

Contrast-Enhanced Magnetic Resonance Imaging of the Internal Auditory Canals and Posterior Fossa

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Abstract

A retrospective review of 314 cases of contrast-enhanced magnetic resonance imaging (MRI) of the posterior fossa and internal auditory canals was carried out correlating the presenting symptoms with the scan findings. 7.2% of the cases showed findings on the MRI which could account for the patients' symptoms. Patients with sensorineural hearing loss were more likely to have a positive scan than those presenting with vertigo and/or tinnitus without hearing loss. Acoustic schwannoma was the most common lesion detected. Labyrinthine lesions e.g. cochlear schwannoma, labyrinthitis, congenital labyrinthine abnormality, and central lesions e.g. multiple sclerosis, brainstem glioma were some of the other lesions detected.

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Key words: Acoustic schwannoma, Endolymphatic sac, Labyrinthitis, Tinnitus, Vertigo

Introduction

Contrast-enhanced magnetic resonance imaging (MRI) is now the gold-standard examination for the exclusion of acoustic schwannomas or other posterior fossa tumours.¹ We retrospectively reviewed 314 cases done over a period of 2 years and 9 months, correlating presenting symptoms with scan findings. Patients with sensorineural hearing loss (SNHL) were found to have higher positive findings compared to those without. There was also a need to scrutinise the entire acoustic pathway so that lesions outside the posterior fossa and internal auditory canal (IAC) e.g. labyrinthine and brainstem lesions responsible for hearing loss or other symptoms would not be missed.

Materials and Methods

Three hundred and fourteen cases of contrast-enhanced MRI of the internal auditory canals and posterior fossa done between November 1993 and July 1996 were retrospectively reviewed. Presenting symptoms were correlated with scan findings. Patients were grouped into those presenting with SNHL and those without SNHL.

The entire acoustic pathway comprising the cochlea, IAC, cerebellopontine angle, cochlear nuclei, trapezoid body, lateral lemniscus, inferior colliculus, medial geniculate

body and the auditory cortex in the superior temporal gyrus,² were scrutinised to detect any lesions responsible for SNHL and/or tinnitus. Special attention was also paid to the vestibule, semicircular canals, vestibular nuclei (which are located medial to the cochlear nuclei at the level of the inferior cerebellar peduncle), and the cerebellum to detect for any lesion responsible for vertigo.

All the cases were scanned with a 1.5T GE Signa MR machine. An axial T2 weighted image of the entire brain was carried out for every case followed by pre and post-enhancement T1 weighted images of the posterior fossa and IAC in the axial and coronal planes. All adult patients were given 10 ml and children 0.2 mmol/kg of MRI contrast agent, Magnevist. Imaging parameters for T2 weighted images were: TR of 3200, TE of 85, thickness of 5 mm skip 2 mm, matrix size of 256 x 256, 22 cm field of view and 1 excitation. Imaging parameters for T1 weighted images were: TR of 500, TE of 11, thickness of 3 mm skip 1 mm, matrix size of 256 x 256, 22 cm field of view and 2 excitations. The post-enhancement scans were done with fat saturations.

None of the 314 patients had undergone computed tomography (CT) scan before the MRI examination. One patient had CT scan done after the MRI to assess for the effect of the middle ear mass lesion on the ossicles.

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Results

Of the 314 patients, 264 presented with SNHL (Table I). Some of these patients also had tinnitus and/or vertigo. Four of the 264 patients also had more complex symptoms e.g. ipsilateral lower motor neurone facial nerve palsy, ataxia or diplopia due to 6th nerve palsy. Fifty out of the 314 patients presented with tinnitus and/or vertigo without SNHL. All the MRI requests were for exclusion of acoustic schwannoma or other cerebellopontine tumours.

