MRI VISUAL RATING OF COGNITIVE IMPAIRMENT IN ELDERLY PATIENT

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Abstract

Background:

Neurocognitive impairment is an acquired deficiency of cognitive abilities that significantly interferes with performance of daily activities. Early diagnosis is key in the management which is dependent on employing appropriate biomarkers. Cerebral atrophy is a valid neuroimaging biomarker. This study aims to correlate cerebral atrophy using standard visual assessment scales on Magnetic Resonance Imaging (MRI) with cognitive status using a standard cognition assessment tool in elderly patients at the University College Hospital, Ibadan Nigeria.

Methods:

Patients over 60 years who presented for cranial MRI and met the inclusion criteria were recruited into the study which spanned a 10-month period. Relevant demographic and clinical information were obtained from the participants. Visual rating scores comprising Medial Temporal Atrophy (MTA), Fazekas, Koedam and Global Cortical Atrophy (GCA) of the images were entered into a standard proforma and the Statistical Package for the Social Sciences (SPSS) software version 20. Data was analyzed using chi square, student t-test, Spearman Rho test, Kruskal-Wallis test, Mann-Whitney Wilcoxon test, and logistic regression analysis.

Results:

Spearman Rho test showed significant association between MTA and Fazekas scores and between Koedam and GCA scores. Significant association between MTA scores and severity of cognitive impairment by Mann-Whitney Wilcoxon test was shown. Kruskal-Wallis showed significant association between Fazekas scores and severity of cognitive impairment.

Conclusion:

Medial temporal lobe atrophy is a useful marker of cognitive impairment and severity of cognitive impairment. White matter disease is significantly associated with the presence of and severity of cognitive impairment. Both can serve as useful neuroimaging biomarkers.

Keywords

Atrophy, brain, cognitive impairment, elderly, medial temporal lobe, MRI.

Introduction

Neurocognitive impairment is acquired deterioration in cognitive abilities and in the mental faculties that impair the successful performance of daily activities.[1] These cognitive abilities include memory, language, visuo-spatial ability, calculation, judgment, problem solving skills,

orientation, registration, attention among others. Memory is the most common cognitive ability loss with neurocognitive impairment.[1] According to the Diagnostic and Statistical Manual of Diseases-5 (DSM-5), neurocognitive impairment is synonymous with dementia and it is currently preferred to dementia- latin word for 'mad' or 'insane'.[2] Dementia is stigmatizing and unacceptable to patients.

Alzheimer's disease, vascular neurocognitive impairment, frontotemporal neurocognitive impairment, Huntington's neurocognitive impairment, neurocognitive impairment with Lewy bodies, neurocognitive impairment associated with Parkinson's disease and Creutzfeldts-Jacob disease are patterns of degenerative neurocognitive impairment. Age is the single strongest risk factor for neurocognitive impairment. Other major risk factors include, female gender, cardiovascular disease, and illiteracy.[3] Individuals with mild cognitive impairment have significant, clinically identifiable and measurable cognitive deficit and memory loss, that does not disrupt successful daily activities, functioning and independent living.[4] It can be extremely difficult to make a distinction between mild cognitive impairment and neurocognitive impairment as an estimated third of individuals with the former progress to the latter.[4] The prevalence of age-related health problems is becoming an important public health concern as proportions of older individuals in populations worldwide grow.[4] There is a population growth due to the decline in deaths attributable to communicable diseases.[5] There is an evident demographic and epidemiologic transition trend over Africa and the impact of population aging in sub-Saharan Africa will increasingly augment the burden of degenerative diseases in the region. For these reasons, age-related diseases like neurocognitive impairment are fast becoming a new healthcare priority for Africa including Nigeria.

Neurocognitive impairment is one of the major causes of disability in older people.[4] Rural living, common in Africa is a documented risk factor for neurocognitive impairment especially Alzheimer's disease.[6] From projections, it is estimated that by 2050 the number of individuals older than 60 years will be approximately 2 billion and will account for 22% of the world's population.[4] Worldwide, the prevalence of neurocognitive impairment among those aged 60 years and above ranges from 5-7%.[3] The Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-V), represents the current 'gold standard' for dementia diagnosis worldwide[5] and puts the overall age adjusted prevalence of dementia in Ibadan, Nigeria at 2.29%.[7]

Neuroimaging plays an essential role in the management of neurocognitive impairment. Cranial magnetic resonance imaging (MRI), cranial computed tomography (CT), positron emission tomography (PET) and single photon emission computerized tomography (SPECT) are neuroimaging modalities relevant to the management of neurocognitive impairment. Neuroimaging studies can rule out primary and metastatic neoplasms, locate areas of infarction, detect subdural hematomas, and suggest normal pressure hydrocephalus (NPH) or diffuse white matter disease and help to establish a regional pattern of atrophy. Support for the diagnosis of Alzheimer's disease (AD) include hippocampal atrophy in addition to posterior-predominant cortical atrophy.

