Proton Magnetic Resonance Spectroscopy in Mild Cognitive Impairment and Alzheimer's Disease: A Preliminary Study

Wiboon Suriyajakryuththana MD*, Lojana Tuntiyatorn MD*,
Nuttika Teepprasarn MD*, Chakrit Sukying MD**

** Department of Diagnostic and Therapeutic Radiology, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand*
*** Department of Psychiatry, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand*

Background: *Mild cognitive impairment (MCI) is recognized as a transitional clinical state between normal aging and Alzheimer's disease (AD) and has significant higher rate of progression to AD.*
Objective: *To compare the changes of metabolites between AD and MCI in specific locations of the brain by using Magnetic Resonance Spectroscopy (MRS).*
Material and Method: *MMSE-Thai 2002 and neuropsychological test were performed in 17 patients with memory problem, classified into AD and MCI (10, 7 patients respectively). All patients and three age-matched cognitively normal volunteers were examined with conventional MRI and MRS of the brain. Volumes of interest were located at both-sided frontal and parietal deep white matter. NAA/Cr, Cho/Cr, and mI/Cr ratios of the patients were analyzed and statistically evaluated relative to cognitively normal volunteers. Statistical analysis was performed using Cohen's kappa coefficient and Kruskal-Wallis test.*
Results: *There was no statistically significant change in metabolites in all brain regions. For AD relative to cognitively normal volunteers, there were strong tendency toward statistically significant decreased NAA/Cr at the left frontal and left parietal regions ($p = 0.043$ each) and decreased Cho/Cr at the left frontal region ($p = 0.028$).*
Conclusion: *The changes of the metabolite ratios of MCI were much closer to AD. Strong tendency toward statistically significant decreased NAA/Cr in the left cerebral hemisphere, predominantly parietal region and strong tendency toward statistically significant decreased Cho/Cr at the left frontal region were indicative of neurodegeneration and replacement by gliosis. MRS may be useful for predict a chance that cognitively normal people may convert to the AD.*

Keywords: *Magnetic resonance spectroscopy, Mild cognitive impairment, Alzheimer's disease*

J Med Assoc Thai 2014; 97 (4): 407-14
Full text. e-Journal: *http://www.jmatonline.com*

Alzheimer's disease (AD) is progressive neurodegenerative disorder associated with disruption of neuronal function and gradual deterioration in cognition, function, and behavior[1]. It is the most common cause of dementia in the elderly. Current consensus statements have emphasized the need for early recognition and the fact that a diagnosis of AD can be made with high accuracy by using clinical, neuropsychological, and imaging assessments. For early recognition; thus, there is a need to develop sensitive markers that may serve as adjuncts to current clinical and neuropsychological tests to facilitate detection and/or monitoring of early brain changes suggestive of AD. Such markers may also facilitate early-intervention studies to prevent or slow disease progression. Mild cognitive impairment (MCI) is recognized as a transitional clinical state between normal aging and AD[2]. Patients with MCI have reduced memory efficiency compared with normally aging elderly and a significant higher rate of progression to clinical AD compared with cognitively normal elderly persons. The pathological process and areas involved in these distinct conditions associated with cognitive impairment are different, including neurofibrillary tangles affecting the transentorhinal and entorhinal cortex, hippocampus and the limbic cortex, and the posterior cingulate gyrus. Pathologically, AD is characterized by damage to the large cortical neurons subserving cognition, initially in the temporal lobes and later in the remaining neocortex and association areas. Damage is believed to occur owing to mechanisms outside the neuron, as well as inside the neuron, and

Correspondence to:
Suriyajakryuththana W, Division of Neuroradiology, Department of Diagnostic and Therapeutic Radiology, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok 10400, Thailand.
Phone: 0-2201-2465, Fax: 0-2201-1297
E-mail: wiboonsuriya@yahoo.com

is characterized by the appearance of extracellular senile (amyloid) plaques and intracellular neurofibrillary tangles[3]. Clinical criteria used for a provisional diagnosis of AD consist of the National Institute of Neurological and Communicative Disorders and Stroke/Alzheimer's Disease and Related Disorders Association (NINCDS/ADRDA) criteria[4] for possible or probable AD or the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria[5] for dementia of the Alzheimer type. Definitive diagnosis of AD is made by means of pathologic examination of tissue derived from autopsy or brain biopsy. Mini-Mental State Examination (MMSE) is objective test used as screening tool to help assess cognitive dysfunction.