We defined a positive scan as the detection of a lesion which could explain for the patient's symptoms. A total of 26 (7.2%) positive scans were obtained. The breakdown of the positive scans is shown in Table II. All except one of the patients in whom a positive scan was detected had SNHL. Nine of the above patients also had tinnitus and/or vertigo. The patient with multiple sclerosis (Fig. 1) had ataxia and other cerebellar signs on top of the SNHL. The patient with brainstem glioma (Fig. 2) had left 6th and bilateral lower motor neurone 7th nerve palsy as well. Two patients with brainstem infarcts had associated lower motor neurone 7th nerve palsy on the same side. The only patient with a positive scan that did not have SNHL presented with tinnitus and was found to have facial nerve schwannoma (Fig. 3) involving the tympanic portion of the facial nerve. The impingement of the schwannoma onto the stapes was believed to be the cause of the tinnitus.

TABLE I: DISTRIBUTION OF THE STUDY POPULATION BY PRESENTING SYMPTOMS

Presenting symptoms	Number of cases
SNHL with/without tinnitus and/or vertigo	264
Tinnitus and/or vertigo without SNHL	50
Total	314

SNHL: sensorineural hearing loss

TABLE II: BREAKDOWN OF THE LESIONS IN THE POSITIVE SCANS

Diagnosis	Number of cases
Acoustic schwannoma	13
Meningioma	1
Labyrinthine schwannoma	1
Epidermoid tumour	1
Brainstem glioma	1
Facial nerve schwannoma	1
Brainstem infarct	2
Multiple sclerosis	1
Large endolymphatic duct and sac syndrome	1
Congenital abnormality of the labyrinth	1
Cochlear otosclerosis	1
Labyrinthitis	2
Total	26

Of all the patients who presented with SNHL, 9.2% had a positive scan whereas only 2% of those without SNHL had a positive scan. This suggested that those who had SNHL were more likely to have an underlying MRI demonstrable cause rather than those without SNHL.

The age of the study population ranged from 9 to 84 years with a mean of 43.37 years. The mean age of the positive scan patients was 45.96 years. The patient with congenital labyrinthine abnormality (Fig. 4) and that of the large endolymphatic duct and sac syndrome (Fig. 5) were 32 and 17 years of age at presentation respectively. Both also had long-standing history of SNHL.

There were 180 males and 134 females in the study population. It was interesting to note that of the 13 cases of acoustic schwannomas, 10 were males and only 3 were females.

Discussion

One main concern of clinicians for patients presenting with SNHL, tinnitus or vertigo is the exclusion of acous-

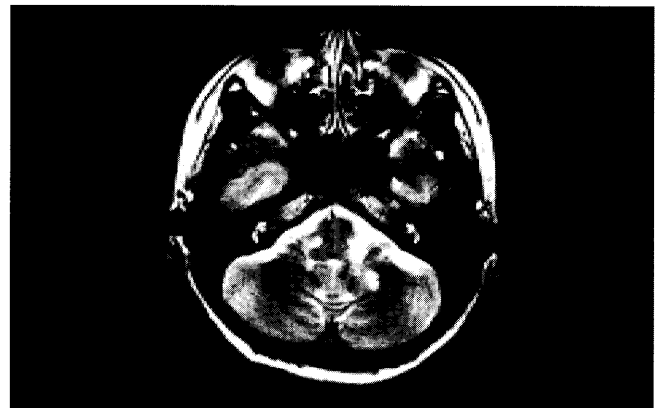


Fig. 1. T2 weighted image of a young patient with multiple sclerosis. Arrowhead points to the plaque in the left inferior cerebellar peduncle where the dorsal and ventral cochlear nuclei are located. The arrow points to another plaque in the left cerebellar hemisphere.

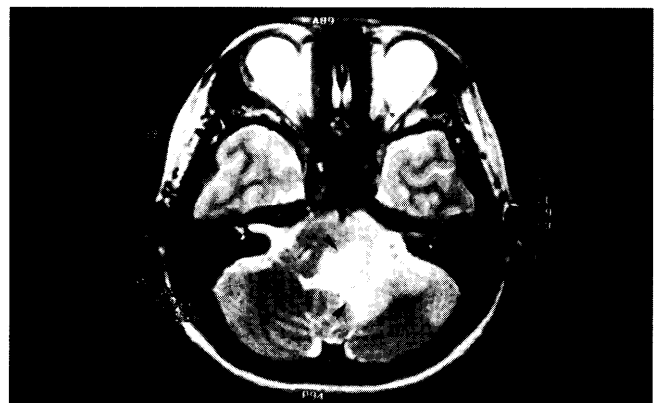


Fig. 2. Brainstem glioma. The arrowheads point to an area with high signal changes involving the pons on the left side, the left inferior cerebellar peduncle and the adjacent left cerebellar hemisphere in this T2 weighted axial image. This area of high signal changes comprises the glioma with surrounding oedema.

