Diagnosis of Alzheimer's disease: possible role of functional imaging technique

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Background
The combination of clinical diagnosis of Alzheimer's disease (AD) with diffusion-weighted imaging (DWI) may improve the diagnostic accuracy of AD.
Objectives
This study primarily aims to determine the relationship between the measures of DWI and clinically diagnosed AD and its severity.
Participants and methods
The sample evaluated comprised three groups: 14 patients with AD, nine patients with minimal cognitive impairment of amnesic type (a-MCI), and 11 healthy controls. They were recruited from 2008 to 2009. The diagnosis of AD was made according to the *Diagnostic and Statistical Manual of Mental Disorders* fourth edition text revision and that of a-MCI was made according to Mayo Clinic's criteria. Dysfunctional severities were assessed using the Mini-Mental State Examination and Alzheimer's disease Assessment Scale. The magnetic resonance DWI technique was used to estimate the diffusivity of water through different cerebral regions as the assumed imaging marker.
Results
DWI measures of AD in different cerebral regions were found to be higher but not significantly different from that of the control group. Apparent diffusion coefficient means of the a-MCI group were closer to and not significantly different from that of the AD group. In addition, there was no significant relation between the diffusivity measures in such regions and the total scores of assessments for the dysfunctional severities.
Conclusion
The only trend toward increased diffusivity was shown with conventional DWI but no statistically conclusive results were obtained either for gray or for white matter among patients with AD.

Keywords:
Alzheimer's disease, diffusion-weighted imaging, minimal cognitive impairment

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Introduction

The diagnosis of definite Alzheimer's disease (AD) can only be made by postmortem neuropathological confirmation of individuals who had been studied in life and met the criteria for dementia [1]. Clinical AD is still diagnosed by excluding other causes of dementia. A combination of clinical examination with neuroimaging may improve diagnostic accuracy using neuropathology as the standard of comparison. Temporal lope atrophy progresses 10 times faster in AD than in normal aging. Indices of atrophy are statistically related to cognitive performance [2]. Such variables were correlated with the severity of neuropathological changes. Hence, Hentschel and Forstel [3] concluded that hippocampal imaging has proven to be a clinically useful and feasible method for identifying patients with early AD, which is demonstrated by diffusion-weighted imaging (DWI) technique. This technique involves noninvasive functional imaging, that is, processing through high-speed magnetic resonance imaging (MRI), with images acquired in a few seconds or less, thus providing motion-free images. The advent of a high-speed gradient has made DWI an everyday routine scan and has led to its establishment as an Food and Drug Administration-approved clinical screening examination for nearly all neurology examinations today [4].

With the modest but important breakthrough in the treatment of AD, the diagnostic focus has increasingly shifted to the accurate detection of the earliest phase of the illness. The challenge of distinguishing preclinical AD from changes in normal aging or established AD has been recognized during several attempts at clinical classification. Of these attempts, Mayo Clinic's MCI has received significant attention. However, as not all individuals diagnosed as having MCI will develop AD; hence, there is a need to predict progression reliably [5]. The diagnosis and prognosis of a-MCI has been studied most thoroughly. It is frequently a prodrome to AD, whereas the prognostic significance of other subtypes of MCI is not well understood. Brain imaging studies are not

routinely used for the diagnosis of MCI, although imaging techniques are rapidly developing and may prove to be clinically useful in future [6].

The aim of this study was to determine the relationship between the measures of diffusivity of water molecules and clinically diagnosed AD and its prototype a-MCI. Changes in DWI in the right and left cerebral regions of AD were assessed. In addition, the relationship between the severity of cognitive and behavioral manifestations of AD as measured by neuropsychological assessments and the DWI was tested. Determination of such a relationship may help in differentiating between normal aging, MCI, and AD and hence, improving the diagnostic accuracy.

