

Role of Magnetic Resonance Imaging and Magnetic Resonance Angiography in Patients with Hemifacial Spasm

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Abstract

Vascular compression of the facial nerve is a well recognized cause of hemifacial spasm (HFS). In this study, we described the magnetic resonance imaging (MRI) and three-dimensional magnetic resonance angiography (MRA) techniques used and findings in 34 patients with hemifacial spasm. A vascular abnormality, defined as a vessel seen in close proximity, touching or compressing the facial nerve, was identified in 22 of the 25 patients (88%) who had both MRI and MRA studies. Vessels identified were anterior inferior cerebellar artery (59.2%), posterior inferior cerebellar artery (13.6%), vertebral artery (18.2%) and basilar artery (4.5%). All vascular abnormalities were ipsilateral to the side of the HFS. Only 3 of the 12 controls (25%) had a vascular abnormality in both MRI and MRA studies. One of the 9 HFS patients (11.1%) who had MRI only had an ipsilateral vascular abnormality. There is a role for combined MRI and MRA studies in the non-invasive evaluation of patients with HFS.

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Introduction

Hemifacial spasm (HFS), characterized by intermittent twitching of muscles supplied by one facial nerve is commonly due to compression of the facial nerve by blood vessel(s) adjacent to its root exit zone (REZ).¹⁻⁶ The facial nerve REZ refers to the part where facial nerve exits from the pons. Recently MRI and MRA techniques have been used to demonstrate vascular compression in HFS with good surgical validation.^{7,8} In this study, we report the incidence and characterize the vascular abnormality on MRI and MRA in patients with HFS.

Materials and Methods

Patients with a history of HFS who had MRI and/or MRA studies between June 1994 and June 1997 in the Singapore General Hospital were retrospectively reviewed. These studies were requested by their treating physicians to rule out compressive lesion on the ipsilateral facial nerve.

The imaging was carried out on a Siemens 1.0 Tesla Magnetom scanner. MRI studies included plain and

enhanced spin echo T1-weighted axial and coronal sections of posterior cranial fossa (TR 500 ms, TE 12 ms, 3 NEX, 3 mm slice thickness without gap) and T2-weighted axial sections of whole brain (TR 2200 ms, TE 80 ms, 1 NEX, 5 mm slice thickness with 1.5 mm gap) to exclude tumours of the cerebello-pontine angle and brainstem lesions such as infarcts or demyelination. A three-dimensional (3-D) time of flight (TOF) MRA (TR 39 ms, TE 6.5 ms, FA 20°, 1 NEX) was utilized to scan a 5.6 cm thick volume slab (46 partition cuts, FOV 20 cm, matrix 192 x 512), centred at the internal acoustic meati. This yielded sub-millimetre axial MRA source images. 5 mm maximum intensity projection (MIP) coronal sectional views, and multiplanar reformatting (MPR) in both oblique coronal and oblique sagittal planes along and perpendicular to the plane of the facial nerve centred at the REZ, were acquired during image post-processing of the MRA.

The MRI and MRA of 12 age- and sex-matched patients who were being evaluated for giddiness and who had no history or clinical finding of HFS were used as "controls". The MRI and MRA protocols for these

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controls were exactly the same as those patients with HFS. Thus a total of 24 facial nerves and adjacent vessels of these patients were studied.

The MRI and MRA were read independently by three authors (CLL, WL, JK) blinded to the side of the HFS. Discussion with consensus was made in cases where their initial assessment differed. In this study we defined a vascular abnormality as a vessel seen in contact with, in close proximity to, or compressing the REZ of the facial nerve. In the latter, there is deformation of the facial nerve by the vessel. We classified "close proximity" when the involved vessel was not in contact with but within 1 to 2 mm away from the facial nerve.

Results

Thirty-four patients were included. Twenty were females (58.8%) and 14 were males (41.2%). Their mean age was 48.4 years (range 29 to 79 years). The mean duration of HFS was 3 years (range 0.5 to 10 years). Twenty-four patients (70.6%) had left- and 10 (29.4%) had right-sided symptoms.

