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Lateral temporal atrophy is a better predictor of baseline MMSE scores than hippocampal atrophy in Alzheimer's disease: A retrospective cross-sectional study

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Abstract

Background: Mini-Mental State Exam (MMSE) is widely used for the cognitive assessment in Alzheimer's disease (AD), but its interpretation could be affected by acute medical conditions, depression, or anxiety. Conversely, brain magnetic resonance imaging (MRI) is an independent diagnostic tool to evaluate specific cerebral pathology. Many studies have investigated the correlation between MRI visual scales such as hippocampal atrophy and baseline MMSE in AD patients. However, the correlation between a comprehensive MRI visual scale and baseline MMSE in people with AD remains less well known. *Method:* We retrospectively collected records of outpatients diagnosed with probable AD according to DSM -5 criteria. The comprehensive visual rating scale (CVRS) was used to semiquantitatively measure structural changes and vascular lesions. The relationship between MRI changes and baseline MMSE was evaluated using Bayesian model averaging (BMA). *Results:* A total of 65 patients, among whom 21 (32.31%) had early-onset AD, were included. Lateral temporal atrophy, level of education, and late age at onset were the strongest independent predictors of baseline MMSE. Furthermore, hippocampal atrophy was only correlated with delayed recall, while temporal atrophy was correlated with orientation, attention, language, and visual-spatial items of the MMSE. The cerebral atrophy and small vessel lesions scores of the late-onset AD group were significantly higher than those of the early-onset group, despite negligible differences in education and MMSE. *Conclusion:* Our study suggests lateral temporal atrophy correlates with baseline MMSE scores in people with AD better than the hippocampal atrophy. Age-related atrophy and silent small vessel disease lesions may have negligible impact on AD patients' cognitive impairment.

Keywords: CVRS, MMSE, early-onset Alzheimer disease, late-onset Alzheimer disease

INTRODUCTION

The number of people living with Alzheimer's disease (AD) more than doubled from 20.2 million in 1990 to 43.8 million in 2016, significantly straining the global healthcare systems.[1] Appropriate diagnostic tools for people with dementia are currently under development to better stage their disease trajectory and deliver effective treatments.[2] Several cognitive tests are being used extensively in clinical settings due to their well-established evidence in diagnostic performance, including the Mini-mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and the Mini-Cog test.[3] However, their interpretation are often heavily subjected to bias from patient's acute cognitive worsening, level of education, language [4], auditory and visual abilities[5], depression, anxiety, or sleep deprivation.[6]

Visual scales from brain magnetic resonance imaging (MRI) scan are independent tools for semiquantitative measurement of biomarkers of AD progression, such as cerebral atrophy, white matter hyperintensity, lacune, and microbleeds.[7] Based on the proposed pathology of AD,

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classification of disease severity using specific cerebral changes could be feasible.[8] Previously, many researchers have utilized the medial temporal atrophy (MTA) score, developed by Schelten *et al*.[9], to semiquantitatively evaluate the correlation between hippocampal atrophy and MMSE scores in people with AD. The resulted correlation was indeed significant, though its strength was only moderate.[10,11] More recently, Ferreira *et al*. showed that AD could be classified into four different biological subtypes based on their neuropathology and neuroimaging characteristics. The study also highlighted the differences in AD pathology between people with early-onset (EOAD) and late-onset AD (LOAD).[12] Therefore, evaluation of only MTA is no longer sufficient to understand the correlations between numerous AD-related structural changes on MRI and the MMSE scores.

The Comprehensive MRI Visual Rating Scale (CVRS) was developed by Jang *et al*. to evaluate specific pathological lesions in people with AD by combining four indices, including hippocampal, cortical, subcortical atrophy, and small vessel disease lesions.[13] The aims of our study are two fold. Firstly, to evaluate the correlation between the CVRS and baseline MMSE to aid in the diagnosis and staging of patients with AD. Secondly, we aim to shed light on the different correlations of MRI and MMSE between people with EOAD and LOAD.

