Mesial temporal sclerosis

D H Jogi
MB BCh, FCRad (Diag) (SA), FRCR (Lond)
N17 East Rand Private Community Hospital Springs

M Patel
MB ChB, FCRad (Diag) (SA)
Department of Radiology Chris Hani Baragwanath Hospital Johannesburg

Introduction

Mesial temporal sclerosis is the commonest cause of partial complex seizures. The aetiology of this condition is controversial, but it is postulated that both acquired and developmental processes may be involved. Familial cases have also been reported.

Magnetic resonance imaging (MRI) is the imaging investigation of choice for the diagnosis and has been shown to be highly sensitive and specific. Once diagnosed, medical treatment is successful in 25% of cases, whilst anterior temporal lobectomy is effective in 70 - 95% of patients.[1]

Case report

The case under discussion involved a 42-year-old woman. She had had numerous previous admissions related to her alcoholism, but presented on this occasion with new onset partial complex seizures and secondary generalisation. On clinical examination she had impaired short-term memory, however, no focal neurology was present. An EEG was performed and was assessed as normal. A MRI scan was done which revealed abnormal high signal in the right hippocampus on the T2-weighted and FLAIR sequences. The head, body and tail of the hippocampus were involved, with associated poor grey-white matter differentiation. In addition, there was atrophy of the hippocampus and fornix, with dilatation of the temporal horn (Figs 1 - 4).

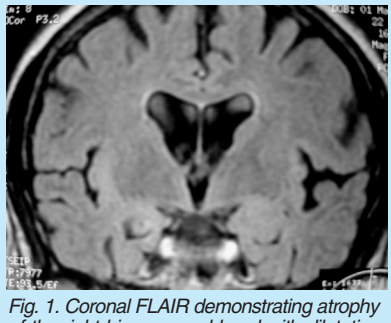


Fig. 1. Coronal FLAIR demonstrating atrophy of the right hippocampal head with dilatation of the temporal horn.

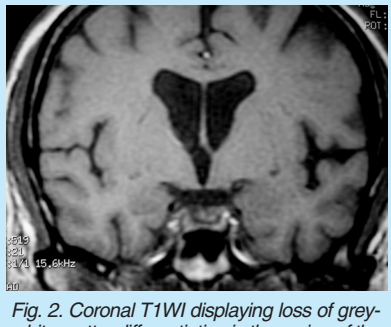
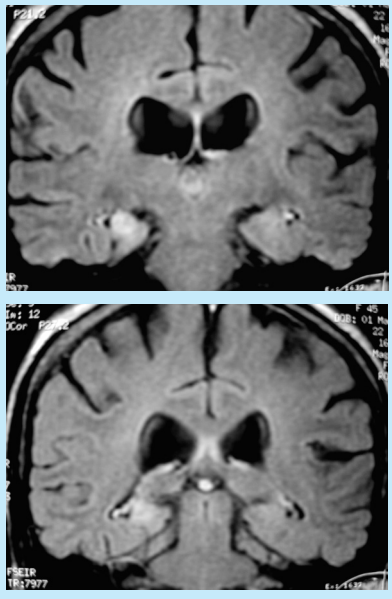


Fig. 2. Coronal T1WI displaying loss of grey-white matter differentiation in the region of the right hippocampal head.



Figs 3 and 4. Coronal FLAIR sequences exhibiting atrophy of the right hippocampal body and tail as well as the right fornix.

Discussion

Mesial temporal sclerosis (MTS) is the commonest cause of temporal lobe epilepsy. Pathologically, there is neuronal loss, atrophy and hippocampal gliosis. These findings are characteristic of this disorder and are thought to serve as an epileptogenic substrate. The histologic substrates can be divided into four other major catergories: (*i*) tumours; (*ii*) disorders of neuronal migration and cortical organisation; (*iii*) vascular malformations; and (*iv*) neocortical sclerosis attributable to brain injury (trauma, infection, inflammation, or infarction).[1,2] In MTS, dual pathology occurs in 15% of cases, with cortical dysplasia being the commonest.[1]

