of each segment was assigned a value as follows: 0, no signal; 1, faint visualization; 2, signal equivalent to normal cerebellum; 3, signal equivalent to enhanced dural sinus.[12]

2.4 | Comparison of signal intensity to other disease status

Signal intensity of the mastoid segment of the facial nerve was compared between before and after remission induction in vasculitis-involved ears (case 1, case 2, and case 4), and between normal control ears and vasculitis-involved ears. Four otolaryngologists, two rheumatologists, and one radiologist were blinded to the clinical information and scored each individual case independently in a single session. Statistical differences were assessed using Mann-Whitney *U* test or Wilcoxon signed-rank test.

3 | RESULTS

Signal intensity of the facial nerve was summarized for each segment in Table 1.

3.1 | Case 1

3.1.1 | Clinical history

A 43-year-old woman presented with symptoms of bilateral progressive hearing loss with postauricular pain, headache, unsteadiness, and cough. The patient had bilateral otitis media with external otitis, rhinosinusitis, normal facial nerve function, and an elevated level of myeloperoxidase-ANCA (MPO-ANCA) to 49.9 U/mL. She was diagnosed with GPA and underwent methylprednisolone pulse therapy (1 g per day) and following intermittent intravenous cyclophosphamide therapy, with a prompt resolution of the symptoms. However, with attempted taper of prednisolone to 20 mg per day, the disease relapsed with a left-sided sensorineural hearing loss (SNHL) with external otitis of the posterior canal wall (Figure S1), and severe cough. Considering the systemic flare of GPA, re-induction therapy was initiated with methylprednisolone pulse and a single course of rituximab (500 mg once a week for 4 weeks) followed by four courses of 500 mg rituximab as maintenance therapy. At the most recent follow-up, she was in remission with normal hearing on the audiogram (Figure S1) and on 5 mg prednisolone per day.

3.1.2 | MRI findings

While the patient was in relapse and receiving 15 mg prednisolone daily, CE-3D-GRE showed that the tympanic (Figure 1A) and mastoid (Figure 1B) segments of the left facial nerve were intensely enhanced, which was associated with inflammation in the left mastoid tip (Figure 1B). Proximal segments of the facial nerve, including the geniculate ganglion, were faintly enhanced or not visualized. In remission, bilateral facial nerves were faintly enhanced (Figure 1C).

3.2 | Case 2

3.2.1 | Clinical history

A 68-year-old man had a long history of left-sided otitis media from his youth. He developed exacerbation of hearing loss with persistent aural discharge on the left and walking difficulty over the prior 3 months. Examination revealed otitis media with extruding granuloma with a profound mixed hearing loss on audiometry (Figure S1) and complete canal paresis on the left. Facial nerve function was normal. Laboratory studies were significant for an elevated level of proteinase 3-ANCA (PR3-ANCA) to 50.8 U/mL. He was diagnosed with GPA and placed on a regimen of high-dose prednisolone combined with 7.5 mg methotrexate. Methotrexate was terminated due to a renal adverse event. At the most recent follow-up, he was in remission and taking 5 mg prednisolone per day, but the hearing loss persisted (Figure S1).

3.2.2 | MRI findings

Before treatment, CE-3D-GRE showed intense enhancement in the tympanic (Figure 2A) and mastoid (Figure 2B) segments of the left facial nerve, which accompanied inflammation in the left tympanic cavity and mastoid. Bilateral geniculate ganglions were intensely enhanced with no obvious difference between them. More proximal segments were faintly enhanced. The MIP image clearly visualized the left fallopian canal along the course from the stylomastoid foramen to the geniculate ganglion, which was associated with inflammation in

TABLE 1 Signal intensity score of facial nerve segments in five affected ears

Segment	Signal intensity score				
	Case 1	Case 2	Case 3 right	Case 3 left	Case 4
Cisternal	0	1	1	1	0
Canalicular	1	1	0	0	0
Labyrinthine	1	1	1	1	1
Geniculate	1	3	3	3	3
Tympanic	3	3	3	3	3
Mastoid	3	3	3	3	3

