Leptomeningeal carcinomatosis and sensorineural hearing loss: Correlation of labyrinthine enhancement patterns with symptoms

Carcinomatose leptoméningée et perte d’audition : corrélation du rehaussement labyrinthique avec la symptomatologie clinique


a Department of Radiology, hôpital de Hautepierre, 1, avenue Molière, 67098 Strasbourg, France
b Department of Surgery, hôpital de Hautepierre, Strasbourg, cedex, France

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Summary

Objectives. — The purpose of this study was to investigate the correlation between hearing loss and inner ear enhancement in patients suffering from leptomeningeal carcinomatosis (LC) involving the internal acoustic canal (IAC). Previous studies have only reported an association between IAC enhancement and sensorineural deafness.

Material and methods. — In a prospective study conducted from 2005 to 2007, 14 patients with LC involving the IAC underwent high-resolution MRI and otolaryngology examination. MRI images were analyzed by two experienced radiologists who were blinded to audiologic investigation results.

Results. — Three (21%) patients had IAC and inner ear enhancement on gadolinium-weighted MRI. All three had a sensorineural hearing loss. Eleven (79%) patients had IAC enhancement without inner ear enhancement. Nine of these 11 patients were free of sensorineural hearing loss. Only two of them had sensorineural deafness.

Conclusion. — These findings are suggestive of a relation between hearing loss and inner ear enhancement in leptomeningeal carcinomatosis, as previously reported for bacterial meningitis. However, further investigations, including radiopathological correlation and a larger number of patients, are warranted to confirm these preliminary results.

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∗ Corresponding author.
E-mail address: gilles.goyault@chru-strasbourg.fr (G. Goyault).

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Introduction

Leptomeningeal carcinomatosis (LC) is a rare complication of diffuse metastatic neoplasia (approximately 5% of patients with cancer). Neoplastic cells disseminate to the central neurological system through the cerebrospinal fluid from a hematogenous spread or a direct contamination from a preexisting lesion. LC can involve any part of the brain, spinal cord or cranial nerves, such as cochleovestibular nerves [1]. Previous reports showed that acoustic nerves or internal acoustic canal (IAC) enhancement on contrast-weighted MRI are associated with hearing loss [2—5]. Several pathophysiological mechanisms have been discussed such as direct nerve infiltration, nerve ischemia induced by IAC vessel compression and labyrinth infiltration [6]. This last hypothesis has also been proposed to explain cochlear dysfunction in bacterial meningitis, with Dichgans et al. demonstrating a correlation between inner ear abnormalities on MRI and the extent of cochlear dysfunction [7]. However, the analysis of inner ear abnormalities requires high-resolution MRI with thin contrast-enhanced T1-weighted sequences associated with heavily T2-weighted sequences focused on the IAC [7].

Moreover, acoustic nerves or ICA enhancement are often detected on contrast-weighted MRI, without cochlear dysfunction.

These mismatched results led us to hypothesize that the hearing loss observed in LC is related to inner ear abnormalities rather than to IAC contrast enhancement.

The purpose of our study was to investigate inner ear contrast enhancement in patients suffering from LC involving the IAC and to assess the correlation between sensorineural deafness and labyrinthine enhancement.

Material and methods

Patients

In a prospective conducted from early 2005 to mid-2007, 14 patients with LC involving the IAC (seven females, seven males; age range, 20—84 years; mean age, 60 years) underwent high-resolution MRI and otolaryngology investigation. Inclusion criteria were leptomeningeal and IAC enhancement on high-resolution MRI of inner ear or gadolinium-enhanced brain MRI, without ongoing infection. Patients underwent MRI exams in the follow-up of a pre-existing cancer or in case of headache, deafness, vertigo or alteration of general condition, with or without a history of cancer. All subjects gave informed consent (Table 1).

Otologic examinations

All patients underwent an audiologic examination and a pure-tone audiometry (PTA) was performed on seven of the 14 patients. The other seven patients did not undergo a PTA because the clinical examination was negative (no clinical hearing loss) or the performance status was poor (the
Audiometric tests were performed in the department of Otolaryngology using a Midimate 622 audiometer (Madsen Electronics, Copenhagen, Denmark).

The audiologic/audiometric findings were assessed semi-quantitatively to be either positive (sensorineural deafness) or negative (no sensorineural deafness), with no history of chronic deafness. Presbycusis was not considered as a pathologic finding when correlated to typical clinical (bilateral, progressive and symmetrical hearing loss) or audiometric (bilateral and symmetrical decrease of high-frequency hearing thresholds) abnormalities.