MRI has the ability to detect subtle alterations in cortical architecture and changes in signal intensity and is therefore the most sensitive and specific imaging technique for non-invasive identification of these epileptogenic foci.[2]

In order to achieve accurate MRI interpretation, it is essential to know the regional anatomy of the inferomedial temporal lobe and its related structures (Fig. 5). The hippocampus comprises the head (pes), body and tail. The hippocampal/dentate com-

plex is located in the medial aspect of the temporal lobe, posterior to the amygdala. It is separated from the amygdala by the uncal recess of the temporal horn and the alveus and its long axis is parallel to temporal gyri. Two interlocking 'C'-shaped sheets of cortex form the hippocampal /dentate complex: (*i*) cornis ammonis (CA1, CA2, CA3 and CA4); and (*ii*) dentate gyrus (Figs 5 and 6).

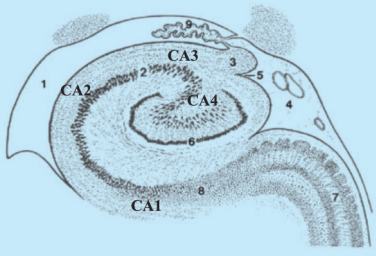


Fig. 5. 1. Temporal horn; 2. Cornu ammonis (Ammon's horn, hippocampus proper); 3. Fornix; 4. Subarachnoid space; 5. Hippocampal sulcus; 6. Dentate gyrus; 7. Parahippocampal gyrus (containing entorhinal cortex); 8. Subiculum; 9. Choroid plexus.[3]

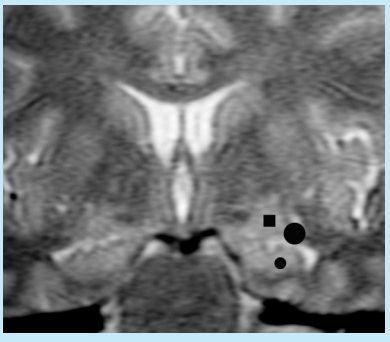


Fig. 6. Coronal T2 at the level of the interpeduncular cistern showing the amygdala (large square), uncal fissure (large dot), hippocampal head (small dot)

On MRI, the hippocampal head is seen in the same coronal plane as the interpeduncular cistern (Fig. 6). The body of the hippocampus is seen at the level of the midbrain (Fig. 7). It is ovoid in shape and is the most uniform portion. It lies inferior to the choroidal fissure and is separated from the parahippocampal gyrus by the hippocampal fissure. The tail of the hippocampus is located at or behind the midbrain where it is seen adjacent to the crura of the fornices (Fig. 8).

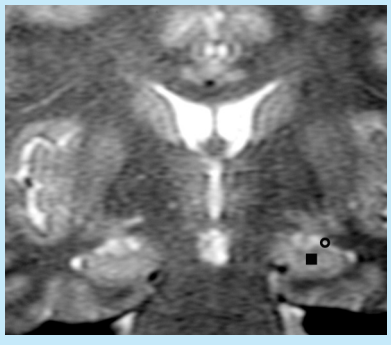


Fig. 7. Coronal T2 WI at the level of the midbrain, demonstrating the ovoid hippocampal body (small sqaure) under the choroidal fissure (circle).

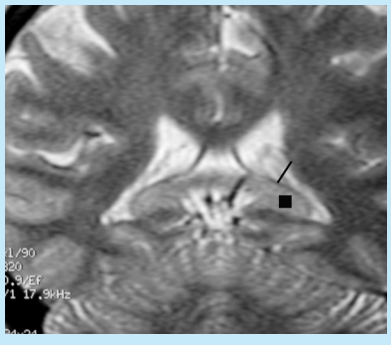


Fig. 8. Coronal T2 WI just posterior to the midbrain illustrating the fornix (line) and hippocampal tail (square).