METHODS

This study included people with AD diagnosed by dementia specialists based on the DSM-5 criteria at Hospital 30-4 and University Medical Center at Ho Chi Minh City, Vietnam, from 2018 to 2021.[14] All patients received a comprehensive dementia evaluation, including brain MRI and neuropsychological tests consisting of the validated Vietnamese version of Mini-mental state examination (MMSE)[15]; Word List Recall: Immediate Recall, Delayed Recall, Delayed Recognition; Trail Making Test A (TMT-A); Trail Making Test B (TMT-B); Digit Span Forward; Digit Span Backward; Verbal Fluency; and Clock drawing test. Institutional review boards at University Medical Center (UMC) at Ho Chi Minh City and Hospital 30-4 approved our study (830/HĐĐĐ – ĐHYD).

We included patients with brain MRI dated within three months from their neuropsychological tests and excluded those with severe visual and auditory conditions, depression, or anxiety. The following information were collected, including baseline characteristics (age at onset, level of education, comorbidities), MMSE scores, and brain MRI scan during their first evaluation.

Brain images were acquired with 1.5 Tesla MRI scanners (Avanto and Amira: Siemens Healthineers) and a 3.0 Tesla MRI scanner (Verio: Siemens Healthineers). The minimum protocol includes T1W 3D gradient echo (three-dimensional (3D) magnetization-prepared rapid gradient-echo: MPRAGE), Axial FLAIR (Fluid-attenuated inversion recovery), Axial T2W SE, and T2*-weighted gradient-echo (GRE)). The images were then reconstructed and analyzed using RadiAnt DICOM Viewer software. CVRS was used to evaluate structural changes in brain images. We used three coronal planes in T1-weighted scans to evaluate the hippocampus, temporal, frontal and parietal atrophy, and the T1-weighted axial plain to evaluate lateral ventricular expansion. Axial plain in FLAIR was used to quantify the small vessel disease, including white matter lesions, cerebral microbleeds, and lacune. Detailed instruction on CVRS evaluation is outlined in Jang *et al*.[13] Two authors, a neurologist (N.V.K) and a radiologist (T.T.S) with three years of experience, blinded to the results of the cognitive tests, independently rated the CVRS score. The final score was resolved by discussion between the two raters. Patients were classified as EOAD and LOAD based on their age of onset before or after 65 years old. We estimated that a sample size of at least 47 patients were necessary to achieve sufficient statistical power to determine a clinically significant correlation between CVRS and MMSE scores with $r > 0.4$, $\alpha = 0.05$, and $\beta = 0.8$. The following formula was used[16]:

$$N = \left(\frac{Z_\alpha + Z_\beta}{C}\right)^2 + 3$$

The T-test or Mann-Whitney U test was used to analyze continuous variable while Chi-square or Fisher exact test was used for categorical variable where applicable. Continuous variables with non-normal distribution are presented as median and interquartile range. Bayesian model averaging (BMA) was employed to select the best model to predict MMSE based on patients' baseline characteristics and CVRS. By using BMA, we could find optimal models to predict MMSE without assuming the single best model, while acknowledging that there is uncertainty in statistical modeling.[17] All computations were performed with R version 4.0.3.[18]

RESULTS

We included 21 patients with EOAD and 44 patients with LOAD. Among them, 60 (92.31%) patients had 1.5T MRI scans and 5 (7.69%) patients had 3T MRI scans. The median and interquartile of delay between dates that neuropsychological tests and MRI were performed was 1 [0,4] day.

Baseline characteristics

Table 1 presents the baseline characteristics of all included patients. We also presented the differece in EOAD and LOADs. There was no significant difference in duration of onset, level of education, gender, and comorbidity profiles between the two groups.

CVRS and MMSE score

The CVRS and MMSE scores of the included patients and EOAD, LOAD subgroups, are presented in Tables 2 and 3. People with LOAD had a significantly higher CVRS total score and subscores, except for right hippocampal atrophy and microbleed subscores, than those with EOAD ($p< 0.05$). In contrast, both groups shared similar MMSE total scores at baseline ($p = 0.720$).