Focal frontal or anterior temporal atrophy suggests frontotemporal dementia (FTD).

MRI is the modality of choice because of the non-requirement of ionizing radiation and its superior soft tissue contrast and details.[8,9] MRI differentiates between neurocognitive impairment associated with metabolic diseases from inflammatory diseases and may also show abnormalities amenable to surgical treatment in a significant percentage of patients with neurocognitive impairment. MRI is able to identify focal lesions. Cost and the presence of metallic implants or medical devices common in the elderly limit the use of MRI.[8] CT is useful when there are contraindications to the use of MRI.[10] Several neuroimaging biomarkers can be employed in the diagnosis and clinical management of dementia.[11,12]

Neurocognitive impairment is one of the major causes of disability and mortality in the elderly. [4,13] It is the fourth major cause of death in the adult populace after heart diseases, cancer and stroke.[8] It has huge health, economic and social implications[4,8] as well as profound psychological and cultural ramifications.[7] The survival time for people living with neurocognitive impairment is about 6-9 years.[14]

A lot of resources are needed in the care of neurocognitive impairment patients.[15] Burden of care in terms of physical work, psychological distress and financial obligations is great.[16] There is no policy for the care of the elderly in Nigeria. Healthcare cover for the elderly in Nigeria is grossly inadequate.[14] There is also a cultural perception that many symptoms of neurocognitive impairment are simply features of old age, so the elderly is relegated to the background. There is no definitive cure for progressive dementia disorders presently but studies have shown that if dementia is detected and diagnosed early it can be better managed.[5]

Early diagnosis of neurocognitive impairment is dependent on using appropriate biomarkers. These biomarkers include neuroimaging biomarkers, chemical biomarkers, histologic biomarkers, and genetic biomarkers. There are many neuroimaging biomarkers of cognitive impairment. However, the visual rating scales evaluated on magnetic resonance imaging have been chosen for this study because they offer a cost-effective diagnostic tool that is ideally suited for implementation in clinical practice. They include Koedam scale, medial temporal lobe atrophy (MTA), global cortical atrophy (GCA) and Fazekas scale. Using these visual rating scales, attention will be focused on brain regions susceptible to change in neurocognitive impairment and there will be structured reporting of these findings. Visual rating scales can improve the sensitivity, reliability and diagnostic value of radiological image interpretation. It is also relatively easy to perform. However, some intra and inter-observer variabilities may occur.[17]

Numerous neuroimaging biomarkers exist and have been studied in details. However, a review of the literature shows paucity of local data in Africa and in particular, Nigeria[3] with respect to these neuroimaging biomarkers. This study will focus on progressive (degenerative) neurocognitive impairment and aims to determine the accuracy of visual assessment scales of cerebral atrophy as independent neuroimaging biomarkers of cognitive impairment and their correlation with age,

educational status and severity of cognitive impairment in patients greater than 60 years of age.

Materials and methodology

Study site

The study was conducted at the Magnetic Resonance Imaging suite, Department of Radiology, University College Hospital, Ibadan.

Duration of study

The study spanned 10 months (September 1, 2016 – June 20, 2017).

Study design

A descriptive study among patients above 60 years with clinical diagnosis of neurocognitive impairment, being managed at the University College Hospital, Ibadan, who met the inclusion criteria. Objective assessment of the neuro-cognitive status of the patient was done by the neurologist using the MMSE while results were blinded to the radiologist before neuroimaging visual analysis. The data collected was recorded in demography, clinical data and mini-mental state examination sheets. The patients had cranial MRI performed using 0.36 T MR (MagSense 360, Mindray). All subjects were scanned according to a standard dementia MRI protocol: Axial, coronal and sagittal T1-weighted, T2-wieghted and FLAIR spin-echo sequences. Radiological evaluation of the patients was performed using the visual assessment scores (Koedam, MTA, GCA, Fazekas) of the brain images acquired. The radiologic findings were documented on the radiology data sheet.

