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Asymmetric Mamillary Bodies: MR Identification

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Summary: We report three cases in which there was marked asymmetry of the mamillary bodies, noted on MR in two and at autopsy in the third. Based on its proposed pathogenesis, we suggest that this finding could have value in locating temporal lobe disease in patients with intractable epilepsy.

Index terms: Hypothalamus; Brain, anatomy; Brain, magnetic resonance; Seizures; Brain, temporal lobe

We recently encountered a patient in whom there was complete absence of one mamillary body. In researching this finding we found clear references to this observation at autopsy in patients with epilepsy. We report three such cases, two seen on magnetic resonance (MR), and review the literature with respect to this finding.

Case 1

A 4½-year-old white boy presented with a history of staring spells lasting 15 to 20 minutes, falling without provocation, and aggressive behavior. After a full-term, normal spontaneous vaginal delivery, the patient had an episode of respiratory distress at 1 month of age and was admitted to the neonatal intensive care unit of another hospital.

An electroencephalogram showed frequent spike-wave complexes in the right parietal region, suggesting a cerebral lesion. MR evaluation included sagittal, coronal, and axial T1-weighted scans (600/15/2 [repetition time/echo time/excitations]) with coronal and axial T2-weighted images (2500/30). Section thickness was 5 to 6 mm. There was no parenchymal abnormality seen within the temporal or parietal lobes. However, the scan showed absence of the right mamillary body (Fig 1).

Case 2

A 74-year-old mentally retarded white woman was admitted to an outside institution with lower-gastrointestinal bleeding. Past medical history was remarkable only for her long history of seizures and mental retardation. The brain was noted at autopsy to have atrophy of the right hippocampus grossly and absence of the ipsilateral mamillary body (Fig 2). Incidentally noted was an old occipital infarct. Microscopic evaluation of the hippocampal formation was not described in the record.

Case 3

This 33-year-old man was evaluated at our institution for intractable epilepsy. His seizures began 10 years before his presentation and were characterized as partial complex. He had a history of head trauma at 5 years of age with loss of consciousness. His evaluation included monitored electroencephalogram, brain MR, hexamethyl-propyleneamine oxime single-photon emission computed tomography, neuropsychologic testing, and Wada evaluation. The interictal single-photon emission computed tomography was nonlocalizing.

MR included axial, coronal, and sagittal T1-weighted images (500/15) and coronal T2-weighted scans (2500/30) using a 5- or 6-mm section thickness. The right hippocampus had increased signal compared with the left, consistent with hippocampal sclerosis (Fig 3). The mamillary bodies appeared asymmetric on the coronal T1-weighted scan with the smaller ipsilateral to the diseased hippocampus.

A limited right temporal resection was performed. Two postoperative MR scans were obtained. On the second, which was 1 year after surgery, an additional sequence was obtained (magnitude preparation–rapid acquisition gradient echo) that allowed 1.0-mm sections through the mamillary bodies with unlimited options for planar reconstruction of the three-dimensional volume. On this scan the asymmetry of the mamillary bodies was again demonstrated.

Discussion

The mamillary bodies are the most posterior structure of the hypothalamus and can be seen as paired, rounded structures in the interpeduncular cistern just anterior to the midbrain on axial images. They are small structures, usually 4 to 5 mm in diameter, and are symmetric in healthy persons. On sagittal MR they can be identified near the midline as a bulbous expansion posterior and inferior to the optic chiasm. They are a component of the limbic system and as such receive input from the associative fibers of the

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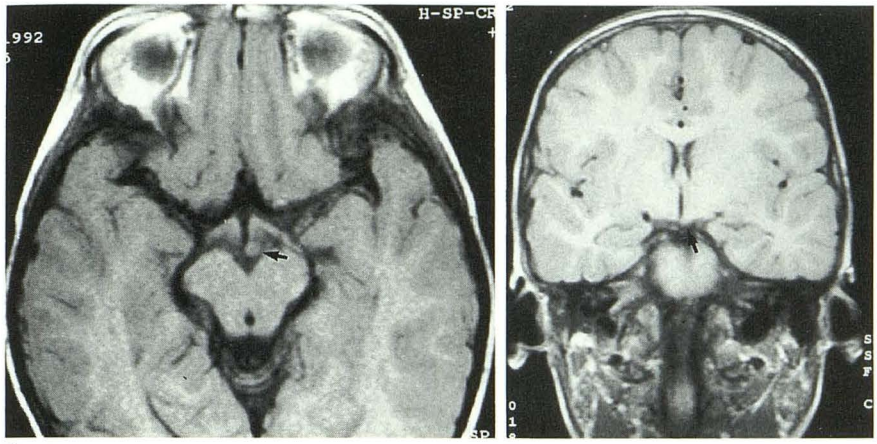

Fig. 1. Case 1: 4½-year-old boy with seizures. The axial (*A*) and coronal (*B*) MR scans (600/15) demonstrated marked asymmetry of the mamillary bodies in this patient with seizures. The normal-appearing left mamillary body is indicated by an *arrow*.