**Acquisition protocol**

All patients underwent a high-resolution MRI of the inner ear with gadolinium injection. Exams were performed on a 1.5T Vision and a 1.5T Avanto (Siemens, Erlangen, Germany) using a carotid surface coil positioned on the outer ear canal associated with a 12-channel head coil. Three series were acquired:

- transverse T1 SE Fat Sat (TR/TE 719/13 ms; NEX, 2; 288 × 384 matrix; field of view, 220 mm; one 17-slice axial slab; slice thickness, 1.5 mm; acquisition time, 7 min);
- transverse T1 SE Fat Sat with gadolinium enhancement (same parameters, manual intravenous gadolinium-based contrast media administration, 0.2 mL/kg of gadodiamide [Omniscan, GE Healthcare, Oslo, Norwa]);
- 3D-CISS (TR/TE 12/6 ms; NEX, 1; 320 × 384 matrix; field of view, 200 mm; one 64-slice axial slab; slice thickness, 0.4 mm; acquisition time, 6–7 min).

**Image analysis**

Two experienced radiologists (F.V. over 25 years experience and S.K. over five years experience) reviewed all MRI studies with consensus determination, blinded to audiologic investigation results. The findings were assessed qualitatively as positive (inner ear enhancement on gadolinium-weighted images) or negative (no inner ear enhancement on gadolinium-weighted images) and only concerned labyrinthine modifications, given that every patient had a known LC involving the IAC.

To exclude solid lesions of the inner ear, 3D-CISS sequences were acquired and perilymphatic fluid signal was assessed qualitatively as normal (hyperintense) or abnormal (iso- or hypointense).

**Results**

The results are summarized in Table 1 and Fig. 1.

All 14 (100%) patients had a uni- or bilateral (three uni-lateral and 11 bilateral) IAC uptake on contrast-weighted images. Three of the 14 (21%) patients had inner ear enhancement and the other 11 (79%) had no inner ear enhancement.

The three (21%) patients with inner ear enhancement had sensorineural deafness (uni- or bilateral, according to MRI findings), confirmed by PTA in two cases. The third patient only had an audiologic examination.

Nine of the 11 patients without inner ear enhancement were free of sensorineural hearing loss; four were confirmed by PTA and five by audiologic examination. Two patients without inner ear enhancement had sensorineural deafness (one confirmed by PTA and the other by audiologic examination).

All patients with inner ear enhancement had a corresponding hearing loss (two bilateral cases and one unilateral case).

The signal of perilymphatic fluid was always normal (hyperintense) on CISS-weighted images.

**Discussion**

LC is a rare and very ominous complication of metastatic cancers [8]. Sensorineural hearing loss is an unusual presentation of LC, reported in only a few cases in the literature, and is usually correlated to IAC enhancement [3,4,9–12]. However, none of these studies reported the possible relation between hearing loss and inner ear enhancement on gadolinium-weighted sequences. This could perhaps be explained by the need for high-resolution MRI (thin contrast-enhanced T1 SE sequences and heavily weighted T2 sequences such as CISS-weighted sequences) focused on the IAC as shown by Dichgans et al. [7].
In addition, Dichgans et al. already showed a significant correlation between the presence of cochlear enhancement and the extent of hearing loss in seven adults with bacterial meningitis [7]. Their MRI findings argue for a cochlear origin of meningitis-associated hearing loss and the pathological findings suggest the direct invasion of bacteria from the meninges into the inner ear [13]. Given the very good agreement between these findings and ours, a similar, or very close, route of infection and leptomeningeal mets can be hypothesized: inner ear enhancement may correspond to direct invasion of neoplastic cells from the meninges of the IAC into the inner ear, as already showed with bacteria. The correspondence between the sides of inner ear enhancement and hearing loss, particularly in patient 8, is another argument for this explanation.

Our study shows a very good correlation between inner ear enhancement on MRI and sensorineural deafness. It shows a strong correlation between absence of deafness and absence of inner ear uptake, as there were no normal audiologic examinations in the group of patients with labyrinthine enhancement. However, one weakness of this study concerns the patients with sensorineural hearing loss who did not show inner ear enhancement. This suggests that in cases of deafness, inner ear involvement cannot always be confirmed at present. Indeed, we may have reached the limits of our current MRI device’s resolution. Further progress may come with 3T MRI and new sequences.

Conclusion

MRI can visualize the involvement of inner ear structures in bacterial meningitis. Our study shows that MRI can also visualize involvement of cochleovestibular structures in LC and highlights a probable relation between hearing loss and inner ear enhancement in patients with LC. Further investigations that include a larger number of patients and pathological correlation are warranted to confirm these findings.

However, a patient presenting with a history of cancer and sudden hearing loss is suggestive of LC involving the inner ear and a high-resolution MRI exploration should be carried out.

Conflicts of interest

None.

References