Participants and methods

Participants

The sample evaluated comprised three groups: 14 patients (eight men and six women) with AD, nine patients (five men and four women) with a-MCI, and 11 controls (six men and five women). The original sample comprised 16 patients with AD, with two dropouts, 11 patients with a-MCI, also with two dropouts. These were because of nonconcordant diagnoses. Data were collected from 2008 to 2009. Patients were recruited from outpatients of the geriatric unit of the Psychiatric Department of the Mansoura University Hospitals. The diagnosis of AD was made according to the *Diagnostic and Statistical Manual of Mental Disorders* fourth edition text revision [7] and that of a-MCI was made according to Mayo Clinic's criteria [8,9]. All patients were diagnosed through information obtained from an extensive clinical history and physical examination by two independent senior psychiatrists. Participants were aged between 50 and 80 years without a history of brain trauma, epilepsy, brain tumor, stroke, psychiatric disorders, and other systemic disease that could affect brain function. Patients with atherosclerotic white matter disease as shown by MRI were also excluded despite the absence of clinical correlates to nullify the confounding effect of mixed pathology. Eleven healthy controls were matched with age and sex of cases and not receiving medical treatment that could affect the brain metabolism. They were attendants of psychiatric patients of the Mansoura University Hospital. Informed consent was obtained from all participants.

Assessments

(I) Assessment Scales:

1. Mini-Mental State Examination (MMSE) [10]: this is a screening instrument that provides a brief assessment of an individual's orientation to time and place, recall ability in terms of short memory, and arithmetic ability. The MMSE has been used extensively in clinical settings. It is emphasized that this instrument should not be used to diagnose dementia but rather as a bedside instrument to grade the cognitive function of a patient.
2. Alzheimer's disease Assessment Scale (ADAS) [11]: this is a rater scale that measures the severity of dysfunction in cognitive and noncognitive behaviors characteristic of AD. Cognitive and memory task items compromise 60% of the total possible points. The ADAS appears to be sensitive to increasing dysfunction as the illness progress.

(II) Brain imaging: DWI; a new noninvasive functional MRI) technique was implemented at the Department of Diagnostic Radiology of the Mansoura University Hospitals. The random movement of water molecules affects the magnetic resonance gradient-echo intensity. Hence, DWI estimates the diffusivity of water molecules through the interstitial tissue. Lesions with reduced water mobility appear very hyperintense and *vice versa*. The resultant signal intensity of a voxel of tissue containing moving protons of the hydrogen atoms 1H in the water molecules for a given pixel is measured using the apparent diffusion coefficient (ADC). The average ADC was electronically generated incorporating all planes, axial, coronal, and sagittal, for each pixel to remove artifacts because of the direction of acquisition [4]. In this study, conventional ADC values, with applied strength (b value) = 1000, were calculated from the DWI in the regions of interest located in the left and right corpus striatum, basal ganglia, thalamus, amygdala, hippocampus, and white and gray matter of the frontal and parietal areas. Areas with less water diffusion indicated more intense regions. Such regions were selected as mostly implicated in AD.

Analysis

Analysis was performed using the Statistical Package for Social Sciences (SPSS, version 12.0 for Windows, SPSS Inc., Chicago, USA). The reliability between the two psychiatrists was examined using the Kappa method. The ADC variables for the groups were analyzed using the independent-samples t test for equality of means in which Equal variances were assumed. Paired samples statistics were used to assess the differences in diffusion in both cerebral hemispheres among the Alzheimer's a-MCI and control groups. Regression analysis was carried out to assess the effect of ADC of the AD group in different cerebral regions as predictors for the severity of the dysfunction as measured by MMSE and ADAS. These scales have total scores that measure interpenetrated global functions of different brain areas. As the statistical summation of the diffusivity measures of different brain areas may not be practically informative for comparison with these total scores, the possible separate contributions of ADC in specific areas to different functions were examined using the regression analysis method. Different predictor regions were tested because several brain functions were measured by the assessment scales. Levels of significance are reached when P value is less than or equal to 0.05 at a confidence interval of 95%.