Conventional magnetic resonance imaging (MRI) has long played a supportive role in the diagnosis of memory disorders and is recommended for the routine evaluation of AD. The structural changes may not be detected until late. Serial volumetric imaging and voxel compression subtraction have emphasized a quantitative approach capable of aiding in detection of subtle changes but they are not readily apparent on routine images obtained. Positron emission tomography (PET), single photon emission computed tomography (SPECT), and functional MR imaging have the potential to enable identification of more subtle pathologic changes earlier during the disease course.

Magnetic resonance spectroscopy (MRS), a non-invasive technique, can be readily incorporated into the conventional MR protocols that continue to provide the mainstay for anatomical imaging to provide localizing biochemical information in vivo including N-acetyl aspartate (NAA), creatine plus phosphocreatine (Cr), choline-containing compounds (Cho) and myoinositol (mI). NAA is found in normal neuron and is reduced in neuronal or axonal damage. Cho reflects membrane synthesis and degradation. Cr is relatively constant and reflects energy metabolism, using as reference to normalize signal intensity of other metabolites, such Cho/Cr or NAA/Cr. mI reflects neural damage with gliosis. MRS in previous studies reported decreased NAA and/or increased mI levels in the posterior cingulate cortex and the temporal lobes in AD. In MCI, a higher mI/Cr and no statistically significant decrease of NAA/Cr ratio have been reported[6].

The aim of the present was to compare the changes of metabolites between AD and MCI in specific locations of the brain by MRS.

Material and Method

The cross sectional retrospective study was performed at a university hospital during a consecutive 18-month period, from April 2006 to October 2007 with Institutional Review Board Approval. Written informed consent was obtained from each patient prior to enrollment.

Subjects

Seventeen patients with memory problems were referred from psychiatric and memory clinic services at Ramathibodi Hospital to perform structural MRI and MRS of the brain. The MMSE-Thai 2002 and neuropsychological tests were administered in all study participants. The battery included the following: Wechsler Adult Intelligence Scale, Wechsler Memory Scale, Boston Naming test (Kaplan, Good glass, Weintraub 1983), Trial making A and B (Lezak, 1995) and Verbal Fluency Test were performed and then classified into AD (10 of total 17 patients) and MCI (7 of total 17 patients) categories. For AD, NINCDS-ADRDA criteria would be used for diagnosis of probable AD. MCI was defined by the following characteristics: subjective memory complaint, normal activities of daily living, normal general cognitive function, abnormal memory for age and not demented, normal or near-normal performance on global cognitive tests (MMSE score >22). Three cognitively normal volunteers were recruited. All of them would not meet the criteria for probable AD and the MMSE scores were >22. The patients with structural abnormalities that could produce dementia including cortical infarction, tumor, subdural hematoma, or having concurrent illnesses or treatments interfering with cognitive function other than AD were excluded.

Imaging techniques

Structural MRI and MRS of the brain were performed using three Tesla MRI machine (Philips, Archive platform software version R1.2) with a sense head coil. MRI of the brain consisting of sagittal and axial T1-weighted, axial T2 FSE (Fast Spin Echoes), and fluid-attenuation inversion recovery sequence (FLAIR) images, with coronal GRE image and 3D T1W SPGR whole brain in parallel plane to the hippocampus were performed in all patients. The imaging criteria of AD including thinning of the entorhinal cortex, atrophic change of the hippocampus, amygdala, and temporal neocortex were used (Fig. 1).

MR axial image was obtained for localized ^{1}H MRS voxels in each patient. The MRS was performed with two acquisitions by using TEs of 33 and 144 ms (millisecond). A TE of 144 ms was chosen to detect metabolite peaks with relatively longer transverse relaxation times (NAA, Cr, and Cho) and underlying broad resonances. A TE of 33 ms was chosen to quantify metabolite with short transverse relaxation times (mI). Four 8.0 cm^3 (2.0 cm x 2.0 cm x 2.0 cm) of volume of interest (VOIs) were located as follows: bilateral frontal and parietal deep white matter areas (Fig. 2). The H-2DCSI data were analyzed with custom spectroscopic imaging software on PHILIPS View Forum workstation. The following metabolite signals were quantified: NAA peak at 2.0 ppm; Cr peak at 3.0 ppm; Cho peak at 3.2 ppm; mI peak at 3.5 ppm. Relative metabolite levels were expressed as the ratio of peak height area of a selected signal divided by the peak height area of Cr resonance. The following ratios were assessed at the specific locations, NAA/Cr, Cho/Cr in long TE spectra, and mI/Cr in the short TE spectra. The investigators separately evaluating structural MRI of the brain and metabolite ratios were blinded to the diagnostic status.