Inclusion Criteria

1. Patients 60 years of age or older (male and female).

2. Patients who have clinical diagnoses of neurocognitive impairment.

Exclusion Criteria

1. Retroviral positive patients.
2. Patients with moderate-severe head trauma (GCS < 13).

Sampling strategy (patient selection)

Serial recruitment (convenience) sampling method was employed. Elderly patients above 60 years with neurocognitive impairment, being managed at the University College Hospital, Ibadan were recruited into the study. Cranial MRI is part of the routine management of patients with neurocognitive impairment, making it relatively easy to recruit these patients. The details of the study and the procedure were thoroughly explained to the participants and their care givers in at least one simple comprehensible language (English and Yoruba) which the patient or care giver understands after which informed consent was obtained. The patients who did not give consent or meet the inclusion criteria were excluded from the study.

Sample size

The sample size after adjusting for attrition was 50.

Clinical evaluation

Baseline demographic data as well as relevant clinical information were obtained using a structured data proforma. The diagnosis of neurocognitive impairment was made according to the DSM –V criteria..[2] The diagnosis of mild cognitive impairment (MCI) is a clinical judgment and is based on the following criteria:

1) memory complaint documented by the patient and collateral source;

2) normal general cognition as determined by measures of general intellectual function and mental status screening instruments;

3) normal activities of daily living;

4) not demented (DSM-V);

5) memory impairment;

6) clinical Dementia Rating score of 0.5.[18]

The mini mental state examination (MMSE) (Appendix 2) was used to classify the patient's findings as mild, moderate or severe neurocognitive impairment.[19]

However, specific diagnosis of neurocognitive impairment subtype may not be attained until after some patient's demise (at autopsy).

The Mini Mental State Examination (MMSE)

The MMSE is the best known, most widely used and the most important measure of cognition in clinical practice worldwide.[16,20] It is commonly used in medicine to screen for neurocognitive impairment as well as to systematically, repeatedly, routinely and thoroughly assess mental status in patients with neurocognitive impairment. It also estimates the severity and progression of neurocognitive impairment and used to follow the course of cognitive changes in an individual over time. Therefore, it is an effective tool for documenting an individual's response to treatment and other intervention. It examines functions including registration, attention and calculation, recall, language, ability to follow simple and complex commands and orientation. The MMSE is effective as a screening instrument to categorize patients with cognitive deficits into those with mild, moderate or severe neurocognitive impairment[21]:

1) ≥ 24 points is Normal cognition.

2) 19-23 points is Mild cognitive impairment

3) 10-18 points is Moderate cognitive impairment

4) ≤ 9 points is Severe cognitive impairment

Anthropometry

The weight (kilogram), height (meters), body mass index (kg/m^2), waist circumference (centimeters) and hip circumference (centimeters), waist hip ratio were obtained for each subject. The weight was measured in kilograms using a beam balance scale with subjects wearing light clothing and no shoes. Height was measured with a stadiometer to the nearest centimeter without the subject wearing shoes, caps or headgear and standing with the straightened back to the measuring rod and looking straight ahead. Body Mass Index was calculated using the formula[22]: $weight/height^2$ (kg/m^2) with normal defined as 18-24.9 kg/m^2, overweight as 25-29.9 kg/m^2, and obesity defined as a body mass index (BMI) ≥ 30 kg/m^2.

The waist circumference was measured with a flexible inelastic measure tape calibrated at 0.1cm intervals and measurements taken directly over the skin. The measurement was taken midway between the xiphisternum and pubic symphysis and the circumference measured in a horizontal plane at the end of normal expiration. The hip circumference was also taken around the maximum circumference of the buttocks with subjects standing with their feet together. The Waist to Hip ratio was calculated and recorded (> 0.85 for

females and > 0.9 for males was taken as indicative of truncal obesity).

Radiological evaluation

Magnetic Resonance Imaging (MRI) Technique

The participants had cranial MRI done without prior knowledge of the MMSE results and the degree of cognitive impairment of the participants. The participants changed into examination clothes and an intravenous access line was secured. They were positioned supine on the MRI couch and a radiofrequency coil applied to the cranium. MR imaging was performed using 0.36 T MR (MagSense 360, Mindray) according to a standard dementia MRI protocol.[23] Post contrast T1-Weighted images were also acquired. Images generated were automatically stored on MRI scanner memory and on the local server and intranet facility- Picture Archiving and Computer System (PACS). Furthermore, copies of images were saved on hardware compact discs to serve as additional backup.