Results

Considering the diffusivity measures, the AD group's means differed nonsignificantly from that of the control

group. Such insignificant findings were obtained despite the higher mean ADC scores of AD groups in all the cerebral regions included. (Table 1). The ADC means of a-MCI patients were closer to that of patients with AD, with no significant difference (Table 2). Table 3 shows a comparison of ADC measures of different regions between the two cerebral hemispheres in the AD group. In addition, no significant difference in the ADC on both sides was found regardless of the region. However, the diffusivity measures were significantly correlated among some of these regions between the right and the left sides, namely the corpus striatum ($P<0.001$), amygdala ($P<0.005$), hippocampus ($P<0.005$), frontal gray matter ($P<0.05$), and parietal white matter ($P<0.005$). Significant correlations were found between certain regions on both sides in the a-MCI group (Table 4) including the corpus striatum ($P=0.001$), thalamus ($P<0.01$), hippocampus ($P<0.005$), and frontal gray matter ($P<0.01$). Low significant correlations were found only in the corpus striatum ($P=0.01$) and thalamus ($P<0.05$) in the control group (Table 5).

The ADCs of the AD group in each of the selected cerebral regions were implicated by regression analysis as pathological predictors for the severity of the dysfunctions. Such dysfunctions measured using the assessment scales were assumed to be dependent variables. There was no significant relation between the diffusivity measures in such regions and the total scores of cognitive dysfunction measured by MMSE (Table 6) and the severity scores of cognitive and noncognitive dysfunction measured by ADAS (Table 7).

Discussion

AD is a neurodegenerative disorder that involves mostly the gray matter, although white matter components such as axons and oligodendrocytes have been strongly implicated by histopathological studies [12]. General brain atrophy occurs, but temporal lobe structures and periventricular white matter including the corpus callosum have been implicated in MRI studies [13]. Most commonly, clinicians refer to clinical criteria to distinguish between normal aging, MCI, and AD. Presumably, there are features of neuroimaging measures that may help distinguish between these conditions for which further studies are needed [14,15]. It may ultimately be the case that a combination of clinical features, neuropsychological testing, biomarkers, and neuroimaging may be necessary to improve the diagnostic accuracy [9].

In this study, higher but nonsignificant mean scores of diffusivity measures were found in the AD group compared with the control group in all of the selected regions. This might account for the low tissue densities

Table 1 Comparison of the ADC between AD and control groups in different cerebral regions

Regions	Groups	*N*	Mean	Standard deviation	*t*	Significance[a] (2-tailed)
Left corpus striatum	Alzheimer	14	108.814	18.3483	2.273	0.033
	Control	11	94.755	10.2186		
Right corpus striatum	Alzheimer	14	107.729	15.0114	1.881	0.073
	Control	11	97.482	11.2861		
Left basal ganglia	Alzheimer	14	103.936	28.0499	1.679	0.107
	Control	11	88.736	11.7747		
Right basal ganglia	Alzheimer	14	101.750	17.6190	2.779	0.011
	Control	11	85.300	9.6332		
Left thalamus	Alzheimer	14	105.393	25.5568	1.445	0.162
	Control	11	93.936	6.4615		
Right thalamus	Alzheimer	14	103.707	13.5036	2.416	0.024
	Control	11	92.855	6.9888		
Left amygdala	Alzheimer	14	106.543	20.6455	0.846	0.406
	Control	11	99.109	23.2309		
Right amygdala	Alzheimer	14	109.621	16.4808	1.999	0.058
	Control	11	96.373	16.4050		
Left hippocampus	Alzheimer	14	107.936	26.1145	1.101	0.282
	Control	11	97.882	17.1712		
Right hippocampus	Alzheimer	14	113.471	34.6008	1.423	0.168
	Control	11	97.027	18.3253		
Left frontal white matter	Alzheimer	14	107.500	23.9186	1.674	0.108
	Control	11	94.473	10.6951		
Right frontal white matter	Alzheimer	14	104.921	14.5526	0.466	0.646
	Control	11	102.336	12.6945		
Left frontal gray matter	Alzheimer	14	116.493	33.2244	1.580	0.128
	Control	11	100.245	7.9998		
Right frontal gray matter	Alzheimer	14	112.921	31.0600	0.696	0.493
	Control	11	104.882	25.1784		
Left parietal white matter	Alzheimer	14	110.464	22.9256	1.625	0.118
	Control	11	98.873	6.1311		
Right parietal white matter	Alzheimer	14	126.564	48.5468	2.040	0.053
	Control	11	96.464	4.7096		
Left parietal gray matter	Alzheimer	14	143.036	63.2490	0.465	0.646
	Control	11	133.064	36.2246		
Right parietal gray matter	Alzheimer	14	148.407	67.3828	1.304	0.205
	Control	11	119.373	33.4168		