Statistical analysis

Cohen's kappa coefficient was used to evaluate agreement between neuropsychological diagnosis and structural MR findings between AD, MCI, and Cognitively normal groups. To assess a statistically significant difference of the metabolite ratios in the specific locations between AD and MCI patients compared with cognitively normal volunteers,

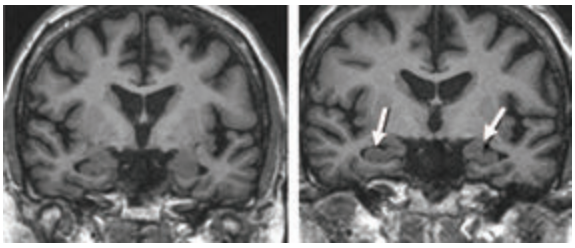


Fig. 1 MRI of AD, Coronal 3D SPGR showed thinning of the entorhinal cortex and atrophic change of bilateral hippocampi (arrows).

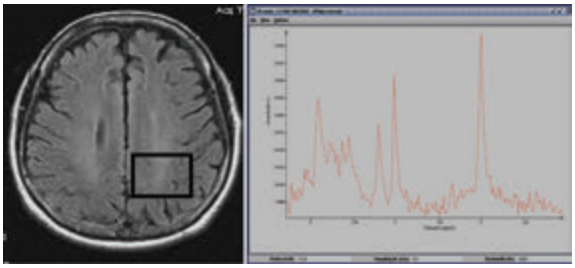
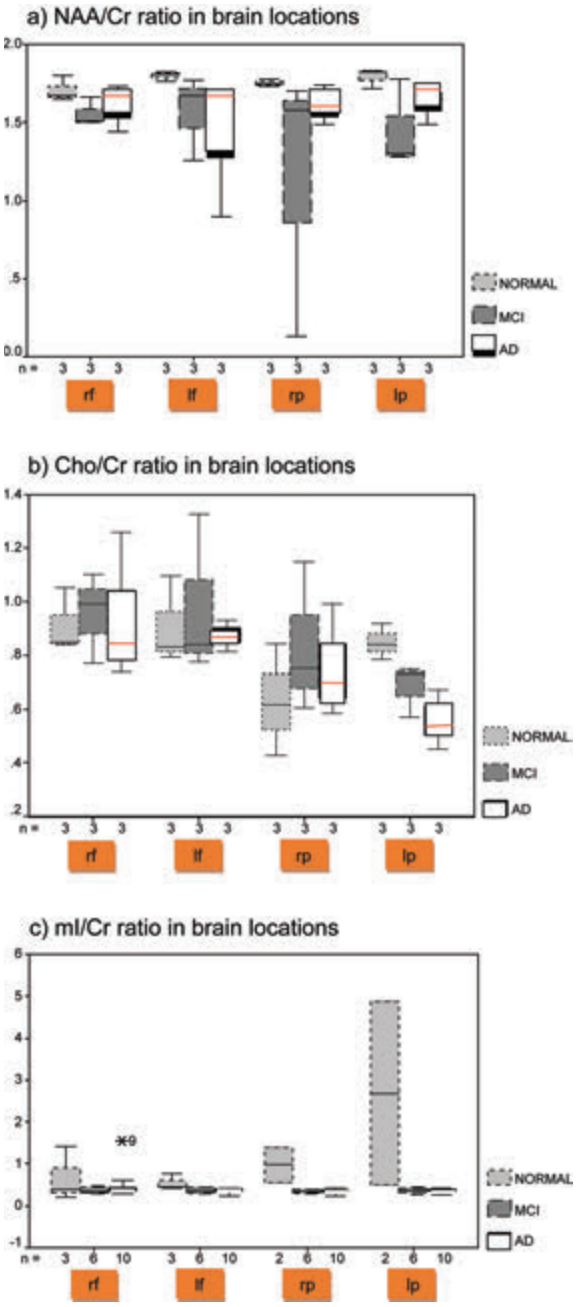


Fig. 2 Single voxel MRS (TE = 33 ms) at the left parietal white matter.



rf = right frontal region; lf = left frontal region; rp = right parietal region; lp = left parietal region

Fig. 3 (a, b, c) Box plots of metabolites ratios in different brain locations.