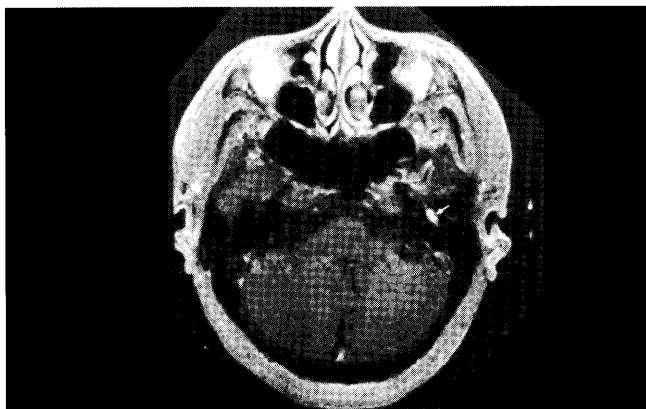


Fig. 3. Post-enhancement T1 weighted image of the patient with facial nerve schwannoma. The small white arrow points to the enhancing lobulated schwannoma involving the tympanic portion of the left facial nerve.

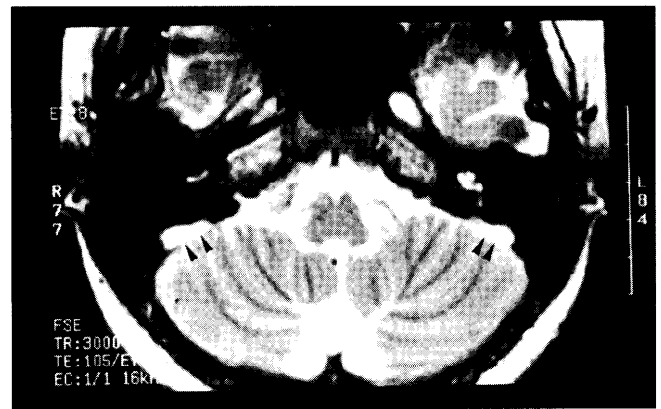


Fig. 5. T2 weighted image of the patient with large endolymphatic duct and sac syndrome. The arrowheads point to the dilated endolymphatic sacs on both sides.



Fig. 4. Axial T2 weighted image of the patient with congenital abnormality of the labyrinth on the right side. The basal turn of the cochlea is dilated (*), and the lateral semicircular canal and the vestibule forms a common cavity (small white arrow).



Fig. 6. Acoustic schwannoma. Post-enhancement T1 weighted image shows the characteristic enhancing ice-cream cone lesion in the left cerebellopontine angle and internal auditory canal (arrow).

tic schwannoma or other cerebellopontine angle tumour. This is particularly so if the SNHL is unilateral and progressive in nature. The current gold standard test for this purpose is contrast-enhanced MRI.¹ Cheaper tests like the auditory brainstem response is at best only 90% sensitive for the detection of acoustic schwannoma and the sensitivity drops to about 80% for tumour 1 cm or smaller in size.^{3,4} Contrast-enhanced CT scan is another less costly examination but it is known to miss small intra-canalicular tumours.⁵ Combination with air cisternography increases its detection rate but this technique has its fair share of complications and interpretive errors.⁶ For these reasons, clinicians generally omit the less sensitive tests and request for contrast-enhanced MRI instead.

In this study, it was observed that SNHL as a symptom had a higher number of positive scans than those presented with just vertigo and /or tinnitus without hearing loss. Several reasons could account for this. Firstly, since acoustic schwannomas (Fig. 6) formed half of all the positive cases, the dominant presenting symptom for this condition would understandably affect our results.