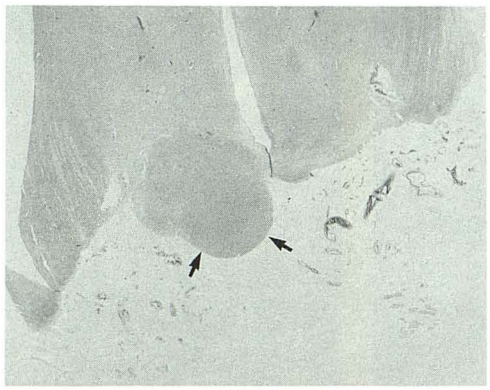

Fig. 2. Case 2: 74-year-old woman with history of seizures. The tissue section demonstrated absence of the patients left mamillary body in this patient with long-standing epilepsy. The normal mamillary body is indicated by *arrows*.

postcommisural fornix. Based on autoradiographic studies in animals, it appears that these afferent fibers originate entirely within the subiculum, not the hippocampus as previously assumed (1, 2). The subiculum is part of the hippocampal formation and lies along the medial, superior curve of the parahippocamal gyrus in close proximity to the hippocampus. It is characterized by its prominent superficial medullary laminae, which may be visible as a band of increased signal on MR images (3). Each mamillary body projects to a single fiber tract, the mamillary tract, which divides into mamillothalamic and mamillotegmental tracts.

Most clinical study of the mamillary bodies has involved acquired disease, specifically Wernicke encephalopathy. In such cases the mamillary bodies are small and soft and demonstrate reactive gliosis histologically (4). Fusion and hypoplasia have been reported rarely at autopsy, usually in association with gross brain malformations (5, 6). However, several previous reports suggest an association between epilepsy and mamillary-body atrophy. One patient with long-standing epilepsy, described in a neuropathology text, had at autopsy a unilateral small mamillary body with ipsilateral temporal and thalamic atrophy (7). Lindboe et al, in a more comprehensive description of this finding, point out an association in three autopsy cases between mamillary body atrophy and histologically evident ischemic and anoxic damage of the ipsilateral Ammon's horn of the hippocampus (4). In two cases the atrophy was unilateral and in one bilateral. In all three of their cases there was no evidence of reactive gliosis of the small mamillary bodies, which would be anticipated in cases of Wernicke encephalopathy. On this basis they argue that mamillary body atrophy reflects deafferentation of the mamillary body secondary to a previous insult to the hippocampal formation, specifically the subiculum.

Although our first case had a seizure focus ipsilateral to the small mamillary body, it was located in the parietal lobe. However, scalp electroencephalographic location may be inaccurate because of wide propogation of electrical activity. It is precisely this limitation of scalp electroencephalography that requires other means of location before epilepsy surgery. Although partial fusion of the mamillary bodies has been reported as an incidental finding at autopsy, unilateral agenesis has not (5). Based on these previous accounts, the patients' histories of seizures seems most likely to be related to the small mamillary body, despite normal-appearing temporal lobes on MR. The second case also had a history of epilepsy but with hippocampal atrophy ipsilateral to the small mamillary body. In the third case there was again hippocampal sclerosis, confirmed both by MR criteria and histologic evaluation of the surgical specimen, ipsilateral to the small