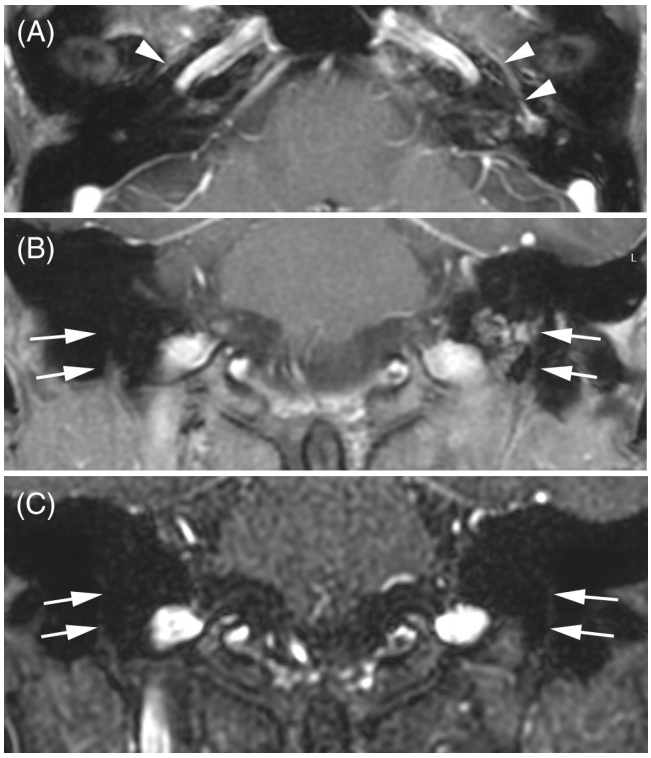

FIGURE 1 3D-gradient-echo images with contrast enhancement in case 1. A,B, In relapse, axial, A, and coronal, B, SPGR visualizes the tympanic (arrowheads) and mastoid (arrows) segment of the fallopian canal on both sides, respectively, with intense enhancement in the left. Inflammation is found in the tympanic cavity and mastoid tip in the left. C, In remission, coronal fat-suppressed FFE shows bilateral mastoid segments (arrows) in faint signal intensity

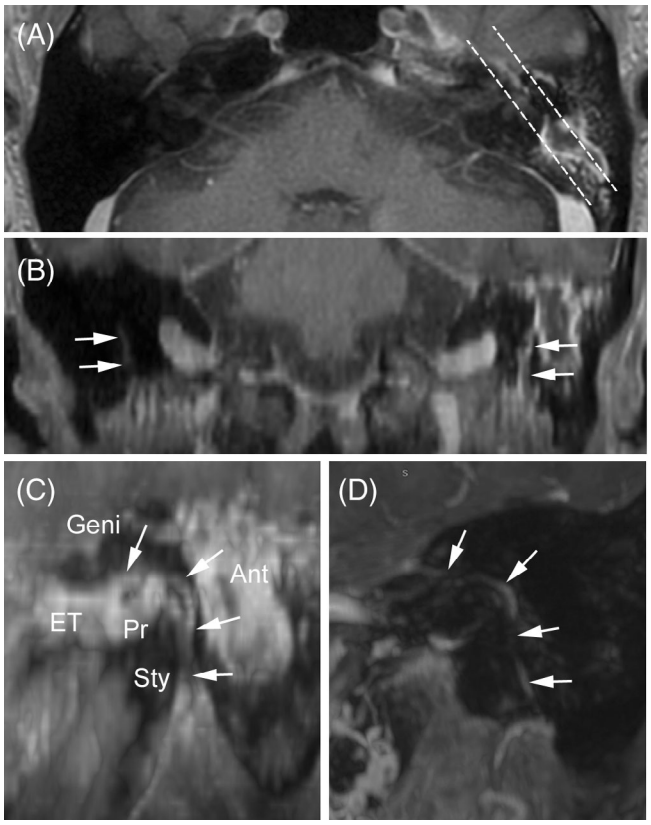

FIGURE 2 3D-gradient-echo images with contrast enhancement in case 2. A,B, Axial, A, and coronal, B, SPGR visualizes the fallopian canal (arrows) with intense enhancement in the left at onset. C, Maximal intensity projection (MIP) image reconstructed between dashed lines in A. The left fallopian canal (arrows) is visualized from the stylomastoid foramen (Sty) to the geniculate ganglion (Geni), accompanying inflammation in the antrum (Ant), promontory (Pro), and eustachian tube (ET). D, MIP image of the left fallopian canal (arrows) in remission. The signal intensity of the tympanic and mastoid segments is attenuated

the antrum, promontory, and eustachian tube (Figure 2C). In remission, the left fallopian canal had a decreased signal intensity on the MIP image, except for the geniculate ganglion, which remained intensely enhanced (Figure 2D).