ADC, Apparent diffusion coefficient.
[a]Degrees of freedom = 23.

Table 2 Comparison of the ADC between AD and MCI groups in different cerebral regions

Regions	Groups	*N*	Mean	Standard deviation	*t*	Significance[a] (2-tailed)
Left corpus striatum	Alzheimer	14	108.814	18.3483	0.310	0.760
	MCI	9	106.511	15.7345		
Right corpus striatum	Alzheimer	14	107.729	15.0114	−0.574	0.572
	MCI	9	111.822	19.0953		
Left basal ganglia	Alzheimer	14	103.936	28.0499	0.670	0.510
	MCI	9	97.189	13.3407		
Right basal ganglia	Alzheimer	14	101.750	17.6190	0.412	0.684
	MCI	9	98.133	24.5348		
Left thalamus	Alzheimer	14	105.393	25.5568	0.097	0.924
	MCI	9	104.478	14.8212		
Right thalamus	Alzheimer	14	103.707	13.5036	0.092	0.928
	MCI	9	103.178	13.4440		
Left amygdale	Alzheimer	14	106.543	20.6455	1.252	0.224
	MCI	9	97.067	11.4703		
Right amygdala	Alzheimer	14	109.621	16.4808	1.361	0.188
	MCI	9	95.000	34.8901		
Left hippocampus	Alzheimer	14	107.936	26.1145	1.067	0.298
	MCI	9	97.522	16.1679		
Right hippocampus	Alzheimer	14	113.471	34.6008	0.066	0.948
	MCI	9	112.278	52.7150		
Left frontal white matter	Alzheimer	14	107.500	23.9186	1.314	0.203
	MCI	9	95.744	14.9052		
Right frontal white matter	Alzheimer	14	104.921	14.5526	0.726	0.476
	MCI	9	100.778	11.1294		
Left frontal gray matter	Alzheimer	14	116.493	33.2244	0.980	0.338
	MCI	9	104.700	16.9758		
Right frontal gray matter	Alzheimer	14	112.921	31.0600	0.712	0.484
	MCI	9	105.033	14.0602		
Left parietal white matter	Alzheimer	14	110.464	22.9256	−1.060	0.301
	MCI	9	122.567	31.9545		
Right parietal white matter	Alzheimer	14	126.564	48.5468	−1.075	0.294
	MCI	9	221.478	328.9487		
Left parietal gray matter	Alzheimer	14	143.036	63.2490	0.253	0.803
	MCI	9	136.600	53.2760		
Right parietal gray matter	Alzheimer	14	148.407	67.3828	0.310	0.760
	MCI	9	140.344	48.6679		

AD, Alzheimer's disease; ADC, apparent diffusion coefficient; MCI, minimal cognitive impairment.
[a]Degrees of freedom = 23.