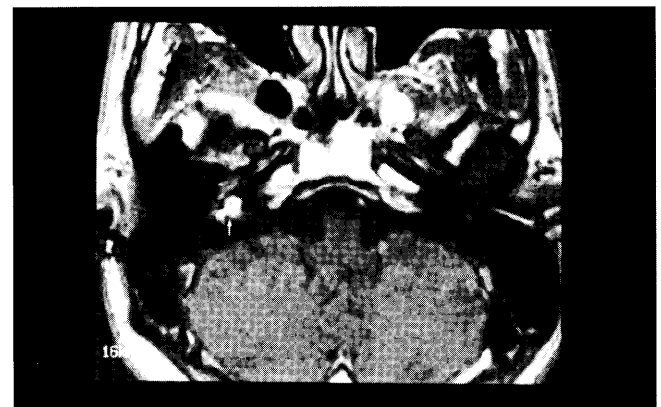


Fig. 7. Labyrinthine schwannoma. Post-enhancement T1 weighted image showing an intensely enhancing mass lesion in the cochlea and vestibule (small white arrow).

It was reported that 95% of patients with acoustic schwannoma had various degree of SNHL.⁷ Secondly, if a lesion affecting the vestibular nerve or labyrinth developed slowly, the brain would have ample time to compensate for loss of vestibular function and that would explained why patients with acoustic schwannoma or cerebellopontine angle meningioma seldom

complained of vertigo.⁸ On the other hand, the brain had no ability to compensate for loss of hearing. Thirdly, Casselman et al⁹ reported that special MRI sequences were required to detect subtle lesions within the labyrinth that were responsible for vertigo. These sequences were not part of our protocol and therefore we could be missing some lesions within the labyrinth responsible for vertigo. Fourthly, the difference in the sample size between the group with SNHL and the group without SNHL may have affected the results.

Therefore, in the group of patients without hearing loss, it may not be cost-effective to use contrast-enhanced MRI as a screening test for acoustic schwannoma or other cerebellopontine angle tumour. Low cost high-resolution Fast Spin Echo (FSE) MRI of the IAC could be a good alternative.¹⁰ This technique produces T2 weighted images of the IAC using FSE sequence. Using 3 mm thickness, a TR of 5000 and TE of 102, acoustic schwannoma as small as 1 x 2 x 2 mm in dimension had been detected.¹⁰ As intravenous contrast is not used, the cost of the examination is lower than that of contrast-enhanced MRI. However, this technique is not as sensitive as contrast-enhanced MRI and early acoustic schwannomas could still be missed. Nevertheless, for those patients without SNHL where the yield of contrast-enhanced MRI is expected to be low, this could turn out to be a more cost-effective examination.

The nuclei of the 6th, 7th and 8th cranial nerves are concentrated within the brainstem. This was the reason why those patients with brainstem lesions had more than one cranial nerve involvement. Some of the lesions, e.g. brainstem glioma, also involved the neural fibres connecting the cerebellum and the brainstem; thereby giving rise to ataxia.

Knowledge of the entire acoustic pathway is essential for any radiologist reading a scan of a patient with SNHL. This was clearly shown by Armington² who demonstrated that besides acoustic schwannomas, other cerebellopontine angle tumours e.g. meningioma, epidermoid, and lesions in the labyrinth and brainstem were detected in patients undergoing contrast-enhanced MRI for unilateral or asymmetrical SNHL. Mark and colleagues³ also described 51 non-acoustic schwannoma lesions along the acoustic pathway that were responsible for SNHL in their paper. With these in mind, we examined the entire acoustic pathway in all our patients with SNHL and managed to detect lesions that were responsible for the patients' symptoms. The breakdown of the lesions is shown in Table II.