3.3 | Case 3

3.3.1 | Clinical history

A 77-year-old woman developed otitis media with effusion on the left. The patient was treated with ventilation tube insertion, but aseptic aural discharge was continued. Three months later, she complained of further exacerbation of hearing loss with retroauricular pain in the left ear and dizziness. Examination revealed left-sided granulomatous otitis media with serous discharge, and bilateral mixed hearing loss on audiometry (Figure S1). Laboratory studies were significant for an increased leukocyte count of 9440/mm^3, and elevated levels of immunoglobulin G to 2591 mg/dL, rheumatoid factor to 174 IU/mL, and MPO-ANCA to 112.8 U/mL. She was diagnosed with GPA and placed on a regimen of high-dose prednisolone and following intermittent intravenous cyclophosphamide therapy. She was currently free from otologic symptoms and taking 20 mg prednisolone combined with 1.5 mg tacrolimus.

3.3.2 | MRI findings

Before treatment, the tympanic (Figure 3A) and mastoid (Figure 3B) segments of bilateral facial nerves were visualized with intense enhancement on CE-3D-GRE, which were associated with inflammation in bilateral tympanic cavities and mastoid cavities with a left predominance (Figure 3B). Bilateral geniculate ganglions were intensely enhanced with no obvious difference between them. More proximal segments of the facial nerve were faintly enhanced or not visualized. On MIP, the fallopian canal was clearly visualized through the entire course both in the right (Figure 3C) and left (Figure 3D) and was accompanied by inflammation in the promontory. The epitympanic recess and eustachian tube were also involved in the left.