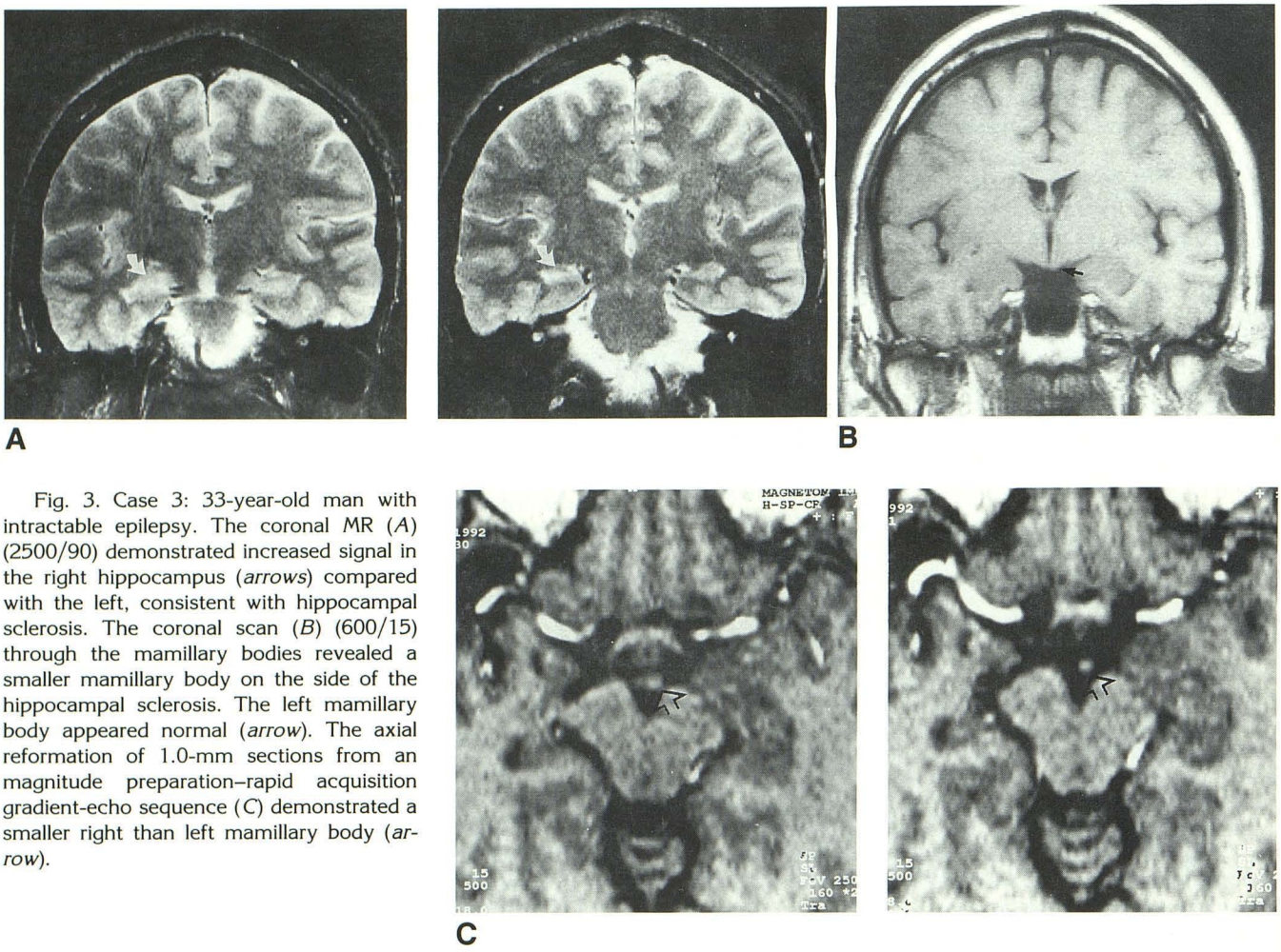

Fig. 3. Case 3: 33-year-old man with intractable epilepsy. The coronal MR (*A*) (2500/90) demonstrated increased signal in the right hippocampus (*arrows*) compared with the left, consistent with hippocampal sclerosis. The coronal scan (*B*) (600/15) through the mamillary bodies revealed a smaller mamillary body on the side of the hippocampal sclerosis. The left mamillary body appeared normal (*arrow*). The axial reformation of 1.0-mm sections from an magnitude preparation–rapid acquisition gradient-echo sequence (*C*) demonstrated a smaller right than left mamillary body (*arrow*).

mamillary body. The preoperative scan demonstrated mamillary asymmetry, which was better demonstrated on the postoperative scan. For that scan a technique that allowed very fine section thickness was available on our scanner. One could argue whether the surgical resection of the hippocampus exaggerated this asymmetry, but in any event it supports the proposed mechanism of secondary mamillary atrophy caused by deafferentation.

It may be difficult to assess mamillary body size and symmetry on conventional MR scanning, which uses section thickness on the order of 5 to 6 mm, unless the discrepancy is marked as in our first case. In a recent review article on the role of imaging in the evaluation of epilepsy, Bronen emphasized the importance of confident location of the seizure focus by neuroimaging in order to eliminate the need for invasive electroencephalographic monitoring (8). Although the hippocampus has received the lion's share of attention in such evaluations, the mamillary bodies could provide additional lateralizing information in those cases in which the subiculum as well as the hippocampus is injured.

In order to assess these small structures accurately, a thin-section technnique such as radiofrequency–spoiled gradient-echo or magnitude perparation–rapid-acquisition gradient-echo is necessary. Many sites are already using these techniques to evaluate hippocampal size, and the mamillary bodies are usually included in this volume acquisition.

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Please see the Commentary by Mirski on page 1336 in this issue.