Twenty-five patients had both MRI and MRA while 9 had MRI alone. A vascular abnormality, all ipsilateral to the side of the HFS, was identified in 22 of 25 patients (88%) who had both MRI and MRA studies. Nine of these patients had vessels compressing the facial nerve at the REZ (Figs. 1a-1d) while the remainder had vessels in close proximity or touching the nerve (Figs. 2a-2d). Only 3 of the 12 patients without HFS (25%) or 3 of the 24 half-brain "controls" (12.5%) showed the presence of a vascular loop in close proximity to/contact with one of the facial nerves, but without compressing it. The difference in the incidence of finding a vascular abnormality between patients with and without HFS was statistically significant (Chi-square: $P < 0.01$, Table I). Of the 9 patients who had only MRI studies, only 1 patient (11.1%) showed ipsilateral vascular abnormality. This patient had a dolichoectatic basilar artery, which had swung into the cerebello-pontine angle ipsilateral to the side of HFS. The vessel was in close proximity with the facial nerve.

In HFS patients with both MRI and MRA studies, vessels that were in close proximity to, touching or compressing the ipsilateral facial nerve were identified as the anterior inferior cerebellar artery (13 patients),

vertebral artery (4), posterior inferior cerebellar artery (3) and the basilar artery (1). The origin of the vessel in one patient could not be determined (Table II).

We had good surgical validation in 1 patient who underwent microvascular decompression and who had immediate cessation of her spasm postoperatively.

Discussion

The introduction of MRI in the mid-eighties has revolutionized the evaluation of HFS. Computerized tomography (CT) and/or cerebral angiography were unable to show the relevant portion of the facial nerve and/or vessel satisfactorily.^{9,10} MRI, on the other hand, can demonstrate the course of the facial nerve from the brainstem to the internal auditory canal, and is free of attendant Hounsfield artifacts in the posterior fossa on CT scans and risks of angiography. In addition to detecting space-occupying lesions, vascular compression of the facial nerve at the REZ can also be readily identified.¹¹

In the past few years, development of MRA techniques have further advanced our ability to evaluate vascular structures non-invasively. In MRA, the contrast between moving blood and stationary tissues allows identification of the vasculature. The 3-D time of flight MRA technique has been shown to be sensitive and specific in demonstration of vascular abnormalities in HFS.^{7,8} Tien and Wilkins superimposed the MRA of the posterior circulation onto coronal T1-weighted images, which permitted better delineation of the relation between the artery and the facial nerve.¹² Stereoscopic short range MRA also allowed good visualization of vascular compression.¹³

The three-dimensional TOF MRA technique used in our study produces flow contrast by inflow of unsaturated spins into the imaging plane. We applied short range MIP using 5 mm thin coronal sectional views cutting across the pons, and MPR in both oblique coronal and oblique sagittal planes along the course of the facial nerve and centred at the REZ during image post-processing. This enabled the relation of vascular structures and

TABLE I: INCIDENCE OF UNILATERAL VASCULAR ABNORMALITY INPATIENTS WITH MRI AND MRA

Vascular abnormality	HFS (n = 25)	Controls (n = 12)
Present	22*	3*
Absent	3	9

HFS: hemifacial spasm

* Chi-square test: $P < 0.01$

TABLEII: VESSELS IN CLOSE PROXIMITY TO, TOUCHING OR COMPRESSING THE IPSILATERAL FACIAL NERVE IN PATIENTS WITH HEMIFACIAL SPASM

Identified vessel	Number (n = 22)	%
AICA	13	59.2
VA	4	18.2
PICA	3	13.6
BA	1	4.5
Undetermined	1	4.5

AICA: anterior inferior cerebellar artery; PICA: posterior inferior cerebellar artery; VA: vertebral artery; BA: basilar artery

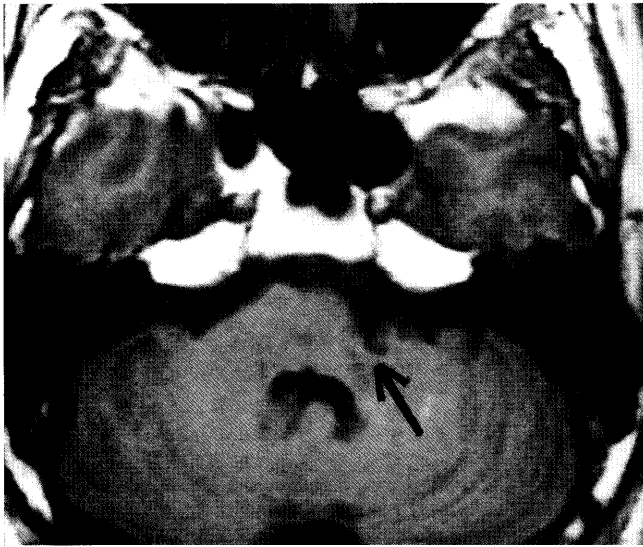


Fig. 1a. Axial T1-weighted scan of a 50-year-old gentleman with left hemifacial spasm, showing distortion of the left lateral pons (arrow).