Correlation between CVRS and MMSE of the included patients

A correlation plot to illustrate the correlation between CVRS and MMSE scores is presented in Figure 1. The CVRS total score, left hippocampal atrophy, right hippocampal atrophy and temporal atrophy were significantly correlated with MMSE ($p < 0.01$) with Spearman correlation coefficient r of -0.36, -0.35, -0.33, -0.49, respectively. The frontal atrophy, anterior horn enlargement, and posterior horn enlargement were also correlated with MMSE with $p < 0.05$. Due to the intercorrelation between predictors of MMSE, BMA was used to control confounders. We included age, age-onset, level of education, and CVRS subscores as variables to find a predictive model for the MMSE total scores. Our BMA model (Table 4) showed that temporal atrophy (Prob = 100%; BMA posterior mean of –4.43), late-onset (Prob = 60.2 %; BMA posterior mean of 3.82), and level of education (Prob = 100%; BMA posterior mean of 0.54) were the strongest predictors of MMSE total score while hippocampal atrophy was not a predictor of MMSE (Prob = 0%; BMA posterior mean of 0). BMA was further used to investigate MMSE subscore predictors. Table 5 presents the MMSE subscores with their strongest predictors. Some of the MMSE subscores were not included as no significant relationships were found. Temporal atrophy was a significant predictor of orientation, attention, verbal language, and visual-spatial abilities, while hippocampal atrophy was only predictive of delayed recall.

Correlation between CVRS and MMSE in LOAD

We have also presented a correlation plot to illustrate the correlation between CVRS and MMSE scores in LOAD in Figure 2. The CVRS total score, left hippocampal atrophy, and temporal atrophy were significantly correlated with MMSE ($p < 0.01$) with Spearman correlation coefficient r of -0.37, -0.30, -0.46, -0.34, respectively. The anterior horn enlargement were also correlated with MMSE with $p < 0.05$.

We have also performed a subgroup analysis on the correlation between CVRS and MMSE in patients in LOAD. The correlation plot between

Table 1: Patients' characteristics

	Total (n = 65)	EOAD (n=21)	LOAD (n = 44)	p-value
Demographic				
Age	71.0 [65.0;78.0]	62.0 [59.0;65.0]	74.5 [71.0;79.0]	**<0.001**
Age of onset	69.0 [63.0;75.0]	60.0 [55.0;63.0]	73.8 [69.0;77.0]	**<0.001**
Disease duration since onset	1.00 [1.00;2.00]	2.00 [1.00;2.00]	1.00 [0.88;2.00]	0.377
Level of education (years)	9.00 [5.00;12.0]	7.00 [6.00;12.0]	10.0 [5.00;12.5]	0.707
Male	24 (36.9%)	7 (33.3%)	17 (38.6%)	0.679
Comorbidities				
Hypertension	31 (47.7%)	8 (38.1%)	23 (52.3%)	0.285
Diabetes mellitus	9 (13.8%)	3 (14.3%)	6 (13.6%)	0.99
Dyslipidemia	37 (56.9%)	13 (61.9%)	24 (54.5%)	0.575

Table 2: CVRS score and subscores between EOAD and LOAD

	Total (n = 65)	EOAD (n=21)	LOAD (n = 44)	p-value
Total scores	17.0 [13.0;21.0]	16.0 [12.0;21.0]	17.5 [13.8;21.0]	0.720
Subscores				
Temporal orientation	2.00 [0.75;3.00]	1.00 [0.00;3.25]	2.00 [1.00;3.00]	0.321
Spatial orientation	4.00 [3.00;4.00]	4.00 [2.75;4.25]	4.00 [3.00;4.00]	0.948
Immediate memory	3.00 [3.00;3.00]	3.00 [3.00;3.00]	3.00 [3.00;3.00]	0.492
Attention	1.00 [1.00;3.00]	2.00 [1.00;4.00]	1.00 [0.75;2.00]	0.244
Delayed recall	0.00 [0.00;1.00]	0.00 [0.00;1.00]	0.00 [0.00;1.00]	0.877
Verbal repetition	0.00 [0.00;1.00]	0.00 [0.00;1.00]	0.00 [0.00;1.00]	1.000
Verbal comprehension	2.00 [2.00;3.00]	2.00 [1.00;3.00]	2.00 [2.00;3.00]	0.410
Reading	1.00 [0.00;1.00]	0.00 [0.00;1.00]	1.00 [0.00;1.00]	**0.039**
Constructional praxis	0.00 [0.00;1.00]	0.00 [0.00;1.00]	0.00 [0.00;1.00]	0.579
Writing	0.50 [0.00;1.00]	0.00 [0.00;1.00]	1.00 [0.00;1.00]	0.277

