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3D-constructive interference into steady state (3D-CISS) labyrinth signal alteration in patients with vestibular schwannoma

Franca Wagner [a,*], Evelyn Herrmann [b], Roland Wiest [a], Andreas Raabe [d], Corrado Bernasconi [e], Marco Caversaccio [c], Dominique Vibert [c]

[a] *Department of Diagnostic and Interventional Neuroradiology, Inselspital, University of Bern, Bern, Switzerland*
[b] *Department of Radiation Oncology, Inselspital, University of Bern, Bern, Switzerland*
[c] *Department of Otorhinolaryngology and Head & Neck Surgery, Inselspital, University of Bern, Bern, Switzerland*
[d] *Department of Neurosurgery, Inselspital, University of Bern, Bern, Switzerland*
[e] *Statistics Division, Inselspital, University of Bern, Bern, Switzerland*

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ABSTRACT

Objective: To evaluate signal intensity of the inner ear using 3D-CISS imaging and correlated signal characteristics in patients with vestibular schwannoma to neuro-otological symptoms.
Methods: Sixty patients with unilateral vestibular schwannoma were retrospectively reviewed. All patients had had initial and follow-up magnetic resonance imaging (MRI). Individual treatment strategies consisted of "wait-and-watch", surgical tumour resection, stereotactic radiosurgery or both surgery and stereotactic radiosurgery. For all patients a complete baseline and treatment course neuro-otological examination was re-studied.
Results: On initial MRI, 3D-CISS sequence signal loss of the membranous labyrinth was present in 20 patients (33.3%); signal loss of cochlea in 20 (33.3%) and coincident signal loss of sacculus/utriculus in 17 (85%) of them. Sequential analysis of follow-up MRI series demonstrated slightly increased labyrinthine signal degradation, independently of the chosen therapy. Correlation of initial MRI results with initial neuro-otological symptoms showed significance only for cochlear obstruction versus vertigo ($p = 0.0397$) and sacculus/utriculus obstruction versus vertigo ($p = 0.0336$). No other statistically significant relationships were noted.
Conclusion: 3D-constructive interference into steady state (3D-CISS) is appropriate for observing inner ear signal loss in patients with vestibular schwannoma. However, except for vertigo, no significant correlation was noted between initial neuro-otological symptomatology and signal loss of the inner ear.

Abbreviations: CISS, constructive interference into steady state; CPA, cerebellopontine angle; FLAIR, fluid-attenuated inversion recovery; IAC, internal auditory canal; MPR, multiplanar; ST, slice thickness; VS, vestibular schwannoma.

* Corresponding author at: Institute for Diagnostic and Interventional Neuroradiology, University Hospital Bern and Inselspital, Freiburgstrasse 4, 3010 Bern, Switzerland. Fax: +41 31 632 48 72.
E-mail addresses: franca.wagner@insel.ch (F. Wagner), evelyn.herrmann@insel.ch (E. Herrmann), roland.wiest@insel.ch (R. Wiest), andreas.raabe@insel.ch (A. Raabe), corrado.bernasconi@insel.ch (C. Bernasconi), marco.caversaccio@insel.ch (M. Caversaccio), dominique.vibert@insel.ch (D. Vibert).

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1. Introduction

Vestibular schwannomas (VS) are benign slow-growing tumours that arise from the eighth cranial nerve. They are the most common neoplasm of the cerebellopontine angle (CPA) and internal auditory canal (IAC) [1] with an annual incidence of 2/100,000 cases [2]. In rare instances, VS may arise primarily from the inner ear itself [3]. Clinical symptoms typically manifest as hearing complaints such as tinnitus or unilateral sensorineural hearing loss. If there is no subsequent treatment, neurological signs – e.g., ataxia, gait disturbance, facial dysfunction or hydrocephalus – may appear. Theoretically, the hearing loss, the hallmark of VS, may result from cochlear nerve dysfunction (retrocochlear mechanism), cochlea dysfunction (cochlear mechanism), or both [4].

Neuro-otologists commonly attribute VS-associated hearing loss to dysfunction of the cochlear nerve. Supporting evidence is based on retrocochlear abnormalities recorded with brainstem evoked auditory responses [5,6] and histopathological findings demonstrating atrophy of the cochlear nerve [7,8].