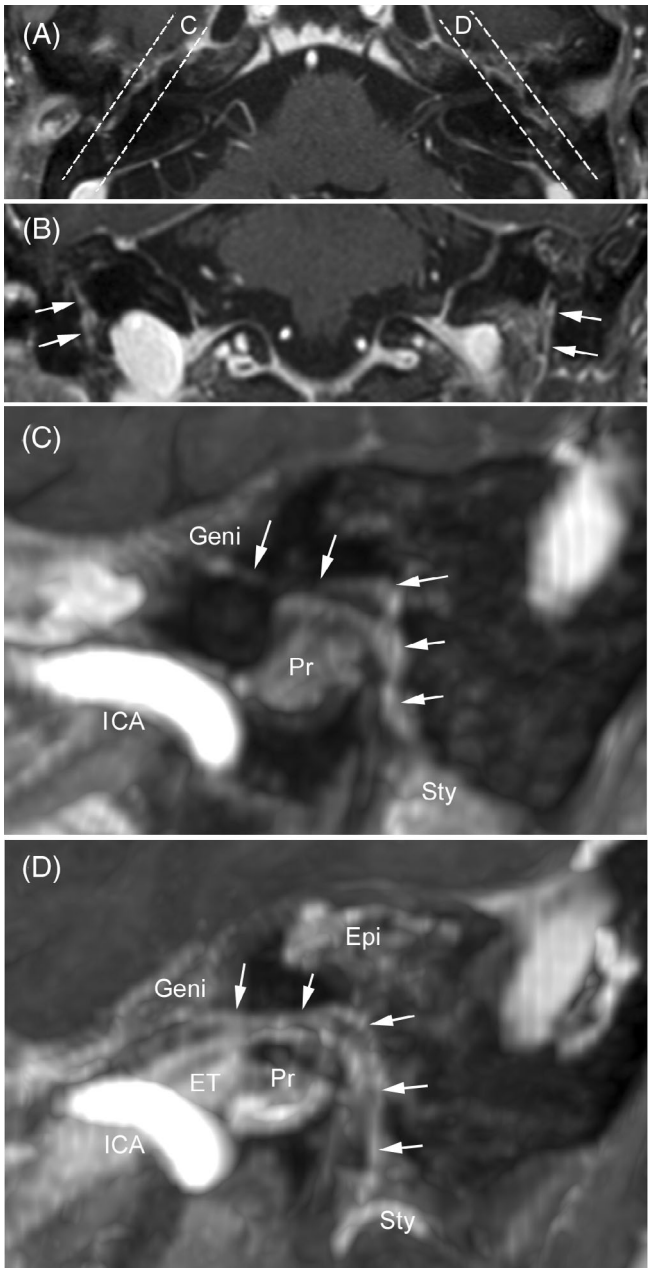

FIGURE 3 3D-gradient-echo images with contrast enhancement in case 3. A,B, In onset, axial, A, and coronal, B, SPGR show intense enhancement in bilateral tympanic and mastoid segments (arrows), respectively. C,D, Maximal intensity projection images reconstructed between dashed lines in A. The fallopian canal (arrows) is intensely enhanced both in the right, C, and left, D, which accompanies inflammation in the promontory (Pro) on both sides, and the eustachian tube (ET) and epitympanic recess (Epi) in the left. Geni, geniculate ganglion; ICA, internal carotid artery; Sty, stylomastoid foramen

3.4 | Case 4

3.4.1 | Clinical history

A 28-year-old man presented with bilateral hearing loss, unsteadiness, exacerbation of asthma, and normal facial nerve function. His medical history was significant for chronic rhinosinusitis with eosinophilic infiltrate and severe asthma from the age of 23 years, and bilateral otitis media, vertigo, headache, and left-sided facial paresis at 25 years of age, which were treated with oral steroid administration. Otoscopy revealed bulky granulomatous tissues extruding from the tympanic membrane and posterior wall of the external auditory canal with a thick, yellowish aural discharge in both ears. Audiometry demonstrated a mixed hearing loss on the right and a conductive hearing loss on the left. Examination of the aural discharge revealed eosinophilic infiltrate without bacterial infection. Laboratory studies showed peripheral eosinophilia and negative ANCA serology. In the absence of a definitive diagnosis of EGPA, a right-sided SNHL with external otitis relapsed three times following repeated taper of prednisolone (Figure S1). A computed tomography scan revealed bilateral transient pulmonary infiltrates. Neoplasm and other blood diseases were excluded with peripheral blood and bone marrow examinations. He was finally diagnosed with EGPA, and at the most recent follow-up, his general condition improved with a combined therapy of 150 mg azathioprine/16 mg prednisolone/mepolizumab but the hearing loss partially persisted (Figure S1).

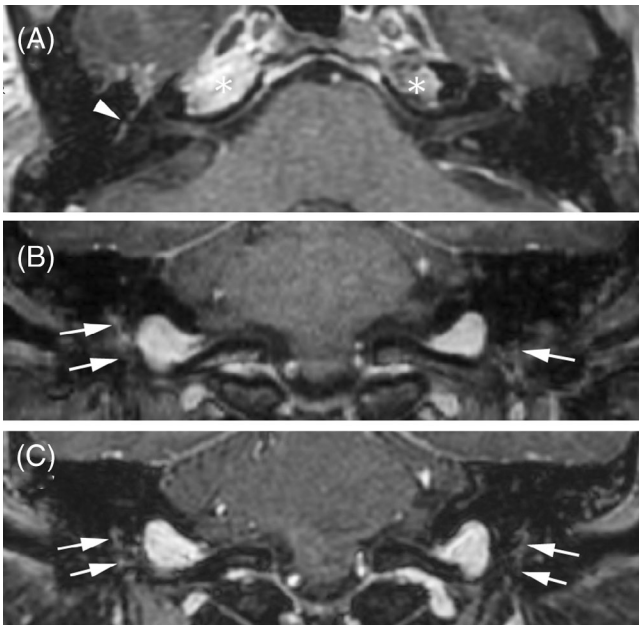

FIGURE 4 3D-gradient-echo images with contrast enhancement in case 4. A,B, In relapse, coronal SPGR shows the tympanic (arrowhead, A) and mastoid (arrows, B) segments, with moderate to intense enhancement in the right. Asterisks indicate fatty marrow at the petrous apex. C, In remission, coronal fat-suppressed FFE shows bilateral mastoid segments (arrows) in faint signal intensity

3.4.2 | MRI findings

While the patient was in relapse and receiving 50 mg prednisolone daily, CE-3D-GRE revealed moderate to intense enhancement in the tympanic (Figure 4A) and mastoid (Figure 4B) segments of the

right facial nerve. Other segments were faintly enhanced or not visualized. In remission, the signal intensity of the right mastoid segment was attenuated to similar level of that in the left (Figure 4C).

