

Ramsay Hunt syndrome mimicking acoustic neuroma on MRI

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Abstract

The authors present a case of Ramsay Hunt syndrome in which the MRI appearance mimicked that of an intracanalicular acoustic neuroma.

Key words: Herpes zoster oticus; Vestibular schwannoma; Magnetic Resonance Imaging

Case history

A 69-year-old retired boiler maker presented with a four-day history of vertigo, a three-day history of clear fluid discharging from his right ear with associated tinnitus and deafness, and a two-day history of progressive right facial weakness. On admission a discharging ear canal, ulcerated posteriorly and a complete VIIth nerve palsy was noted. A sensorineural hearing loss of 40–80 dB was present in both ears, although the right ear was 10–20 dB worse. A diagnosis of Ramsay Hunt syndrome was made clinically. He was treated with intravenous acyclovir, together with ciprofloxacin for a superadded bacterial infection as *Staphylococcus aureus*, *Bacteroides spp.* and anaerobic *Streptococcus sp.* had been isolated from an aural swab. His eye was patched. On review one week later his symptoms had not improved and in view of his asymmetric hearing deficit an outpatient MRI was requested. Two weeks later, however, he was readmitted with nausea, vomiting and vertigo. His facial palsy was still complete but he had now developed third degree nystagmus. A right lateral tarsorrhaphy was undertaken and a CT scan of the petrous temporal bone was obtained. The CT was normal.

His outpatient MRI, eight weeks after presentation, however, showed a small 5 mm spherical enhancing intracanalicular mass around the right facial and vestibulocochlear nerves but without any labyrinthine enhancement (Figure 1). As his clinical picture did not fit in with an acoustic neuroma he was simply followed-up and over the next year a gradual recovery of motor power and improvement in his vestibular symptoms was seen. Repeat MRI one year later showed some residual enhancement around the VIIth and VIIIth cranial nerves on the right, compatible with a resolving perineuritis secondary to herpes zoster (Figures 2a and b).

Discussion

Most acoustic neuromas originate from the VIIIth nerve in the lateral third of the internal auditory canal, where they grow medially along the line of least resistance, remodelling and expanding the canal and extending

through into the cerebellopontine angle. In contrast, VIIth nerve neuromas are rare and may occur on any portion of the nerve. When in the internal acoustic meatus (IAM) they too cause localized erosion and expansion of the canal (Phelps and Stansbie, 1993). They most commonly present with tinnitus and a progressive asymmetrical sensorineural hearing loss, although unusually they may present with sudden hearing loss, vestibular symptoms or from the effects of pressure on the Vth or VIIIth cranial nerves.

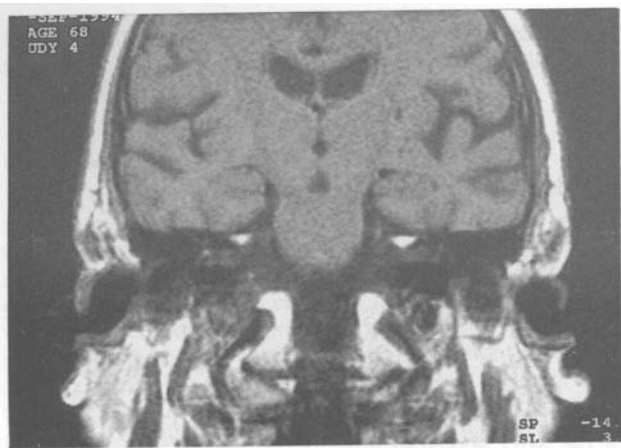
Historically periorbital plain films of the IAM, and more recently CT, have been used to detect acoustic neuromas. While CT may detect expansion of the IAM it misses the