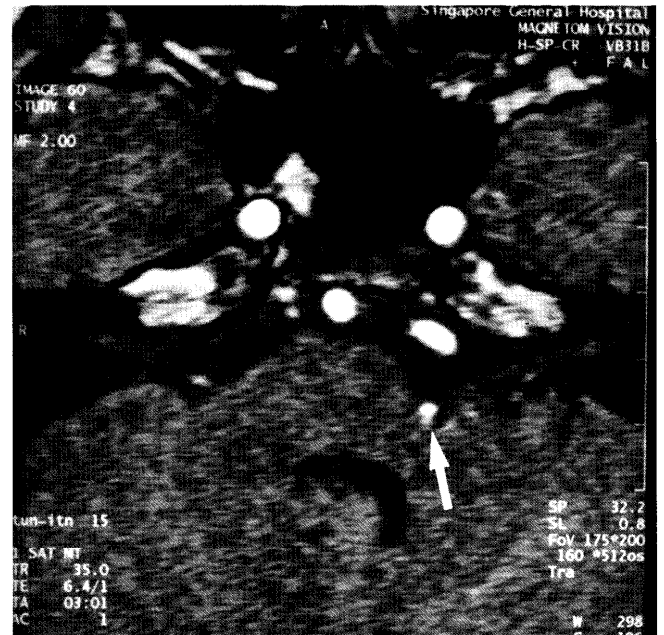


Fig. 1b. Corresponding axial MRA source image clearly demonstrated a high signal vascular loop (white arrow) causing the compression on the REZ of the left facial nerve.

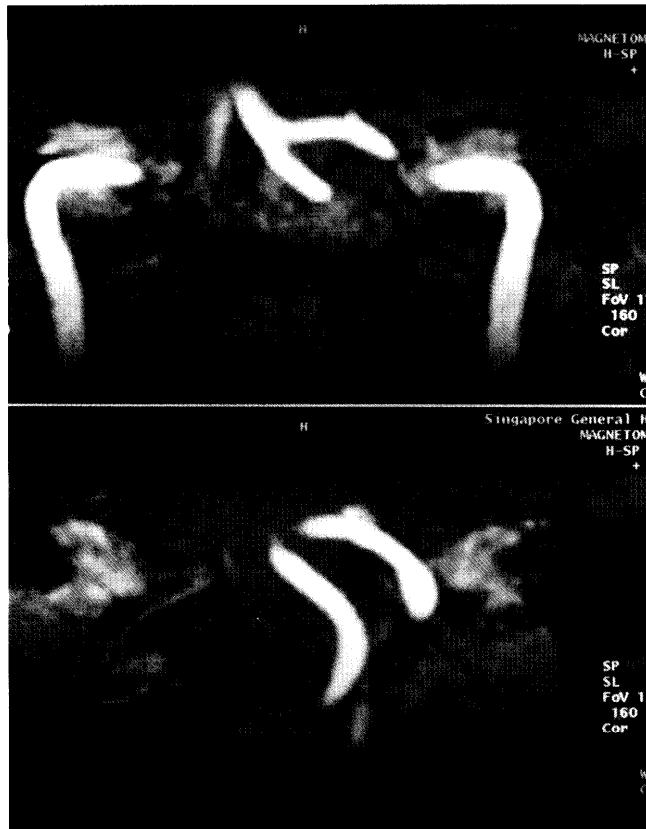


Fig. 1c. Consecutive MIP coronal sectional views of the MRA identified the arterial loop (black arrows) as the left posterior inferior cerebellar artery, arising from a dolichoectatic vertebrobasilar system.