3.5 | Comparison of signal intensity to other disease status

The median signal intensity scores of the mastoid segment of the facial nerve in normal ears without vasculitis, involved ears with active disease, and involved ears in remission were 1 [0-3], 3 [1–3], and 1 [0-3], respectively (median [range]). Involved ears with active disease showed significantly higher signal intensity than normal ears (Figure 5, $P < .001$). Signal intensity of involved ears was decreased significantly in remission ($P < .001$), to a level similar to that of the normal ears ($P = .597$).

4 | DISCUSSION

In this prospective study, CE-3D-GRE was performed at 3.0 Tesla on four patients with otologic manifestations of AAV as the major initial symptoms. In all patients, intense enhancement in the tympanic and mastoid segments of the fallopian canal was associated with development of otologic symptoms, including external otitis, otitis media, and SNHL. Furthermore, MIP images visualized the close relationship between enhanced fallopian canal and inflammation in the middle ear, including the mastoid, promontory, and eustachian tube. Increased enhancement in the mastoid segment was attenuated to the normal level in accordance with clinical improvement after immunosuppressive treatment. The finding is novel and shows the feasibility of using CE-3D-GRE as a potential marker to demonstrate vasculitis of the temporal bone.

We utilized CE-3D-GRE for imaging signs of vasculitis in the fallopian canal because the method is common in clinical settings and able to visualize the vascularity inside the canal with greater signal intensity and higher contrast compared to conventional MRI.[11,12] The significant finding on CE-3D-GRE in this study was intense enhancement along the tympanic and mastoid segments of the facial nerve in affected ears compared with unaffected ears and other facial nerve segments. In pathological conditions, such as Ramsey-Hunt syndrome, the facial nerve is intensely enhanced because disruption of the blood-nerve barrier causes leakage and further accumulation of contrast material inside the fallopian canal.[16] The facial nerve occupies only 50% and 30% of the fallopian canal in the tympanic and mastoid segment, respectively, and is enclosed in large connective tissues,[17] which may serve as a large space for accumulation of contrast agents. Our results suggest CE-3D-GRE images intense enhancement in these segments due to vascular inflammation occurred in the tympanic and mastoid segments. Large connective tissues in the tympanic and mastoid segments may also serve as a reservoir that prevents serious ischemia and edema of the facial nerve secondary to vasculitis.[17] On the other hand, in more proximal segments, we only found normal enhancement due to the increased pool of contrast material in the intrinsic capillary plexus of the facial nerve.[11] Pathological evidence of vasculitis has not been reported in proximal segments, including the geniculate ganglion and internal auditory canal.

Our findings provide insights into the pathophysiology of vasculitis in the ear. The distribution of enhancement in the tympanic and mastoid segments is consistent with pathologic findings of vasculitis in the fallopian canal in patients with GPA[9] and polyarteritis nodosa,[18] which is also known to present with otitis media and facial paresis. The SMA enters the fallopian canal through the stylomastoid foramen and predominantly supplies the mastoid and tympanic segments, giving off numerous branches to the mastoid, tympanic cavity, and the

