TEMPORAL LOBE HYPOGENESIS ASSOCIATED WITH ARACHNOID CYST IN PATIENTS WITH EPILEPSY

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ABSTRACT - Objective: To determine the frequency of temporal lobe hypogenesis (TLH) associated with arachnoid cysts (AC) in patients with epilepsy. Method: We retrospectively revised 655 consecutive MRI scans from patients followed in our epilepsy clinic. We identified patients with temporal AC and then performed careful visual analysis in a workstation. Patients with evident expansive or destructive lesions were excluded. Results: Only 4 (0.6%) patients had AC in the left temporal lobe, all associated with TLH. In addition, there were also ipsilateral dysgenetic characteristics in the ipsilateral hippocampus including abnormal shape and axis, and hyperintense T2 signal. In one patient this hippocampal abnormality was bilateral. Conclusion: AC with TLH was rarely found in our patients with epilepsy and it was always associated with hippocampal dysgenesis. Although volumetric reduction of the temporal lobe can be observed in patients with epilepsy and hippocampal abnormalities, the presence of adjacent AC points to a malformative etiology.

KEY WORDS: hippocampal dysgenesis, cortical development malformation, partial epilepsy, seizures, magnetic resonance imaging.
The relationship between cysts and seizure foci, however, have proved negative.6,7

The objective of this study is to determine the prevalence of temporal lobe hypogenesis associated with middle fossa arachnoid cysts in our patients with epilepsy.

METHOD

We retrospectively revised 655 consecutive MRIs from patients with epilepsy from UNICAMP Clinical Hospital. MRIs were acquired in a 2Tesla scanner (Elscint Prestige®), and kept in optical disk for revision in a workstation (OMNIPRO®). Exclusion criteria were evident destructive or expansive abnormalities identified in MRI.

Acquisition parameters were: (1) Sagittal T1 spin echo, 6mm thick, flip angle= 180°; repetition time (TR)=430, echo time (TE)=12, matrix 200X350, field of view (FOV)=25X25cm; (2) Coronal images, perpendicular to long axis of hippocampus, defined on the sagittal images: (a) T2-weighted and proton density “fast spin echo” (FSE), 3mm thick, flip angle= 160°; TR=4800, TE=108/18, matrix 256X256, FOV=22X22cm; (b) T1-weighted inversion recovery (IR), 3mm thick, flip angle=200°; TR=2800, TE=14, inversion time (TI)=840, matrix 130X256, FOV=16X18cm; (3) Axial images parallel to the long axis of the hippocampi: (a) T1-weighted gradient echo, 3mm thick, flip angle=70°, TR=200, TE=5, matrix 180X232, FOV=22X22 cm; (b) T2-weighted FSE, 4mm thick, flip angle=120°, TR=6800, TE=129, matrix 252X328, FOV=21X23cm; (4) T1-weighted 3D gradient echo with 1mm isotropic voxel, acquired in the sagital plane for multiplanar reconstruction (1mm thick, flip angle=35°; TR=22, TE=9, matrix 256X220, FOV=23X25cm).

RESULTS

Only 4/655 (0.6%) from evaluated scans presented middle fossa arachnoid cysts, all in the left side and associated with signs of temporal lobe hypo-

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex, Age (years)</th>
<th>Epilepsy syndrome</th>
<th>Outcome</th>
<th>EEG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M, 13</td>
<td>Simple febrile seizure</td>
<td>good</td>
<td>Left temporal slow waves</td>
</tr>
<tr>
<td>2</td>
<td>F, 38</td>
<td>Mesial TLE</td>
<td>poor</td>
<td>Bitemporal epileptiform discharges</td>
</tr>
<tr>
<td>3</td>
<td>M, 53</td>
<td>GTCS sleep</td>
<td>good</td>
<td>Left temporal epileptiform discharges</td>
</tr>
<tr>
<td>4</td>
<td>F, 39</td>
<td>GTCS sleep</td>
<td>good</td>
<td>Not available</td>
</tr>
</tbody>
</table>

TLE, temporal lobe epilepsy; GTCS sleep, generalized tonic-clonic seizures during sleep.
genesis (Fig 1). A summary of patients characteristics is shown in Table 1.

Ipsilateral hippocampus had dysgenetic characteristics in all of them, with rounded shape, vertical axis and hyperintense T2 signal. In one patient this hippocampal abnormality was observed also in the contralateral hippocampus (Fig 2). The morphology of hippocampal abnormality found in these patients is quite different from that observed in hippocampal sclerosis.

DISCUSSION

Although temporal lobe abnormalities are frequently observed in patients with epilepsy, arachnoid cysts were rarely found among our patients. However, in the four patients with middle fossa arachnoid cysts, we found not only signs of temporal lobe hypogenesis, but also signs of hippocampal dysgenesis (bilateral in one of them).

Hippocampal dysgenesis is due to a malformation associated with abnormalities in the complex process of rotation and folding, which ends in abnormal shape, axis and internal structure8,9. Such findings have been reported in asymptomatic individuals from families with either febrile convulsions or temporal lobe epilepsy10,11. Recently, post-mortem pathological study of hippocampal malformation in an adult patient with temporal lobe epilepsy revealed abnormal position and complex convolutional malformations isolated to the hippocampal formation12. All these evidences point to an inherited abnormality in the mesial temporal region that is not necessarily associated with epilepsy.

Although temporal volume reduction can be found in patients with epilepsy and hippocampal abnormalities, the co-existence of arachnoid cysts indicates a malformative etiology. In the small number of patients with middle fossa arachnoid cysts identified in this study, the origin of the cyst can be attributed to temporal lobe hypogenesis.

REFERENCES