EOAD: early onset Alzheimer's disease; LOAD, late onset Alzheimer's disease

CVRS subscore and MMSE also showed that the CVRS total score, left hippocampal atrophy, anterior horn and temporal atrophy were significantly correlated with MMSE (Figure 2). BMA was then used to study the correlation between age, age-onset, level of education, and CVRS subscores with MMSE. The BMA model (Table 5) also showed that temporal atrophy (Prob = 93%; BMA posterior mean of –3.22), and level of education (Prob = 100%; BMA posterior mean of 0.47) were the strongest predictors of MMSE total score while hippocampal atrophy was not a predictor of MMSE (Prob = 0%; BMA posterior mean of 0) in LOAD.

DISCUSSION

Previous reports have revealed the modest correlations between hippocampal and parietal atrophy and baseline MMSE in people with AD[10,11,19] using the hippocampal[9] and parietal atrophy (PA) scores, respectively.[20] However, to the best of our knowledge, the correlation of combined visual scale with baseline MMSE in people with AD has not yet been examined.

Table 3: MMSE score and subscores between EOAD and LOAD

	Total (n = 65)	EOAD (n=21)	LOAD (n = 44)	p-value
CVRS	12.0 [8.00;15.0]	8.00 [4.00;12.0]	13.5 [10.0;18.2]	**<0.001**
Frontal atrophy	1.00 [1.00;2.00]	1.00 [1.00;1.00]	2.00 [1.00;2.00]	**<0.001**
Left Hippocampal atrophy	1.00 [1.00;2.00]	1.00 [0.00;2.00]	2.00 [1.00;2.00]	**0.013**
Right hippocampal atrophy	1.00 [1.00;2.00]	1.00 [0.00;2.00]	2.00 [1.00;2.00]	**0.061**
Temporal atrophy	2.00 [1.00;2.00]	1.00 [0.00;2.00]	2.00 [1.00;2.00]	**0.001**
Parietal atrophy	2.00 [1.00;2.00]	1.00 [1.00;2.00]	2.00 [1.00;2.00]	**0.006**
Anterior horn enlargement	1.00 [1.00;2.00]	0.00 [0.00;1.00]	1.00 [1.00;2.00]	**<0.001**
Posterior horn enlargement	1.00 [1.00;2.00]	1.00 [0.00;1.00]	1.00 [1.00;2.00]	**0.005**
White matter Hyperintensity	1.00 [1.00;2.00]	1.00 [0.00;1.00]	1.50 [1.00;2.00]	**0.001**
Lacune	0.00 [0.00;1.00]	0.00 [0.00;0.00]	0.00 [0.00;1.00]	**0.017**
Microbleed	0.00 [0.00;0.00]	0.00 [0.00;0.00]	0.00 [0.00;1.00]	0.083