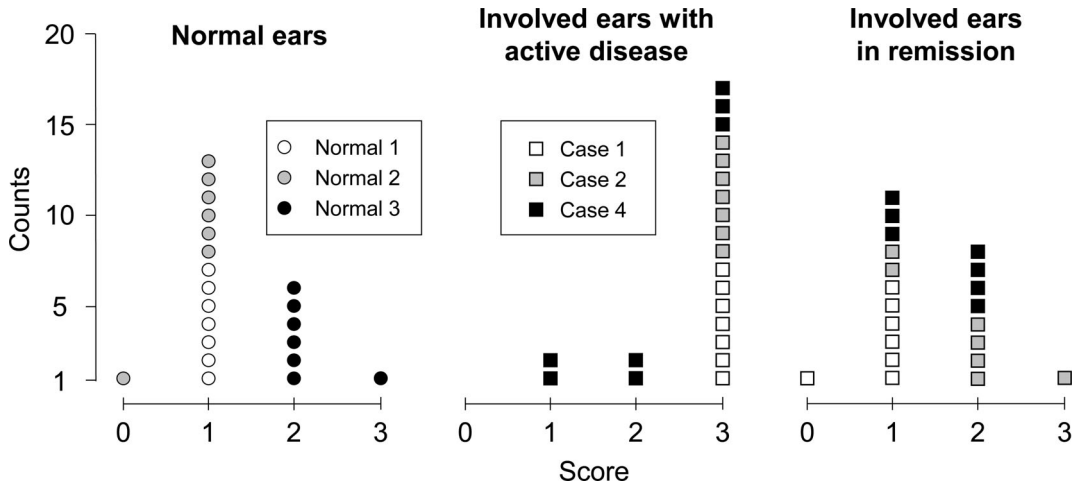

FIGURE 5 Comparison of signal intensity of the mastoid segment of the fallopian canal. At relapse, vasculitis-involved ears show significantly higher signal intensity than normal ears ($P < .001$ by Mann-Whitney U test). The signal intensity decreases significantly at remission ($P < .001$ by Wilcoxon signed-rank test), to a level statistically similar to that of normal ears ($P = .597$ by Mann-Whitney U test). Individuals in each group are indicated by different colors

posterior wall of the external ear canal,[10] which are in line with clinical presentations and MRI findings in this study. We therefore consider that otologic involvement of AAV is at least in part caused by vasculitis of the SMA. Nogaki et al reported a negative pathologic study of vasculitis in the fallopian canal in GPA patients without facial paresis,[19] possibly due to the effect of immunosuppressive treatment or absence of otologic involvement. MIP images also visualized mucous inflammation in the eustachian tube and epitympanic recess. Occlusions of the eustachian tube by granulomatous tissues are usually found in GPA patients, which may cause otitis media with effusion.[4,9] The findings indicate possible involvement of other arteries in vasculitis of the ear, such as the middle meningeal artery and anterior tympanic artery.[10]

CE-3D-GRE is potentially useful in assessing disease activity of AAV because it showed decreased enhancement of the fallopian canal following immunosuppressive treatment. In this study, signal intensity of the mastoid segment appeared weaker while in relapse during the therapy (Figure 1 and Figure 4) compared with that before treatment (Figure 2 and Figure 3). We could not evaluate radiological features of AAV patients in relation to their ANCA status. Further investigations using more patients are necessary for defining the value of this method in the diagnosis and monitoring of patients with AAV. Another issue in this study is the detailed protocol of CE-3D-GRE. Based on our experience, the difference between SPGR and fat-suppressive FFE was not important in evaluation of the facial nerve. Rather, thin slice thickness (less than 1.0 mm) seemed essential for visualizing affected vessels in relationship with the involved organs. Imaging protocol should be optimized for better assessment of vasculitis and disease activity in the temporal bone to allow early diagnosis and therapy optimization for AAV.

5 | CONCLUSION

We evaluated the feasibility of CE-3D-GRE for imaging signs of vasculitis in the temporal bone on four patients with otologic involvement of GPA and EGPA. We found that intense enhancement of the tympanic and mastoid segments of the facial nerve in the affected ears was closely related to disease activity and severity of vasculitis symptoms in the external ear, middle ear, and inner ear. These findings are potentially useful in the diagnosis of otologic involvement in patients with AAV in a less invasive manner.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose related to this article.

MEETING INFORMATION

The content of this article has not been presented in any previous conference.

