Short report

Magnetic resonance imaging in the management of resistant focal epilepsy: pathological case report and experience of 12 cases

R GRANT*, D M HADLEY,* B CONDON,* D DOYLE,† J PATTERSON,* I BONE,‡
S L GALBRAITH,§ G M TEASDALE§

From the Magnetic Resonance Unit, Department of Neuropathology,† Department of Neurology,‡ Department of Neurosurgery,§ Institute of Neurological Sciences, Southern General Hospital, Glasgow, UK*

SUMMARY Clinical improvement in epilepsy following temporal lobectomy is more often obtained when an abnormality is found on subsequent histological examination. Pre-operative MRI demonstrated an abnormal signal in the temporal lobe of a patient with pathologically proven mesial temporal sclerosis with microvascular anomaly. MRI may therefore be helpful in the selection of patients for temporal lobectomy. MRI findings of 12 patients with resistant focal epilepsy are reviewed. A wide range of T1 and T2 weighting is suggested to maximise selection of patients.

In 30–40% of patients with complex partial seizures, control of epilepsy is difficult and operation may be considered.¹ Before proceeding to operation, it is necessary to demonstrate a discharging focus on electro-encephalography. It is also reassuring to have radiological proof of an abnormality at the same site.²

Magnetic Resonance Imaging (MRI) may be more sensitive in detecting temporal lobe pathology than other imaging techniques.^{3,4} We studied 12 patients with resistant focal epilepsy who were being considered for operation to assess whether MRI aids diagnosis or improves management.

Case report

A 25 year old, right handed female factory worker, with a history of febrile convulsions as an infant, subsequently developed complex partial seizures, with occasional secondary generalisation, at the age of seven years. These seizures

took the form of an unpleasant sensation of tenseness in her stomach, associated with a stare. If these feelings lasted longer than 30 seconds they then developed into a grand mal seizure. There was no obvious relationship to menstrual cycle, no history of birth trauma or hypoxia, meningitis or head injury and no family history of epilepsy. Despite numerous alterations in her drug regimen over the years, minor seizures occurred 4–5 times per day and became generalised several times per week. Carbamazepine and phenobarbitone levels had been closely monitored and were within the range considered to be therapeutic.

Routine, sleep, and sphenoidal electro-encephalograms, demonstrated a right anterior temporal focal abnormality, but also minor abnormal changes on the left, of uncertain significance. There were also minor left sided abnormalities on psychometric testing. The presence of these bilateral disturbances made more difficult the decision about operation. Cranial computed tomography (CT) with contrast (EMI 1010 scan) did not reveal any abnormality. MRI was carried out, using a standard head coil in a Picker vista 1100, 0.15T resistive magnet operating at 6.38 MHz. T2 weighted Spin Echo (SE2200/80) and T1 weighted Inverse Recovery (IR2000/600/40) sequences provided multiple 8 mm contiguous axial slices. Single slice, seven echo (Carr-Purcell) axial sequences, through the temporal lobes were also performed to obtain computed T1 and T2 values. There was not an obvious abnormality on the initial SE and IR sequences (fig 1a); however, the Carr-Purcell sequence clearly identified

Address for reprint requests: Dr R Grant, Southern General Hospital, Institute of Neurological Sciences, 1345 Govan Rd, Glasgow G51 4TF, UK.

Received 28 October 1986. Accepted 28 November 1987

Fig 1 (a) 2200/80 pixel intensity profile shows similar intensity in both temporal lobes. (b) SE1500/168 pixel intensity profile shows increased signal intensity in right temporal lobe.

an abnormality in the medial aspect of the right temporal lobe on the SE1500/168 images (fig 1b). T1 and T2 measurements in the abnormal region of the right temporal lobe were 13% and 14% higher, respectively, than a comparable area in the opposite, normal, temporal lobe.

A standard temporal lobectomy was performed, with excision of the hippocampus and amygdala and disconnection of the fimbria. Since operation, the patient continued her previous medication but has remained free from seizures for 6 months.

Macroscopically, an area in the excised hippocampus (approx. 4 mm³) appeared abnormally white, and on microscopy there was complete loss of neurons in Sommer's sector, with hippocampal atrophy. Lymphocytes and corpora amylacea were seen surrounding medium and small deep cortical blood vessels over an area of 5 mm³ within the white matter. These findings were compatible with a vascular abnormality in the white matter of the uncus (fig 2a) with secondary hypoxic damage to the hippocampus (2b Ammon's horn).

Review of cases

Including this case, we have now studied a total of 12 patients with intractable focal epilepsy, who were

being assessed for possible temporal lobectomy. Abnormalities were seen in seven including the above case. In three of these patients a focal abnormality in one temporal lobe, consistent with mesial temporal sclerosis was demonstrated on MRI. The abnormality was best seen on a SE1500/120 sequence in two cases and on the SE1500/168 sequence in the third patient. Two of these patients had temporal lobectomy and subsequent histological studies confirmed the diagnosis. These abnormalities have not been observed in any of the 68 normal volunteers we have imaged nor in patients imaged for other pathology outwith this area. The MRI scans of the other patients demonstrated a left temporo-parietal tumour in one, a large previously unrecognised left subdural haematoma in a second, and showed right sylvian and occipital infarcts in the third. The MRI of a patient with multifocal seizures demonstrated generalised cortical atrophy and cortical biopsy showed histological changes suggestive of cortical dysplasia. The remaining five patients had a normal MRI and operation, so far, has not been carried out.