Various MRI sequences are recommended for evaluation of the temporal lobe. These include T1WI sagittal images for the localisation of the hippocampus and thin-section high-resolution T2WI and FLAIR acquisitions angled perpendicular to the long axis of the hippocampus (Table I). Contrast is unnecessary unless there is a focal lesion.[1] A spoiled gradient recalled (SPGR) echo sequence using 1.5 mm cuts in the oblique coronal plane can also be done. This provides a high-resolution T1-weighted volume data set which can be reformatted in any plane, and can also be used to measure hippocampal volumes and co-register functional data.[4,5]

Primary findings in MTS are hippocampal atrophy (recognised by asymmetry in the case of unilateral atrophy) and increased signal intensity of the hippocampus on T2WI. This is best appreciated on the coronal FLAIR sequence, which suppresses out the cerebrospinal fluid (CSF) signal from the uncal recess and the choroidal fissure thus avoiding false positive high signal changes. Recent studies have shown that visual MRI interpretation of these features has sensitivities of 87 - 100%. [1,4]

There are numerous secondary MR features that support the diagnosis of MTS (Table II). These include temporal horn dilatation, loss of hippocampal internal architecture, decreased hippocampal signal on T1WI and poor parahippocampal grey-white matter definition. Other findings include ipsilateral atrophy of the temporal lobe, thalamus, fornix and mamillary body. These secondary features are present in 40 - 60% of

Table I. MRI imaging protcols

- High-resolution MR of the temporal lobes
- T1 WI sagittal for localising the hippocampus
- Coronal high-resolution T2 WI and FLAIR perpendicular to hippocampal axis (3 - 4 mm)
- Coronal SPGR T1 perpendicular to hippocampal axis (1.5 mm)

Table II. Imaging findings

Primary signs	Secondary signs
Hyperintense signal on T2 and FLAIR sequences	Enlarged temporal horn of lateral ventricle Loss of internal architecture Decreased signal on T1WI
Atrophy of hippocampus	Poor grey-white matter definition Fornix, mamillary body, temporal lobe and thalamic atrophy

patients with MTS. On their own, the above signs are unreliable, but in conjunction with the primary findings the diagnostic accuracy is improved.[4]

Decreased apparent diffusion coefficient (ADC) levels may be seen on diffusion-weighted imaging. MR spectroscopy would show reduced N-acetylaspartate levels in the ipsilateral mesial temporal lobe assisting in the lateralisation of temporal lobe epilepsy (TLE), even in cases with negative MR images.[1,4,5] There is bilateral involvement in 20% of cases and in these cases MRI-based hippocampal volumetry has been shown to quantitatively indicate the presence of hippocampal volume loss.[1,6]

MRI also provides information on the predictive value concerning neurologic outcome in patients undergoing temporal lobe surgery. MRI can identify hippocampal volume loss and coexisting extrahippocampal lesions which predict an unfavourable postoperative neurocognitive outcome.[1,4]

The MRI findings of the patient discussed in this case report are compatible with MTS. She is currently on medical treatment and is being followed up monthly.

Conclusion

MRI is the radiological investigation of choice for diagnosing MTS. Familiarity with the regional medial temporal lobe anatomy is important for correct MRI interpretation. Coronal high-resolution FLAIR is the best sequence to diagnose MTS, where hyperintensity and atrophy of the hippocampus are the most sensitive signs.

References

1. Osborn AG, Cooper JA, Castillo M, *et al. Diagnostic Imaging – Brain*. St Louis: WB Saunders, 2004.
2. Jack CR jun. Magnetic resonance imaging in epilepsy. *Mayo Clin Proc* 1996; **71:** 695-711.
3. Hippocampus. www.laxtha.com/bhbae/hippocampus/hippocampus.htm. (Last accessed 4 April 2005).
4. Connor EJ, Jarosz JM. Magnetic resonance imaging of patients with epilepsy. *Clin Radiol* 2001; **56:** 787-801.
5. Cascino G. Clinical correlations with hippocampal atrophy. *Magn Reson Imaging* 1995; **13:**1133-1136.
6. Arı´stides A. Capizzano, Kenneth DL, *et al.* Temporal lobe epilepsy: Qualitative reading of 1H MR spectroscopic images for presurgical evaluation. *Radiology* 2001; **218:**144-151.