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BIBLIOGRAPHY

1. Jennette JC, Falk RJ. Pathogenesis of antineutrophil cytoplasmic autoantibody-mediated disease. *Nat Rev Rheumatol*. 2014;10(8):463-473.
2. Rasmussen N. Management of the ear, nose, and throat manifestations of Wegener granulomatosis: an otorhinolaryngologist's perspective. *Curr Opin Rheumatol*. 2001;13(1):3-11.
3. Seccia V, Fortunato S, Cristofani-Mencacci L, et al. Focus on audiologic impairment in eosinophilic granulomatosis with polyangiitis. *Laryngoscope*. 2016;126(12):2792-2797.
4. Takagi D, Nakamaru Y, Maguchi S, Furuta Y, Fukuda S. Otologic manifestations of Wegener's granulomatosis. *Laryngoscope*. 2002;112(9):1684-1690.
5. Harabuchi Y, Kishibe K, Tateyama K, et al. Clinical features and treatment outcomes of otitis media with antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis (OMAAV): a retrospective analysis of 235 patients from a nationwide survey in Japan. *Mod Rheumatol*. 2017;27(1):87-94.
6. Kühn D, Hospowsky C, Both M, Hey M, Laudien M. Manifestation of granulomatosis with polyangiitis in head and neck. *Clin Exp Rheumatol*. 2018;36(suppl 111):78-84.
7. Devaney KO, Travis WD, Hoffman G, Leavitt R, Lebovics R, Fauci AS. Interpretation of head and neck biopsies in Wegener's granulomatosis. A pathologic study of 126 biopsies in 70 patients. *Am J Surg Pathol*. 1990;14(6):555-564.
8. Goldfarb JM, Rabinowitz MR, Basnyat S, Nyquist GG, Rosen MR. Head and neck manifestations of eosinophilic granulomatosis with polyangiitis: a systematic review. *Otolaryngol Head Neck Surg*. 2016;155(5):771-778.
9. Santos F, Salviz M, Domond H, Nadol JB. Otopathology of vasculitis in granulomatosis with polyangitis. *Otol Neurotol*. 2015;36(10):1657-1662.
10. Nager GT, Nager M. The arteries of the human middle ear, with particular regard to the blood supply of the auditory ossicles. *Ann Otol Rhinol Laryngol*. 1953;62(4):923-949.
11. Hong HS, Yi BH, Cha JG, et al. Enhancement pattern of the normal facial nerve at 3.0 T temporal MRI. *Br J Radiol*. 2010;83(986):118-121.
12. Dehkharghani S, Lubarsky M, Aiken AH, Kang J, Hudgins PA, Saindane AM. Redefining normal facial nerve enhancement: healthy subject comparison of typical enhancement patterns—unenhanced and contrast-enhanced spin-echo versus 3D inversion recovery-prepared fast spoiled gradient-echo imaging. *Am J Roentgenol*. 2014;202(5):1108-1113.
13. Watts R, Lane S, Hanslik T, et al. Development and validation of a consensus methodology for the classification of the ANCA-associated vasculitides and polyarteritis nodosa for epidemiological studies. *Ann Rheum Dis*. 2007;66(2):222-227.
14. Masi AT, Hunder GG, Lie JT, et al. The American College of Rheumatology 1990 criteria for the classification of Churg-Strauss syndrome (allergic granulomatosis and angiitis). *Arthrit Rheum*. 1990;33(8):1094-1100.
15. Al-Noury K, Lotfy A. Normal and pathological findings for the facial nerve on magnetic resonance imaging. *Clin Radiol*. 2011;66(8):701-707.

16. Martin-Duverneuil N, Sola-Martinez MT, Miaux Y, et al. Contrast enhancement of the facial nerve on MRI: normal or pathological? *Neuroradiology*. 1997;39(3):207-212.
17. Ogawa A, Sando I. Spatial occupancy of vessels and facial nerve in the facial canal. *Ann Otol Rhinol Laryngol*. 1982;91(1 Pt 1):14-19.
18. Joglekar S, Deroee AF, Morita N, Cureoglu S, Schachern PA, Paparella M. Polyarteritis nodosa: a human temporal bone study. *Am J Otolaryngol*. 2010;31(4):221-225.
19. Nogaki T, Keskin N, Azuma T, Paparella MM, Nadol JB, Cureoglu S. Quantitative assessment of vestibular otopathology in granulomatosis with polyangitis: a temporal bone study. *Laryngosc Investig Otolaryngol*. 2018;3(6):473-477.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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