Comparison of Hippocampal sulcus width and cavities between patients with Alzheimer disease and nondemented elderly patients

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Purpose

The most common cause of dementia is Alzheimer disease (AD). The preclinical phase of AD is the subject of intense investigation, since prompt diagnosis could allow drug therapy to be started earlier, thereby improving the chances for a positive clinical response.

Clinical criteria provide a greater than 90% sensitivity for diagnosing dementia of any type, including AD, but they have a specificity of less than 70% for the actual diagnosis of AD and have significant limitations as:

- difficulty in patients with severe depression, aphasia, and apraxia.
- necessity of longitudinal clinical testing to distinguish between the early memory loss in normal aging (slow progression) and very early AD (more rapid progression).
- Other degenerating dementias can mimic AD on the basis of clinical criteria alone.

Neuropathologic changes underlying AD first occur in the medial temporal lobe.

Enlarged CSF spaces in the hippocampus have been noted in MRI imaging studies of the medial temporal lobe in aging and AD. These spaces include the perihippocampal fissures, uncal sulcus, and the hippocampal sulcus residual cavity (HC).

The hippocampal fissure is a fetal sulcus around which occurs the hippocampal folding. It begins as a small indentation in the primitive hippocampus at approximately the 10th week of fetal development. Later, the sulcus deepens, but between the 18th - 21st week of fetal development, it is obliterated almost completely by fusion between the cornu ammonis and the dentate gyrus. (Figure 1) on page 4

Hippocampal cavities are generally considered residual cysts resulting from lack of hippocampal fissure obliteration, and are regularly found in routine MRI imaging studies, believed to represent a normal variant reflecting CSF collection. Because brain atrophy results in enlargement of the CSF spaces, either hippocampal sulcus (HS) enlargement or an increase in the number or size of HC could be associated with medial temporal lobe atrophy (MTA) occurring in Alzheimer disease.

One study on this subject has showed that enlargement of the HS is associated with MTA in Alzheimer disease, but enlargement of the HC is not related with AD. Another one reported similar findings but also concluded that patients with extremely high HCs could be excluded from AD risk with 93% specificity.

The aim of this study was to compare the Hippocampal sulcus (HS) width and cavities (HC) between patients with AD and nondemented elderly subjects.
**Fig. 0:** No more than the most medial part of the hippocampal fissure persists as an open and shallow groove between the dentate gyrus and the subiculum, just below the fimbria and the fimbriodentate sulcus, on the medial surface of the hippocampus—the superficial hippocampal sulcus.

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Methods and Materials

The study performed on demented and control groups, each containing 36 patients. Demented group consisted of patients with diagnosis of AD. Mini-Mental State Examination (MMSE) score was recorded by the psychiatrist as an indicator of AD(<25). These patients were referred for MR imaging before initiating antidementia therapy.

In control group, there were nondemented elderly individuals referring by the same psychiatrist for evaluation by MRI for complains other than dementia (like headache, vertigo, dizziness, ..) These patients had normal MMSE (25<), and were age-matched with the patient group.

None of 72 subjects had evidence or history of structural brain alterations including mass, stroke, lacunar infarcts, hydrocephalus or demyelinating diseases.

**MR Imaging Protocol:**
MR imaging examinations were performed by using a superconductive magnet operating at 1.5T system and a routine imaging protocol for dementia for both groups (High-resolution 3D T1-weighted gradient-echo sequence, perpendicular to the long axis of the temporal lobe).

**Image Assessment:**
Two observers who rated the images of both groups, were blinded to clinical information.

The relation between prevalence, number, size of HCs and HS width with MTA score were examined.

**HS Width measurement:**
After choosing the best section displaying the fimbria, to evaluate maximal width of the fimbriosubicular distance we measured it linearly perpendicular to the visible longitudinal extent of the HS on both sides, at the anterior part of the hippocampal body, which was done either vertically or obliquely. *(Figure 1)* on page 7

In cases that the HS was too shallow to be measured, we considered it equal to 0 mm. *(Figure 2)* on page 7

**HC Measurement:**
HCs are defined as sharply demarcated cystic structures (isointense with CSF) localized at the apex of the hippocampal fold. *(Figure 3)* on page 8
The greatest dimension of each of the HCs was determined on coronal T1-weighted images. (Figure 4) on page 9

Focal Abnormalities measuring <1 mm were ignored.

Some cystlike structures were localized in medial part of the hippocampus and were appeared to be the hippocampal sulcus continuation in contiguous sections. We considered them as part of the sulcus, not hippocampal cavities. (Figure 5) on page 9

**MTA Score:**

We used a visual rating scale to evaluate MTA on coronal T1-w images (possible range of scores for each side, 0-4), validated by linear measurements of the medial temporal lobe including the hippocampal formation and surrounding spaces occupied by cerebrospinal fluid. (Figure 6) on page 10
Fig. 0: Identifiable part of the HS is normally a shallow groove just below the fimbria and above the subiculum.

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Fig. 0: In cases that the HS was too shallow to be measured, we considered it equal to 0 mm.

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Fig. 0: HCs are defined as sharply demarcated cystic structures (isointense with CSF) localized at the apex of the hippocampal fold.

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Fig. 0: The greatest dimension of each of the HCs was determined on coronal T1-weighted images.

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Fig. 0: Cystlike structures localized in medial part of the hippocampus were appeared to be the hippocampal sulcus continuation in contiguous sections (Blue arrow). Red arrows show two hippocampal cavities in one side.

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↑ Increase  ↓ Decrease  N: Normal

**Fig. 0:** Visual rating of medial temporal lobe atrophy.

Results

**AGE:** Mean age of patients in case group was 62.8+/-8.7 and in control group was 57.5+/-6.8.

**MMSE Score:** Its mean in case group was 20.1+/-3.4 and in control group was 27.6+/-1.6.

**MTA Score:** Higher grades of MTA were presented in case group (P=0.002).

**MTA & HS Width:** There was significant correlation between MTA and HS width (P=0.003 ,r=0.00323).

**MTA & HC Presence:** There wasn't significant correlation between MTA and HC presence (HCP).

**MTA & HC Size:** There wasn't significant correlation between MTA and HC size, but it had a trend to be significant (P=0.08 ,r=0.00314).

**MTA & HC Number:** There wasn't significant correlation between MTA and HC number (HCN).

**Age & Other variables:**

In *control* group there wasn't any correlation between studied variables and age.

In *case* group, there wasn't any correlation between HC size and age of studied subjects. There was significant relationship between age and MTA, HS width, HC number, as mean age of patients with hippocampal cavity was higher than those without. 65.9 +/-8.07 and 59.8 +/-7.08 respectively, (P<0.05).

**Interobserver agreement:**

For the presence of hippocampal cavity on right and left side, interobserver agreement were 91.7% (P<0.05) and 88.9% (P<0.05), respectively. For other variables see figure 1 on page 13.
**Fig. 0:** Interobserver agreements for the studied variables

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Conclusion

Enlargement of the hippocampal sulcus, is associated with MTA in patients with Alzheimer disease and can be used as a measure to rate MTA severity. By contrast, hippocampal cavities are not found to be significantly associated with MTA or Alzheimer disease and do not seem to have pathologic value.

These MRI measures may also be useful in identifying individuals at particularly high risk for progression, or for guiding the treatment decisions. The use of neuroimaging for the early detection of the effects of AD on the brain has been successful even in the earlier stages of disease when clinical symptoms are not fully expressed and the regional brain damage may be limited.
References


Personal Information

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