Table 3 Difference[a] of the ADC between the two cerebral hemispheres in the AD group

Regions	Mean	Standard deviation	Correlation	Significance	*t*	Significance (2-tailed)
Corpus striatum						
Left	108.814	18.3483	0.941	0.000	0.616	0.549
Right	107.729	15.0114				
Basal ganglia						
Left	103.936	28.0499	0.306	0.288	0.290	0.776
Right	101.750	17.6190				
Thalamus						
Left	105.393	25.5568	0.234	0.421	0.243	0.812
Right	103.707	13.5036				
Amygdala						
Left	106.543	20.6455	0.699	0.005	−0.773	0.454
Right	109.621	16.4808				
Hippocampus						
Left	107.936	26.1145	0.699	0.005	−0.834	0.419
Right	113.471	34.6008				
Frontal white matter						
Left	107.500	23.9186	0.326	0.255	0.409	0.689
Right	104.921	14.5526				
Frontal gray matter						
Left	116.493	33.2244	0.545	0.044	0.435	0.671
Right	112.921	31.0600				
Parietal white matter						
Left	110.464	22.9256	0.747	0.002	−1.725	0.108
Right	126.564	48.5468				
Parietal gray matter						
Left	143.036	63.2490	0.192	0.510	−0.242	0.813
Right	148.407	67.3828				

ADC, apparent diffusion coefficient.
[a]Paired-samples statistics, degrees of freedom = 13.

among patients with AD that may attain significance with large-scale research projects. Some conventional DWI studies of AD obtained no statistically significant results for either white or gray matter [16,17]. However, some other conventional studies showed significant elevation in the hippocampal ADC [18,19]. Several other AD studies used more sophisticated techniques and showed more conclusive results. One study concluded that a high b value DWI is more sensitive to AD-related white matter degeneration than conventional DWI [20]. However, Moseley and Bammer [4] argued that the quantification of the ADC at higher b values will not be totally correct.

Table 4 Difference[a] of the ADC between the two cerebral hemispheres in the a-MCI group

Regions	Mean	Standard deviation	Correlation	Significance	*t*	Significance (2-tailed)
Corpus striatum						
Left	106.511	15.7345	0.904	0.001	−1.916	0.092
Right	111.822	19.0953				
Basal ganglia						
Left	97.189	13.3407	0.629	0.069	−0.148	0.886
Right	98.133	24.5348				
Thalamus						
Left	104.478	14.8212	0.802	0.009	0.433	0.676
Right	103.178	13.4440				
Amygdala						
Left	97.067	11.4703	0.571	0.108	0.208	0.841
Right	95.000	34.8901				
Hippocampus						
Left	97.522	16.1679	0.846	0.004	−1.107	0.300
Right	112.278	52.7150				
Frontal white matter						
Left	95.744	14.9052	0.655	0.056	−1.331	0.220
Right	100.778	11.1294				
Frontal gray matter						
Left	104.700	16.9758	0.808	0.008	−0.100	0.923
Right	105.033	14.0602				
Parietal white matter						
Left	122.567	31.9545	−0.117	0.765	−0.888	0.400
Right	221.478	328.9487				
Parietal gray matter						
Left	136.600	53.2760	0.654	0.056	−0.264	0.799
Right	140.344	48.6679				

ADC, apparent diffusion coefficient.
[a]Paired-samples statistics, degrees of freedom = 8.

Table 5 Difference[a] of the ADC between the two cerebral hemispheres in the control group

Regions	Mean	Standard deviation	Correlation	Significance	*t*	Significance (2-tailed)
Corpus striatum						
Left	94.755	10.2186	0.736	0.010	−1.149	0.277
Right	97.482	11.2861				
Basal ganglia						
Left	88.736	11.7747	0.361	0.275	0.932	0.373
Right	85.300	9.6332				
Thalamus						
Left	93.936	6.4615	0.605	0.049	0.598	0.563
Right	92.855	6.9888				
Amygdala						
Left	99.109	23.2309	0.482	0.133	0.432	0.675
Right	96.373	16.4050				
Hippocampus						
Left	97.882	17.1712	0.360	0.277	0.141	0.891
Right	97.027	18.3253				
Frontal white matter						
Left	94.473	10.6951	0.408	0.213	−2.032	0.070
Right	102.336	12.6945				
Frontal gray matter						
Left	100.245	7.9998	−0.145	0.670	−0.559	0.588
Right	104.882	25.1784				
Parietal white matter						
Left	98.873	6.1311	0.076	0.824	1.074	0.308
Right	96.464	4.7096				
Parietal gray matter						
Left	133.064	36.2246	−0.210	0.536	0.838	0.422
Right	119.373	33.4168				