Table 1. Demographic data and MMSE-scores of the subjects

Characteristics	Cognitively normal (n = 3)	MCI (n = 7)	AD (n = 10)
Age (years), mean ± SD	64.0±4.58	68.4±7.2	73.7±8.11
Range	59-68	55-76	56-82
Sex			
Male	1 (33.33%)	3 (42.85%)	4 (40%)
Female	2 (66.66%)	4 (57.14%)	6 (60%)
MMSE, mean ± SD	30.0±0.0	28.7±1.8	21.3±6.0

MMSE = mini-mental state examination; MCI = mild cognitive impairment; AD = Alzheimer's disease

the Kruskal-Wallis test was used with Bonferroni correction alpha. A *p*-value less than 0.017 was considered statistically significant.

Results

The demographic data and MMSE scores of AD, MCI patients and cognitively normal volunteers were demonstrated in Table 1. There was no statistically significant difference in age between the AD and MCI and between the MCI and cognitively normal volunteers with $p = 0.064$ and 0.210 respectively, however there was significant difference in age between AD and cognitively normal volunteers ($p = 0.043$). There was a significant difference of the MMSE scores between AD and MCI and between AD and cognitively normal volunteers ($p = 0.009$ and 0.02 respectively). There was no significant difference in MMSE scores between MCI and cognitively normal volunteers ($p = 0.205$). The underlying diseases were detected in three patients (20%) including hypertension in one patient and hyperlipidemia in three patients. The mean of education was 13.9 years (SD = 4.58)

The neuropsychological test was performed in all patients. The agreement between neuropsychological diagnosis and MRI findings in AD and cognitively normal volunteers revealed an almost perfect degree of agreement 92.31%, kappa = 0.806 (Table 2). As compared with the neuropsychological diagnosis and structural MRI in MCI and cognitively normal volunteers, which resulted in normal or abnormal MRI findings including white matter ischemia of small vessel disease, it showed a fair degree of agreement 60%; kappa = 0.310 (Table 3).

Lists of the median and range of metabolite ratios were obtained at the specific locations of the brain from each clinical group, shown in Table 4 showed statistical analysis of metabolite ratios between groups.

Regarding NAA/Cr ratios, no statistically significant decreased NAA/Cr were measured for patients with AD and cognitively normal volunteers, MCI and cognitively normal volunteers and AD and MCI in all brain regions but at the left frontal and left parietal regions, there were strong tendency toward statistically significant decreased NAA/Cr in AD and cognitively normal volunteers ($p = 0.043$ each). AD and cognitively normal volunteers, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.499, 0.043, 0.069, and 0.043 respectively. MCI and cognitively normal volunteers, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.732, 0.087, 0.305, and 0.087 respectively. AD and MCI patients, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.495, 0.118, 0.922, and 0.845 respectively.

According mI/Cr ratios, increased mI/Cr was discovered in all four regions in AD but all are no statistically significant. AD and cognitively normal

Table 2. Agreement between neuropsychological diagnosis and MRI findings in AD and cognitively normal subjects

MRI findings	Neuropsychological diagnosis		Kappa
	AD (n = 10)	Normal (n = 3)	
AD	9	0	0.806
Normal	1	3	

MRI = magnetic resonance imaging; AD = Alzheimer's disease

Table 3. Agreement between neuropsychological diagnosis and MRI findings in MCI and cognitively normal subjects

MRI findings	Neuropsychological diagnosis		Kappa
	MCI (n = 7)	Normal (n = 3)	
Abnormal	3	0	0.310
Normal	4	3	

MRI = magnetic resonance imaging; MCI = mild cognitive impairment

Table 4. Metabolite ratios (median and range) in cognitively normal, MCI and AD subjects