CVRS: Comprehensive Visual Rating Scale

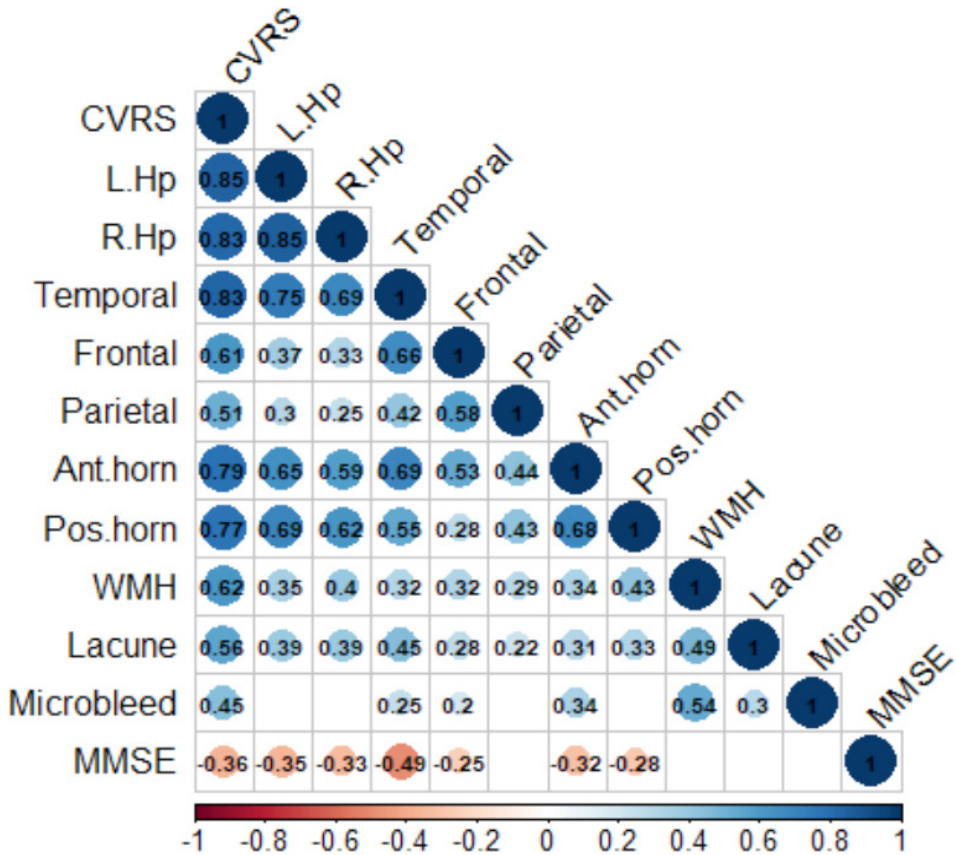

Figure 1. Correlation plot between MMSE and CVRS subscores.
Correlation plot between MMSE and CVRS subscores. Only the correlations with p-value < 0.05 are shown. Blue and red circles indicate a positive and negative correlation, respectively. The size and the shade of the circle both express the strength of correlation. Ant. horn, anterior horn; CVRS, Comprehensive Visual Rating Scale; L. Hp, Left hippocampal; MMSE, Mini-Mental State Examination; Pos. horn, posterior horn; R. Hp, Right hippocampal; WMH, white matter lesions.

Our study shows that the visual rating scale of temporal atrophy was more strongly correlated with baseline MMSE scores in people with AD than other brain structural changes. Specifically, BMA analysis on MMSE subscores revealed that temporal atrophy affected orientation, attention, language, and visual-spatial abilities, while hippocampal atrophy only affected delayed recall.

The coronal image to evaluate temporal atrophy in CVRS was sliced through the temporal stems and reveals the atrophy of the lateral temporal gyrus. This could explain why a correlation between the lateral temporal lobe and baseline MMSE was detected. Other studies using Relevance Vector Regression[21] and Voxel-Based Morphometric[22] also found significant correlations of lateral temporal atrophy with baseline MMSE in people with AD. Hudson *et al*. also reported that people with temporal lobe epilepsy had a lower score on attention tests than the control group.[23] This correlation between the lateral temporal lobe and MMSE could be attributed to the temporoparietal junction area and the ventral attention system's role in attention.[24] Our study also revealed significant differences in correlations of MRI changes and cognitive performance between people with EOAD and LOAD. Despite similar MMSE scores and level of education, people with LOAD had significantly higher CVRS scores than those with EOAD. This result was in accordance with Eckerström *et al*.[25], suggesting that age-related atrophy and silent small vessel disease lesions may not significantly impact the cognitive reserve in AD.