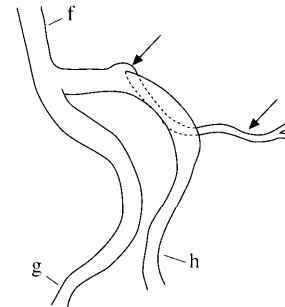


Fig. 1d. Semantic drawing of the vertebrobasilar system as depicted on the MR4 MIP views in Figure 1c. (f basilar artery, g: right vertebral artery, h. left vertebral artery)

facial nerves to be identified and explains the high yield of vascular abnormality in our study. Twenty-three out of 34 patients (67.7%) with HFS had vascular abnormality correctly identified ipsilateral to the side of the HFS. If we excluded patients with MRI done alone, this figure

improved to 88% (22 out of 25). The coronal MIP images allowed easy identification of the offending vessel, compared to tedious review of the source images especially where the vascular loops were long and tortuous. The utility of the MPR images rests in its ability to demonstrate the relation of the facial nerve to the offending vessel.

The predominant left-sided involvement could reflect anatomic variations. For instance, the left vertebral artery is bigger than the right in 40% of normal individuals.¹⁴

Some authors have suggested that vascular contact alone may not be sufficient to produce hemifacial spasm as 6% to 20% of controls had radiologic demonstration of abnormal vessels.¹⁵⁻¹⁷ In our study, 12.5% of the half-brain controls had neurovascular conflict. However the finding of a significantly higher incidence of ipsilateral MRA abnormality in patients with HFS, compared to those without, strongly suggested that the vascular abnormality on MRA was very likely the cause of patients'

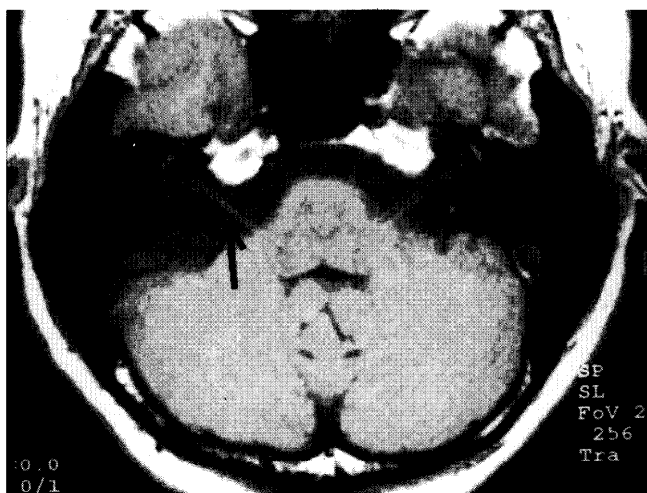


Fig 2a. Axial T1-weighted scan of a 56-year-old lady with right hemifacial spasm excluded the presence of a mass at the right cerebello-pontine angle (arrow).

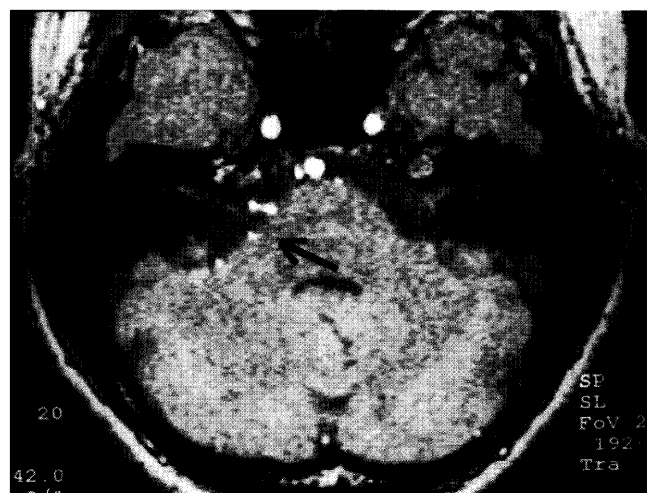


Fig 2b. However, the axial MRA source image showed a curvilinear high signal vascular loop (arrow) in close proximity to the REZ of the right facial nerve.