	Normal (n = 3)	MCI (n = 7)	AD (n = 10)
NAA/Cr			
Right frontal	1.66 (1.65-1.80)	1.66 (1.50-2.12)	1.64 (1.35-1.81)
Left frontal	1.81 (1.76-1.81)	1.74 (1.26-1.77)	1.54 (0.90-2.00)*
Right parietal	1.74 (1.73-1.78)	1.60 (0.12-1.80)	1.67 (1.47-1.82)
Left parietal	1.82 (1.71-1.82)	1.55 (1.28-1.80)	1.66 (0.99-1.81)
Cho/Cr			
Right frontal	1.06 (0.98-1.09)	0.99 (0.69-1.11)	0.88 (0.73-1.25)
Left frontal	1.01 (0.97-1.16)	0.83 (0.77-1.32)	0.87 (0.50-1.09)
Right parietal	0.76 (0.58-0.81)	0.75 (0.42-1.14)	0.68 (0.45-0.91)
Left parietal	0.79 (0.58-0.81)	0.73 (0.57-1.28)	0.69 (0.45-0.91)
mI/Cr			
Right frontal	0.34 (0.29-0.44)	0.29 (0.20-0.46)	0.41 (0.31-1.51)
Left frontal	0.41 (0.38-0.41)	0.38 (0.22-0.44)	0.41 (0.31-0.78)
Right parietal	0.32 (0.32-0.34)	0.31 (0.24-1.39)	0.38 (0.27-0.54)
Left parietal	0.33 (0.30-0.38)	0.37 (0.26-4.87)	0.40 (0.26-0.48)

NAA = N-acetyl aspartate; Cr = creatine plus phosphocreatine; Cho = choline-containing compounds; mI = myoinositol

volunteers, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.271, 0.866, 0.116, and 0.078 respectively. MCI and cognitively normal volunteers, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.606, 0.606, 0.796, and 0.606 respectively. AD and MCI patients, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.083, 0.278, 0.262, and 0.637 respectively.

The Cho/Cr ratios were lower in all MCI and AD relative to the cognitively normal volunteers but no statistically significant. Strong tendency toward statistically significant differences in decreased Cho/Cr were detected in patients with AD relative to cognitively normal volunteers at the left frontal region ($p = 0.028$). AD and cognitively normal volunteers, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.091, 0.028, 0.612, and 0.398 respectively. MCI and cognitively normal volunteers, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.909, 0.569, 0.732, and 0.909 respectively. AD and MCI patients, *p*-values at the right frontal, left frontal, and right and left parietal regions were 0.696, 1.000, 0.922, and 0.435 respectively.

Discussion

MCI is recognized as a transitional clinical state between normal aging and AD[2]. The persons with MCI have reduced memory efficiency and pathological changes corresponding to an early AD phase, and a significant higher rate of progression to clinical AD (12-15%) when compared with cognitively normal elderly persons (1-2%)[6]. Therefore, there is a need to develop sensitive markers that may serve as adjuncts to current clinical and neuropsychological tests to facilitate detection and/or monitoring of early brain changes suggestive of AD. Such markers may also facilitate early-intervention studies to prevent or slow disease progression. Proton MR Spectroscopy (H MRS) is non-invasive assessment of metabolite levels in brain tissue. It allows in vivo assessment of NAA, glutamine and glutamate, aminobutyric acid, myo-inositol, glycine, mobile choline, creatine and phosphocreatine, lipids, and lactate. NAA is present primarily in neurons within the central nervous system but not in glial cells or other non-neuronal tissue. It is generally thought to represent a marker of neuronal function. The NAA level is decreased in cases of neuronal loss or damage yet may return to normal levels during recovery. Elevated mI levels may mark gliosis, membrane dysfunction, and/or cytoskeletal abnormalities. Elevated choline levels may reflect cellular proliferation, as in neoplasia, or myelin breakdown[7].