Our study used BMA to select the best predictors of baseline MMSE among the AD-specific cerebral changes. BMA is an application of Bayesian inference to find optimal models to

Table 4: BMA model for MMSE total score

	Probability (%)	Model 1	Model 2	Model 3
Intercept	100.0	16.39	5.18	5.73
Left hippocampal atrophy	0	.	.	.
Right hippocampal atrophy	0	.	.	.
Temporal atrophy	100.0	-4.43	-4.49	-4.38
Frontal atrophy	0	.	.	.
Parietal atrophy	0	.	.	.
Anterior horn enlargement	0	.	.	.
Posterior horn enlargement	0	.	.	.
White matter hyperintensity	0	.	.	.
Lacune	0	.	.	.
Microbleed	0	.	.	.
Age	12.8	.	.	0.19
LOAD	60.2	3.82	.	0.18
Age onset	27.0	.	0.21	.
Level of education	100.0	0.54	0.50	0.50
Total variable in the model		3	3	3
r2		0.55	0.54	0.52
BIC		-34.77	-33.16	-31.67

BIC: Bayesian information criterion; BMA: Bayesian Model Averaging; MMSE, Mini-Mental State Examination

predict without assuming the single best model, while acknowledging that there is uncertainty in statistical modeling.[17] Although BMA suggested several models in our study, other models had higher Bayesian information criterion (BIC), and the MRI structural lesions predictors were similar across all proposed models.

There are several limitations worth mentioning. Firstly, as this is a retrospective study, the MMSE was rated by different raters and subjected to inter-rater bias. Interpretation of MMSE could also be influenced by acute cognitive impairment, auditory and visuality capacity[5], depression, anxiety, or sleep deprivation.[6] Therefore, we have attempted to minimize this by including people with AD only in the outpatient department, who are less likely to experience acute cognitive impairments during their visits. All our patients presented with amnestic AD. Also, our study also did not account for the different biological subtypes of AD, including posterior cortical atrophy subtypes of AD. Although the CVRS included the evaluation of parietal lobe, the occipital lobe atrophy was omitted. These

Table 5: BMA model for MMSE subscores

MMSE subscores	Temporal atrophy	Hippocampal atrophy	Posterior horn enlargement	Lacunes	Level of education	Age onset
Temporal orientation	+		+	+	+	+
Spatial orientation	+				+	
Attention	+				+	
Delayed recall		+		+		
Naming	+					
Verbal language	+					+
Written language					+	+
Visual-spatial	+				+	
Writing	+				+	

BMA: Bayesian Model Averaging; MMSE, Mini-Mental State Examination. (+) indicates statistically significant predictors in each model that predict MMSE items.