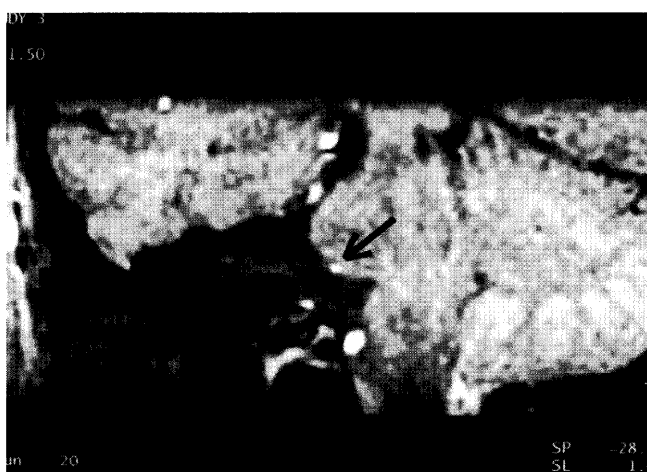


Fig 2c. Oblique coronal reformatted MRA image along the plane of the right facial nerve (arrow in b) clearly depicted the high signal vessel in contact with the REZ of the right facial nerve.

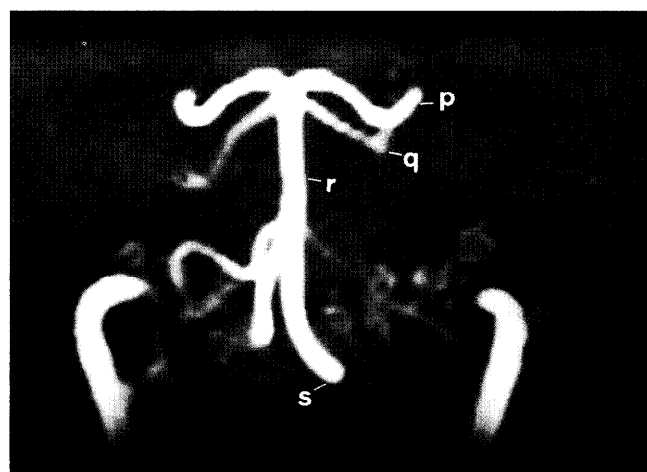


Fig 2d. Thin and thick slab MIP coronal sectional views of the MRA readily identified the vascular loop (black arrow) as the right anterior inferior cerebellar artery. (p: left posterior cerebral artery, q: left superior cerebellar artery, r: basilar artery, s: left vertebral artery)

symptoms. Ideally, MRI and MRA abnormalities should be correlated with surgical findings, but unfortunately most of our patients were averse to surgical intervention due to their cultural beliefs.

The need for a routine MRI to exclude mass lesions in patients with clinically classic HFS is sometimes debatable as most surgical series showed that tumours were uncommonly identified. In a survey by 190 investigators comprising treatment records of 1676 HFS patients, only 9 tumours were found in 881 patients (1.02%) who had neuroimaging studies.¹⁸

Digre et al¹⁰ reviewed 1688 cases of HFS from the literature. In 539 cases for which an aetiology was specified, only 6% were not due to compression by arterial structures. In these cases, the facial nerve was compressed by tumours, venous structures and bony abnormalities. Tumours included extra-axial epidermoids, neuromas, meningiomas and astrocytomas of the

cerebello-pontine angle. Basilar impression, cranio-occipital malformation, and Paget's disease were the aetiology in a few. Another rare cause of hemifacial spasm is a large posterior fossa mass with brainstem compression.¹⁹ In another large series by Kobata et al²⁰ who reported 8 cases (out of 926 patients) of surgically proven HFS in patients younger-than 30 years of age, thickened arachnoid membranes were found encasing and compressing the facial nerve and the surrounding artery. Unlike older patients, there was no finding of elongated or tortuous arteries in these patients.

Hence one cannot presume that all patients with classical HFS are due to vascular compression of facial nerve. MRI of the brain, being more sensitive than CT scan in identifying compressive structural lesions, is the imaging modality of choice in this group of patients, especially for those who have no other neurological

signs. Given the low incidence of tumour in the literature and the small numbers in our series, it was not unexpected that none of our patients had tumour. Nevertheless, we felt that it was worthwhile performing combined MRI and MRA in patients with HFS, since the finding of a compressive mass rather than a vascular abnormality would probably have altered the management of these patients.

In conclusion, our study using MRI and MRA had shown that a statistically significant proportion of our patients had a vascular abnormality ipsilateral to the side of hemifacial spasm, with one case that was surgically correlated. The anterior inferior cerebellar artery was the most common offending vessel identified. In view of the immensely different management of patients with HFS due to tumours (albeit uncommon) and the high sensitivity of the MRA technique in detecting vascular abnormalities, we recommend that MRI together with MRA be incorporated into the noninvasive evaluation of patients with HFS.

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