The present study demonstrated no statistically significant regional metabolite changes in all metabolites of all brain regions but at the left frontal and left parietal regions. There were strong tendency towards statistical significant decreased NAA/Cr with AD relatively to cognitively normal volunteers, indicative of gradual process of brain degeneration. Since NAA is a neuronal marker, the decreased NAA/Cr ratios in the left frontal and left

parietal areas in patients with AD and tendency toward decreased NAA/Cr in the rest of the brain suggests that the pathological process should be occurring, a disruption of neuronal function or neuronal cell death. In vitro studies on postmortem brains, decreases in NAA levels have been demonstrated in patients with AD, as compared with levels in controls. Moreover, there has been a positive correlation between the magnitude of NAA decreases and the severity of neuropathological findings (counts of amyloid plaques and neurofibrillary tangle)[8,9]. In addition, in vivo studies have demonstrated decreases in NAA in patients with AD in both the temporal and parietal lobes. The neuronal dysfunction is likely to occur before neuronal loss while chemical changes measured by MRS may occur before tissue volume loss in AD[10,11]. Therefore, the authors hypothesize that highly toward tendency to reduction in NAA/Cr, particularly in the left cerebral hemisphere in individuals with AD may be valuable in predicting future development of AD and monitoring early disease progression for preventive therapies.

The present study showed no statistically signicant increased mI/Cr ratio in all patients and all brain regions. Increased mI/Cr ratio reflected a loss of neurons and replacement by glia or gliosis, indicative of distribution of the neurodegeneration differently in asymmetry with predominant reduction in the left posterior cortices. Shonk et al[12], determined that changes in the mI/NAA ratio would help distinguish patients with AD from control subjects with a sensitivity of 83% and a specificity of 98% and that changes in the mI/Cr ratio would help distinguish patients with AD from elderly person with other forms of dementia, with a sensitivity of 82% and a specificity of 64%. For AD, a higher mI/Cr with a non-significant decrease of NAA/Cr has been reported[13]. Parnetti L et al[14] had shown NAA and mI metabolite levels in the left parietal area with MCI to be between those of cognitively normal person and AD. The changes of the metabolites in MCI were much closer to AD than to the cognitively normal volunteers in the present study. The Cho/Cr ratios were not statistically significant decreased in all patients and all brain regions. However, in AD, there was strong tendency toward statistically significant decreased Cho/Cr ratio at the left frontal region.

The present study had a limitation by a small number of patients available for study and the absence of patients concentrated within a specific age group. Studying more patients restricted to a narrower age range would yield a more accurate evaluation of the brain metabolite ratios in normal aging, AD, and MCI groups. In addition, exploration of other cortical areas, such as the hippocampus and temporal lobe could help increase the probability of more accurate postulations.

Conclusion

In the present study, MRS showed that the changes of the metabolite ratios of the patients with MCI were much closer to the patients with AD than to the cognitively normal volunteers. Strong tendency toward statistically significant decreased NAA/Cr at the left cerebral hemisphere and Cho/Cr at the left frontal region were indicative of neurodegeneration and replacement by gliosis. MRS may be useful for predict a chance that cognitively normal people may convert to the AD.

What is already known on this topic?

The previous topics show that in the patient with AD relative to cognitively normal volunteers, Cho/Cr ratios were strong tendency toward statistically significant decreased in the left frontal region and tendency toward statistically significant decreased NAA/Cr at the left cerebral hemisphere. In the MCI, there was no significant difference in metabolite ratios in all of the brain regions. Shonk et al[14], determined that changes in the mI/NAA ratio would help distinguish patients with AD from control subjects and that changes in the mI/Cr ratio would help distinguish patients with AD from elderly person with other forms of dementia.

What the study adds?

There is agreement between neuropsychological diagnosis and MRI findings in MCI and cognitively normal subjects. The metabolite ratios detected by MRS of the patients with MCI were much closer to the patients with AD than to the cognitively normal volunteers. There was no statistically significant change in mI/Cr ratio (different from the previous articles). Strong tendency toward decreased NAA/Cr in the left cerebral hemisphere and Cho/Cr at the left frontal region in AD relative to cognitively normal volunteers is shown by the study (different from the other articles that seen change only in frontal not parietal region). The findings support that MRS may be useful for predict a chance that cognitively normal people may convert to the AD.

Acknowledgement

We are deeply grateful to Amnuay Thithapandha for proofreading this article.

Potential conflicts of interest

None.