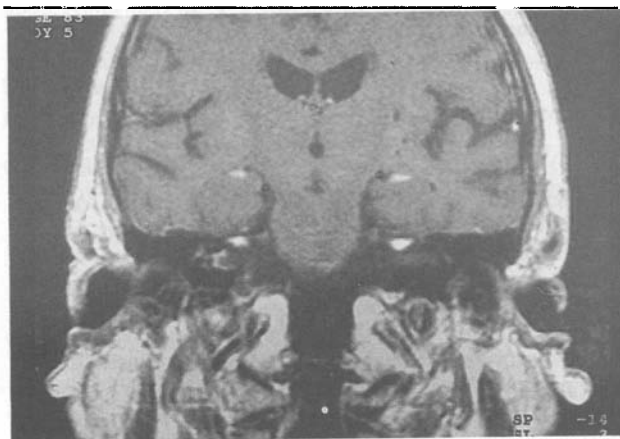
FIG. 1

Gadolinium-enhanced T₁-weighted coronal MRI of the internal auditory meati at presentation, showing intense right intracanalicular enhancement. (Published with permission of the *British Journal of Radiology*).

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(a)



(b)

Fig. 2

(a) Pre- and (b) post-contrast T₁-weighted coronal MRI showing residual perineural enhancement. (Published with permission of the *British Journal of Radiology*).

smaller intracanalicular lesions for which gadolinium-DTPA-enhanced MRI has now become the investigation of choice. Neuromas are typically identified as areas of focal enhancement around the nerves. However enhancement has also been noted in non-neoplastic disease such as Bell's palsy (Daniels *et al.*, 1989; Tien *et al.*, 1990), post-operative states (Daniels *et al.*, 1989), Ramsay Hunt syndrome (Anderson and Laskoff, 1990) and post-traumatic facial paralysis. In contrast to that seen in the neuromas, the inflammatory enhancement has in most cases been reported as diffuse without any mass effect and may be due to surrounding hypervascularity and perineural inflammation or disruption of the blood nerve barrier (Daniels *et al.*, 1989; Tien and Dillon, 1990).

Ramsay Hunt syndrome is caused by reactivation of the herpes zoster virus and presents with facial paralysis, or rarely an VIIIth nerve syndrome, the diagnosis being made clinically by the cutaneous manifestations of vesicles and ulceration. The prognosis is favourable, with recovery of facial motor function normally being seen in over 85 per cent of patients. Pain and hearing loss also recover, although some persistent deficit may remain (Chadwick, 1993). Those cases imaged by MRI report a diffuse enhancement of the facial nerve and labyrinthine enhancement (Osumi and Tien, 1990; Han *et al.*, 1991; Canellas *et al.*, 1993; Tada *et al.*, 1994). Enhancement of the VIIIth nerve in those patients with VIIIth nerve symptoms has less consistently been reported: Tada *et al.* (1994) detected changes in only one out of seven patients with VIIIth nerve

symptoms, in contrast to Yanagida (1993) who found a corresponding inflammation in most cases with VIIIth nerve symptoms in his large survey.

In contrast to the diffuse enhancement usually seen, isolated intracanalicular enhancement has been reported in one patient with Ramsay Hunt syndrome with VIIth and VIIIth nerve symptoms (Anderson and Laskoff, 1990). The MRI was performed acutely on presentation, the changes having nearly completely resolved at follow-up scan four months later. This presumed resolution of neuritic changes as seen on MRI has been suggested as a means of distinguishing inflammatory lesions from tumour by repeat scanning (Mark *et al.*, 1992). Downie *et al.* (1994) are the only group to report changes six months after the onset of symptoms. All other reports with scans performed during the acute phase of the illness reported no significant enhancement seen beyond six weeks. Their case showed enhancement in the cochlea and vestibule only (Downie *et al.*, 1994), a very rare site for acoustic neuroma (Brogan and Chakeres, 1990).

Conclusion

Isolated intracanalicular enhancement on MRI suggestive of acoustic neuroma must be viewed with caution in patients with an atypical history, even when MRI is not performed in the acute phase of the illness. Due consideration must be given to the possibility of inflammatory neuropathies.

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