Fig 2 (a) Vascular abnormality in the uncus ($\times 50$). (b) Hypoxic damage in Sommer's sector of hippocampus ($\times 50$).

Discussion

Mesial temporal sclerosis is the most frequent cerebral abnormality found at necropsy in chronic epileptic patients, and occurs as a result of hypoxic damage sustained either at birth or during febrile convulsions in infancy. Swelling of the astrocytes in the pyramidal layer cells of the hippocampus, occurs early in the course of temporal lobe epilepsy and eventually develops into scarring which affects the medial temporal grey matter of the uncus, amygdaloid nucleus and hippocampus.⁶ If such changes are seen when the temporal lobe is histologically examined and if the affected area appears to have been completely resected there is a good chance that the patient will be subsequently free from seizures.⁷ By contrast, postoperative prognosis is poor if an abnormality is not found on histological examination of the temporal lobe, which is the case in approximately 20% of operations performed for temporal lobe epilepsy.⁸ It would therefore be valuable if mesial temporal sclerosis could be identified by an imaging technique such as MRI.

The MRI of the patient described in the case report demonstrated a non space occupying structural lesion in the medial temporal lobe, corresponding with the site identified as causing the main electro-encephalographic abnormality. There was not an abnormality in the MRI of the contralateral temporal lobe. The finding of a unilateral structural abnormality on MRI confirmed the site of the lesion and a decision to proceed with operation resulted.

This case stresses the importance of choosing the appropriate imaging sequence for the clinical situation, as the abnormality could have been missed easily if the Carr-Purcell sequence had not been used. This sequence provides a set of single slice images at seven different echo times (24, 48, 72, 96, 120, 144, 168 ms) and is performed at a repeat time of 500 ms and 1500 ms (total imaging time 10.6 min), thus covering as wide a range of T1 and T2 dependent weighting as possible.⁹

MRI may play an important role in management of patients with temporal lobe epilepsy where a structural lesion, such as mesial temporal sclerosis, is suspected or the lateralisation of the disturbance is in

doubt. It is sensitive to small changes in brain water content, and therefore, gives better grey matter/white matter contrast than CT.¹⁰ We would suggest that, if mesial temporal sclerosis is considered, a wide range of T1 and T2 weightings should be employed, imaging through the temporal lobes, such as a seven echo Carr-Purcell sequence. The option to perform coronal slices is also readily available and makes the presence of associated temporal lobe atrophy more easily identifiable in an area where interpretation of CT scans may be hampered by streak and bone hardening artefact.¹¹ MRI is useful in the diagnosis and management of patients with epilepsy refractory to medical treatment.

This study was supported by grants from the Medical Research Council (ID81), the Greater Glasgow Health Board and the University of Glasgow and the Scottish Hospitals Endowment Research Trust.

References

- 1 Mattson RH, Gamer JA, Collins JF, *et al.* Comparison of carbamazepine, phenobarbital phenytoin and primidone in partial and secondarily generalized tonic-clonic seizures. *N Engl J Med* 1985;**313**:145–51.
- 2 Cahan LD, Engel J (Jr). Surgery for epilepsy: a review. *Acta Neurol Scand* 1986;**73**:551–60.
- 3 Laster DW, Penry JK, Moody DM, Ball MR, Wicofski RL, Riela AR. Chronic seizure disorders: contribution of MR imaging when CT is normal. *AJNR* 1985;**6**:177–80.
- 4 Latack JT, Abou-Khalil BW, Seigel GJ, Sackellares JC, Gabrielsen TO, Aisen AM. Patients with partial seizures: evaluation by MR, CT and PET imaging. *Radiology* 1986;**159**:159–63.
- 5 Sano K, Malamud N. Clinical significance of sclerosis of the cornu ammonis. *Arch Neurol Psychiat* 1953;**70**:40–53.
- 6 Evans MC, Griffiths T, Meldrum BS. Kainic acid seizures and the reversibility of calcium loading in vulnerable neurons in the hippocampus. *Neuropathol Appl Neurobiol* 1984;**10**:285–302.
- 7 Falconer MA. Mesial temporal (Ammon's horn) sclerosis as a common cause of epilepsy. Aetiology, treatment and prevention. *Lancet* 1974;**2**:767–70.
- 8 Meldrum BS, Corsellis JAN. Epilepsy. In: Hume Adams J, Corsellis JAN, Duchon LW eds. *Greenfield's Neuropathology 4th ed.* Edward Arnold Publishers, 921–50.
- 9 Farrar TC, Becker ED. In: *Pulse and Fourier Transform NMR. Introduction to Theory and Methods.* Academic Press 1971; 25–7.
- 10 Bydder GM, Steiner RE, Young IR, Hall AS, Thomas DJ, Marshall J, Pallis CA, Legg NJ. Clinical NMR imaging of the brain: 140 cases. *AJR* 1982;**139**:215–36.
- 11 Bydder GM, Steiner RE, Thomas DJ, Marshall J, Gilderdale DJ, Young IR. Nuclear magnetic resonance imaging of the posterior fossa: 50 cases. *Clin Radiol* 1983;**34**:173–88.