Causes of SNHL could be divided into tumours, congenital lesions, ischaemia, inflammation and others. Acoustic schwannoma was the most common lesion in our series, similar to the findings in Armington's series.² This was expected as it had been reported that acoustic

schwannoma was the most common radiologically demonstrated lesion on contrast-enhanced MRI responsible for SNHL.¹¹ This tumour usually arises from schwann cell sheath that surrounds the peripheral portion of the 8th cranial nerve; most commonly from the superior vestibular division. They formed 8% to 10% of all intracranial neoplasms and 75% to 80% of cerebellopontine angle tumours. They had been reported to be slightly more common in females than in males.¹² In our series however, we noted a male predominance among all the cases of acoustic schwannomas. It could be that this tumour behaved slightly differently in the local population but due to the small number of cases, a definite conclusion cannot be drawn at present.

Labyrinthine schwannoma (Fig. 7) is like an acoustic schwannoma except that instead of arising from the schwann cells of the central fibre of the 8th nerve in the IAC, it arises from the dendrites or peripheral branches within the labyrinth.¹³ These are rare tumours and before the era of MRI, they were usually diagnosed during surgery or incidentally in post-mortems. On MRI, it is seen as a focal mass within the cochlea or vestibule which enhances with contrast.

Facial nerve schwannoma arises from the outer layer of the nerve sheath of the facial nerve and can occur along any segment of the nerve. It has, however, a predilection for the geniculate ganglion.¹⁴ Although facial nerve palsy is generally regarded as the most common mode of presentation, the actual symptom that the patient presents is largely dependent on which segment of the nerve is involved. A lesion in the cerebellopontine angle and/or IAC usually produces SNHL due to compression of the acoustic nerve. A lesion in the mastoid portion of the facial nerve usually presents with facial nerve palsy because the canal here is small relative to the size of the nerve and is therefore more susceptible to compression. A lesion involving the tympanic portion usually presents with conductive hearing loss due to encasement or erosion of the ossicles. In our case, tinnitus was the presenting complaint. A CT scan performed on the patient after the MRI showed that the schwannoma had impinged onto the stapes and deviating it inferiorly. This was believed to be the cause of the tinnitus.

It is also worthwhile to note that younger patients with long-standing history of SNHL could be having congenital lesions within the labyrinth, as is the case of congenital abnormality of the cochlea and semicircular canal as well as in the patient with large endolymphatic duct and sac syndrome. It is important to note the latter diagnosis as hearing loss can deteriorate further as a result of minor head trauma¹⁵ and the patient should be advised against taking up contact sport like soccer.

Infarct or haemorrhage involving the cochlear nuclei

should be considered in older patients presenting with SNHL especially if the hearing loss is of sudden onset. These patients often have predisposing factors like diabetes mellitus, hypertension and history of smoking as well.

Labyrinthitis may be described as toxic (ototoxic drugs), viral, serous or suppurative. A diagnosis of labyrinthitis is made when there is enhancement of the membranous labyrinth and/or the 8th cranial nerve in the absence of a mass lesion. Although viral infection is probably the most common cause, the two patients with labyrinthitis in our series had bacterial infection as both of them had middle ear infection associated with labyrinthine enhancement seen on MRI.

A patient with multiple sclerosis could present with SNHL if the disease involve part of the central acoustic pathway (Fig. 1). There are usually multiple plaques present at the same time and the patient would most likely have other central nervous system symptoms and signs besides SNHL. This is considered as an uncommon cause of SNHL in the local context as the disease is much less common than in temperate countries. Other uncommon causes of SNHL includes cochlear otosclerosis, arterio-venous malformation, sarcoid, metastasis and so on.

Conclusion

The following observations were made from the above study:

1. Patients presenting with tinnitus and/or vertigo without SNHL had a low likelihood of finding a MR abnormality in the posterior fossa and IAC responsible for the symptoms. A feasibility study to look into the usage of low cost high-resolution FSE MR as a screening test for this group of patients should be carried out.
2. For patients with unilateral or asymmetrical SNHL, the entire acoustic pathway in contrast-enhanced MR should be scrutinised in order not to overlook the less common lesions in the labyrinth or brainstem that may be responsible for hearing loss.
3. Congenital lesions of the labyrinth should be considered in a young patient with a long-standing history of SNHL.

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