ADC, apparent diffusion coefficient.
[a]Paired-samples statistics, degrees of freedom = 10.

Table 6 ADC Predictability for MMSE scores of the AD group in different cerebral regions

Predictors	Standardized coefficients β	*t*	Significance	F (Significance)[a]
Left corpus striatum	−0.885	−1.035	0.359	0.857 (0.642)
Right corpus striatum	−0.464	−0.406	0.705	
Left basal ganglia	0.997	1.033	0.360	
Right basal ganglia	−0.491	−0.410	0.703	
Left thalamus	−0.676	−0.628	0.564	
Right thalamus	1.279	0.745	0.498	
Left amygdala	−0.700	−1.470	0.216	
Right amygdala	0.240	0.533	0.622	
Left hippocampus	−0.143	−0.195	0.855	
Hippocampus right	0.413	0.442	0.682	
Left frontal white matter	−0.601	−1.275	0.271	
Right frontal white matter	0.447	0.974	0.385	
Left frontal gray matter	0.365	0.669	0.540	
Right frontal gray matter	−0.223	−0.319	0.766	
Left parietal white matter	0.711	1.768	0.152	
Right parietal white matter	0.005	0.005	0.996	
Left parietal gray matter	0.251	0.531	0.624	
Right parietal gray matter	−0.336	−0.665	0.542	

ADC, apparent diffusion coefficient; MMSE, Mini-Mental State Examination.
[a]Regression analysis' dependent variable: MMSE total.

Table 7 ADC Predictability in different cerebral regions for the total ADAS scores of the AD group

Predictors	Standardized coefficients β	*t*	Significance	F (Significance)[a]
Left corpus striatum	0.526	0.517	0.632	0.542 (0.836)
Right corpus striatum	0.869	0.640	0.557	
Left basal ganglia	−0.273	−0.238	0.824	
right basal ganglia	0.611	0.430	0.690	
Left thalamus	−0.440	−0.343	0.749	
Right thalamus	−1.802	−0.883	0.427	
Left amygdala	0.342	0.605	0.578	
Right amygdala	−0.023	−0.043	0.968	
Left hippocampus	0.093	0.107	0.920	
Right hippocampus	−0.879	−0.791	0.473	
Left frontal white matter	0.651	1.162	0.310	
Right frontal white matter	−0.100	−0.183	0.864	
Left frontal gray matter	−0.660	−1.017	0.367	
Right frontal gray matter	0.429	0.516	0.633	
Left parietal white matter	−0.130	−0.272	0.799	
Right parietal white matter	0.846	0.660	0.545	
Left parietal gray matter	−0.022	−0.040	0.970	
Right parietal gray matter	−0.365	−0.609	0.576	

ADAS, Alzheimer's disease Assessment Scale; ADC, apparent diffusion coefficient.
[a]Regression analysis' dependent variable: ADAS total.

In another research, with a map of subcortical mean diffusivity superimposed on the corresponding anatomic image, significant mean diffusivity elevation was observed in the medial aspect of the right frontal, bilateral parietal, and right temporal lobes [21]. Diffusion differences between patients and controls were observed after controlling for volumetric differences in some of the cerebral regions, whereas independent volumetric differences were observed in other regions [22].