Less attention has been focused on the cochlea as the origin of the hearing loss, although clinical observations suggest that cochlear dysfunction might be frequent in patients who suffer from VS-induced hearing loss. Studies have shown that more than 80% of cochlear neurons must be lost before there is a significant shift in pure tone thresholds and that such severe neuronal losses are associated with a considerable drop in speech discrimination ability [9,10]. Clinical data have demonstrated mild-to-moderate degrees of threshold shift with relatively preserved speech discrimination in patients with VS [11–15]. Clinical studies also found no correlation between the size of VS and the degree of hearing loss [16,17], indicating that compression of the cochlear nerve within the IAC may not be the sole cause of hearing loss. In other words, the commonly observed hearing loss for pure tones (in the absence of poor speech discrimination scores) in patients with VS may be due to cochlear mechanisms rather than retrocochlear dysfunction. Indeed, previous studies showed that protein levels in the endolymph were 5–15 times greater in VS patients than in normal controls [18,19]. However, the mechanism generating this increased protein concentration is unknown.

The appropriate magnetic resonance imaging (MRI) approach for detecting haemorrhage or high protein concentration within the body fluids is the sequence FLAIR (fluid-attenuated inversion recovery). Based on the hypothesis that increased protein content may be detectable in cochlear endolymph of patients with VS, Bhadelia et al. [20] investigated cochlear signal intensity changes in patients with unilateral VS using a two-dimensional (2D) FLAIR sequence. The authors reported that cochlear signal intensity was significantly higher on the affected than the unaffected side. However, 2D-FLAIR images employing the thick section slices used by Bhadelia et al. [20] provide suboptimal images of the inner ear [21–23]. Furthermore, Bhadelia et al. [20] did not evaluate the correlation between hearing levels, clinical status, and signal intensity of the labyrinth. Assessment of this relationship might be helpful to understand whether signal intensity changes in the labyrinth accurately reflect the hearing prognosis of patients with VS: e.g., signal intensity changes versus the initial level of hearing loss, or initial normal hearing versus verified signal changes of the labyrinth in MRI.

Accordingly, the purpose of this study was to evaluate the signal intensity of the cochlear endolymph and sacculus/utriculus in VS patients using 3D-constructive interference into steady state (3D-CISS) sequence in initial and follow-up MRI. According to our literature search, and to our knowledge, no such study has yet been published. The baseline MRI was performed before VS treatment, which consisted of either a "wait-and-watch"-strategy, surgical tumour resection, stereotactic radiosurgery or both surgery and stereotactic radiosurgery. The baseline MRI characteristics enabled us to correlate the signal characteristics with the initial neuro-otological symptomatology, the tumour volume, and tumour infiltration of the cochlear aperture. The follow-up MRI was used to assess signal changes of 3D-CISS and MRI findings and determine their correlation with the neuro-otological follow-up results.

2. Material and methods

This retrospective study was approved by the Ethics Committee of the Canton of Bern, and was performed in accordance with the Declaration of Helsinki (2015) [24]. The requirement for informed consent was waived due to the retrospective nature of the study.

2.1. Patient data

Sixty patients with a unilateral VS and a complete dataset of initial MRI with 3D-CISS, follow-up MRI with 3D-CISS, and complete baseline and follow-up neuro-otological examination were retrospectively reviewed. For each patient the individual treatment regime consisting of the "wait-and-watch"-strategy, surgical tumour resection, stereotactic radiosurgery, or both surgery and stereotactic radiosurgery, was intensively discussed during regular sessions of our Interdisciplinary Schwannoma Board. Patients with neurofibromatosis type 2 and patients with a purely intralabyrinthine schwannoma were a priori excluded.

2.2. Imaging analysis

Evaluation of MRI scans was performed by one neuroradiologist (FW; a neuroradiologist with extensive experience in head and neck imaging). The tumour size was graded according the Koos classification [25]: Koos stage 1; tumour confined to the IAC; stage 2, tumour <2 cm in diameter; stage 3, tumour >2 cm, no compression to brain stem; stage 4a, tumour compressing the brain stem without midline shift and stage 4b, tumour compressing the brain stem with midline shift.

To analyse the signal intensity and signal loss of the cochlear endolymph and sacculus/utriculus in VS patients we used the 3D-CISS sequence in initial and follow-up MRI. For the qualitative visual analysis of the membranous labyrinth in initial and follow-up MRI we compared the signal intensity of the inner ear (cochlear endolymph and sacculus/utriculus) to the signal intensity of the liquor cerebrospinalis, which should normally be of equal signal intensity in healthy patients. We

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