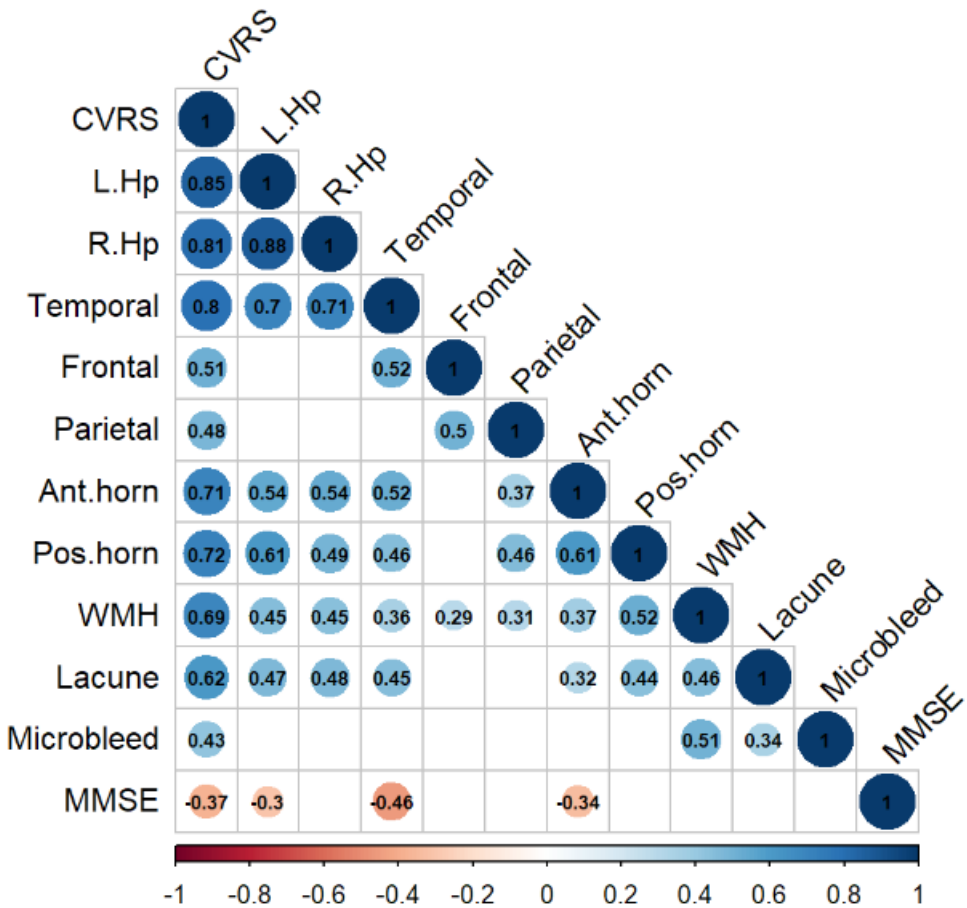

Figure 2. Correlation plot between MMSE and CVRS subscores in LOAD patients.
Correlation plot between MMSE and CVRS subscores in LOAD patients. Only the correlations with p-value < 0.05 are shown. Blue and red circles indicate a positive and negative correlation, respectively. The size and the shade of the circle both express the strength of correlation. Ant. horn, anterior horn; CVRS, Comprehensive Visual Rating Scale; L.Hp, Left hippocampal; MMSE, Mini-Mental State Examination; Pos. horn, posterior horn; R.Hp, Right hippocampal; WMH, white matter lesions.

limitations could explain the modest predictive value of our model.

Overall, our study investigated the correlation of CVRS and its subscores with MMSE in people with probable AD. We found that lateral temporal atrophy in CVRS most strongly correlates with MMSE and its subscore than hippocampal atrophy, using Spearman correlation and multivariate analysis with BMA. Therefore, lateral temporal atrophy could be used as a predictor of baseline MMSE scores in people with AD. Finally, our results also suggested that some pathological indicators on MRI, such as age-related atrophy and silent small vessel disease, may not have a significant impact on the cognitive performance of people with AD.

In conclusion, our study suggests that lateral temporal atrophy correlates better with baseline MMSE scores in people with AD than hippocampal atrophy. Future investigations are needed to further clarify the correlation between lateral temporal atrophy and more comprehensive neuropsychological measures. Age-related atrophy and silent small vessel disease lesions may have negligible impact on AD patients' cognitive impairment.

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Table 6: BMA model for MMSE total score in LOAD patients

	Probability (%)	Model 1	Model 2
Intercept	100.0	18.85	16.95
Left hippocampal atrophy	0	.	.
Right hippocampal atrophy	0	.	.
Temporal atrophy	93	-3.22	0
Frontal atrophy	0	.	.
Parietal atrophy	0	.	.
Anterior horn enlargement	7	.	-2.35
Posterior horn enlargement	0	.	.
White matter hyperintensity	0	.	.
Lacune	0	.	.
Microbleed	0	.	.
Age	0	.	.
Level of education	100	0.47	0.43
Total variable in the model		2	2
r2		0.43	0.35
BIC		-14.91	-9.77

BIC: Bayesian information criterion; BMA: Bayesian Model Averaging; MMSE, Mini-Mental State Examination

DISCLOSURE

Ethics: This study was approved by the Institutional Review Board at University of Medicine & Pharmacy at Ho Chi Minh city to conduct at University Medical Center at Ho Chi Minh City and Hospital 30-4 (830/HĐĐĐ – ĐHYD). Patient written informed consents were not obtained as this is a retrospective and observational study. The conduct of this study did not affect the patients' treatment course.

Data availability: The data used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Financial support: None

Conflict of interest: None

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