This result showed a marked correlation of diffusivity measures between the right and the left sides regarding the corpus striatum, amygdala, hippocampus, parietal white matter, and frontal gray matter in order, in the AD group. In addition, similar correlations were found in the a-MCI group including the corpus striatum, hippocampus, gray matter, and thalamus. Only low significant correlations were found in the corpus striatum and thalamus in the control group. This may suggest that in the dementia and predementia stages, microstructural diffusion changes in such regions occur on both sides. However, the DWI did not predict the cognitive or the behavioral dysfunctional severities of AD manifestations measured by the neuropsychological ratings. In a previous study, diffusion measures did not correlate with the severity of dementia [18]. Weak correlations were found in another study between clinical scales and ADC values in the hippocampi, temporal lobes, left frontal lobe, and left occipital lobe [23]. However, more sophisticated diffusivity techniques might be helpful.

In this study, the ADC means of the MCI group were closer and not significantly different from that of the AD group in almost all regions. These differences from the control group may reach significance in large-scale research projects. It was argued that brain imaging provides useful information, but these diagnostic tools remain imprecise and have not been validated for routine use. The process of the diagnosis must therefore focus on the analysis of the cognitive impairment of the predementia phase of AD. This will help identify 'hippocampal amnesia syndrome' suggestive of the

diagnosis of AD [14]. However, higher baseline hippocampal diffusivity was associated with a greater hazard of progression to AD in a-MCI [24]. Hence, it was argued that DWI may help identify patients with a-MCI who will progress to AD as efficiently as or better than structural MRI measures of hippocampal atrophy.

Limitations

The sample size was small as it was difficult to recruit pure concordantly diagnosed AD and a-MCI cases. The wide age range (50–80 age years) may result in differences between individuals in different stages of age-related brain involution changes.

Conclusion

Conventional DWI only showed a trend toward increased diffusivity, with no statistically significant results obtained for either gray or white matter. This may reach significance with large-scale research projects. In addition, the use of more sophisticated diffusion techniques may yield more precise results. In the dementia and predementia stages, microstructural diffusion changes in certain regions occur on both sides. These can contribute toward the diagnostic accuracy of AD and the predementia stage but may not help in the assessment of cognitive or behavioral dysfunctional severity.

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Conflicts of interest

There is no conflict of interest to declare.

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الملخص العربي

تشخيص مرض الزهيمر: الدور المحتمل لتقنية التصوير الوظيفى

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يفترض فى هذا البحث أن أضافه التصويرالوظيفى لنفاذية جزئ الماء فى المخ بالرنين المغناطيسى إلى التشخيص الأكلينكى لمرض الزاهيمر قد يساعد فى دقة التشخيص. ولذلك يهدف البحث فى الأساس إلى إيجاد رابط بين تلك النفاذيه والتشخيص. وقد تم تكوين ثلاث مجموعات: الأولى من 14 مريض الزاهيمر طبقا للدليل الامريكى التشخيصي والاحصائى الرابع للاضطرابات العقلية والثانية من 9 مرضى بالقصور المعرفي البسيط من نوع النسيان طبقا لمعايير مايو كلينك والثالثة من 11 شخص كعينة ضابطة. وقد استخدم كل من اختباري المقياس المختصر للحالة العقلية ومقياس تقييم مرض الزهيمر وذلك لقياس شدة الأعراض المعرفية والسلوكية. وتم قياس نفاذية الماء خلال مناطق المخ المختلفة بواسطة التصوير بالرنين المغناطيسى للنفاذيه الموزونه. ولقد أظهرت النتائج ازدياد نفاذيه جزئ الماء فى كل مناطق المخ التي تم اختيارها ولكن بدون دلاله احصائيه. وكانت متوسطات النفاذية بين مرضى القصور المعرفي البسيط اقرب لتلك المتوسطات لمرض الزهيمر وبدون فروق إحصائية. ولم تظهر أية علاقة إحصائية بين النفاذية ومقياسي شدة الأعراض. وخلص البحث إلى انه هناك ميل لزيادة النفاذية فى مناطق المخ المختارة بين مرضى الزهايمر ولكن بدون دلالة إحصائية.