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การตรวจหาระดับเมตะบอไลท์ด้วยเครื่องตรวจคลื่นสนามแม่เหล็กไฟฟ้าในกลุ่มผู้ป่วยภาวะสติปัญญาบกพร่องและโรคอัลไซเมอร์

วิบูลย์ สุริยจักรยุทธนา, โลจนา ตันติยาพร, ณัฐฐิกา ทีปประสาร, จักรกฤษณ์ สุขยิ่ง

ภูมิหลัง: ภาวะสติปัญญาบกพร่องเป็นระยะแปรเปลี่ยนทางคลินิกระหว่างผู้สูงอายุปกติกับโรคอัลไซเมอร์ และพบว่าในกลุ่มนี้มีอัตราการดำเนินโรคเปลี่ยนไปเป็นโรคอัลไซเมอร์สูงอย่างมีนัยสำคัญ

วัตถุประสงค์: เพื่อศึกษาเปรียบเทียบระดับเมตะบอไลท์ระหว่างผู้ป่วยโรคอัลไซเมอร์ และผู้ป่วยภาวะสติปัญญาบกพร่องที่ตำแหน่งต่าง ๆ ของสมองด้วยเครื่องตรวจคลื่นสนามแม่เหล็กไฟฟ้า

วัสดุและวิธีการ: ได้ศึกษาผู้ป่วยที่มีปัญหาเรื่องความจำบกพร่อง จำนวน 17 ราย ทุกรายได้รับการตรวจทางจิตเวชชนิด MMSE Thai 2000 และ neuropsychological test เพื่อแบ่งกลุ่มออกเป็นผู้ป่วยโรคอัลไซเมอร์ (10 ราย) และผู้ป่วยภาวะสติปัญญาบกพร่อง (7 ราย) ผู้ป่วยทุกรายและอาสาสมัครสูงอายุที่มีสติปัญญาปกติ (3 ราย) ได้รับการตรวจสมองแบบพื้นฐานและหาระดับเมตะบอไลท์ด้วยเครื่องตรวจคลื่นสนามแม่เหล็กไฟฟ้า การวัดระดับเมตะบอไลท์ได้กระทำที่บริเวณสีขาวของสมองกลีบหน้าและหลังทั้งสองข้าง เพื่อหาอัตราส่วน NAA/Cr, Cho/Cr, mI/Cr แล้วนำมาวิเคราะห์และประเมินผลทางสถิติ ในผู้ป่วยทั้ง 2 กลุ่มโดยเปรียบเทียบกับกลุ่มผู้สูงอายุที่มีสติปัญญาปกติ การวิเคราะห์ทางสถิติใช้ Pearson's correlation coefficients และ Kruskal-Wallis test

ผลการศึกษา: ผู้ป่วยทุกกลุ่มไม่พบการเปลี่ยนแปลงอย่างมีนัยสำคัญของระดับเมตะบอไลท์ทุกตัวในทุกบริเวณของเนื้อสมองที่ตรวจ กลุ่มโรคอัลไซเมอร์ เมื่อเปรียบเทียบกับผู้สูงอายุปกติพบมีแนวโน้มสูงในการลดลงของ NAA/Cr ที่สมองกลีบหน้าและหลังด้านซ้าย ($p = 0.043$) มีแนวโน้มสูงในการลดลงของ Cho/Cr ที่สมองกลีบหน้าด้านซ้าย ($p = 0.028$)

สรุป: การเปลี่ยนแปลงของเมตะบอไลท์ในผู้ป่วยภาวะสติปัญญาบกพร่องมีความใกล้เคียงกับผู้ป่วยโรคอัลไซเมอร์ มีแนวโน้มสูงในการลดลงของ NAA/Cr ในสมองด้านซ้ายโดยเฉพาะกลีบหลัง และมีแนวโน้มสูงในการลดลงของ Cho/Cr ในสมองด้านซ้ายกลีบหน้า แสดงถึงการเสื่อมสภาพของเซลล์ประสาทและถูกแทนที่ด้วยเกลียลเซลล์ ดังนั้นการตรวจหาระดับเมตะบอไลท์ด้วยเครื่องตรวจคลื่นสนามแม่เหล็กไฟฟ้าอาจใช้ประโยชน์ในการทำนายโอกาสของการเปลี่ยนแปลงจากผู้สูงอายุปกติไปเป็นโรคอัลไซเมอร์ และอาจทำให้สามารถค้นหาแนวทางรักษาป้องกันการเปลี่ยนแปลงนี้ก่อนที่จะป่วยเป็นโรคอัลไซเมอร์