The MMSE data as well as the visual assessment scores- Koedam, medial temporal lobe atrophy, global cortical atrophy and Fazekas scores, with the details of strategic infarcts, extracted from each patient's images were appropriately documented in prepared standard study data sheets and analyzed.

Data management and statistical analysis

The data generated was entered and analyzed using the Statistical Package for the Social Sciences (SPSS) software version 20 (SPSS Inc. Chicago, IL, USA.) spread sheet. All data are presented as texts, frequency tables, proportions and percentages, charts and figures. Categorical variables are presented as proportions and percentages while medians and means ± standard deviation were used to present the results of continuous variables (age and anthropometric measurements of the participants).

The independent student's t-test was used to test association between continuous variables at 5% level of significance. Chi square test at 5% level of significance was used to test association of strategic infarcts with severity of cognitive impairment. The presence of strategic infarcts was grouped into right, left or both parieto-temporal and temporo-occipital association areas.

Visual rating scores were analyzed using their median values since these are ordinal data. For statistical analysis, the averaged atrophy scores of the right and left hemisphere for each participant's Koedam scores and MTA scores, as well as the GCA and Fazekas scores were used. The visual rating scores were categorized into high and low scores based on the median score of each visual rating scale. All values below the median score were categorized as low scores while the median score and higher than median scores were categorized as high scores.

The Spearman's Rho test was used to determine the association between the 4 visual rating scales as independent neuroimaging biomarkers of cognitive impairment. The Kruskal-Wallis and Mann-Whitney Wilcoxon tests were used to determine the association of cerebral atrophy and small vessel disease with severity of cognitive impairment. The chi square test (Pearson's chi-square test and Fisher's exact test) was used to determine the association of gender, educational level and vocation with cerebral atrophy. The odds ratio and logistic regression analysis

were used to test for association and significance of confounders. Statistical significance level was defined as $p < 0.05$.

Results

A total of 50 participants were recruited into the study. Tables 1 gives a summary of the socio-demographic and clinical data of the participants. The mean age of the patients was 73.4 ±7.14 years with a minimum age of 62 years and a maximum of 90 years. The participants comprised 27 (54%) males and 23 (46%) females with a male to female ratio of 1.17:1. Married participants constituted about thirty-two (64%) of the study population. Twenty-seven participants (54%) obtained tertiary education and 11 (22%) had primary education. Thirteen (26%) of the participants had at least a vocation while 44 (88%) of the participants were of Yoruba ethnicity. Seven (14%) of the participants had previous mild cognitive impairment. Six (12%) participants had a positive family history of cognitive impairment. A significant history of smoking and alcohol intake was elicited in 7 (14%) participants as shown in Table 1.

Table 1: Socio-demographic characteristics of the study population

Variables	**Frequency**	**Percent**
Sex		
Female	27	54.0
Male	23	46.0
Marital Status		
Married	32	64.0
Others	18	36.0
Educational Level		
Primary	11	22.0
Secondary	12	24.0
Tertiary	27	54.0
Vocation	13	26.0
Ethnicity		
Yoruba	44	88.0
Others	6	12.0
Lives alone	1	2.0
History of previous mild cognitive impairment	7	14.0
Presence of associated co-morbidity	38	76.0
Positive family history of cognitive impairment	6	12.0
Significant smoking/alcohol intake	7	14.0

There was statistically significant difference between the mean height of male participants m = 168.6 ± 7.97 cm and the mean height of female participants m = 155.3 ± 6.74 cm ($p < 0.001$, 95% CI: 9.10; 17.58). Also, the mean weight of male participants m = 68.63 ± 8.63 kg was statistically significantly higher than the mean weight of female participants m = 58.77 ± 5.78 kg ($p < 0.001$, 95% CI: 5.60; 14.12). Likewise, there was statistically significant difference between the mean waist circumference of male participants m = 87.93 ± 9.64 cm and the mean waist circumference of female participants m = 80.96 ± 10.21 cm ($p = 0.017$, 95% CI: 1.32;

12.62). Also, the mean hip circumference of male participants m = 98.74 ± 7.40 cm was statistically significantly higher than the mean hip circumference of female participants m = 92.35 ± 9.93 cm (p = 0.015, 95% CI: 1.31; 11.47). However, there was no statistical significant difference between the means of the age, BMI and waist-hip ratio of male and female participants.

Thirty-eight (76%) participants had at least one associated co-morbidity. The most frequent co-morbidities were hypertension in 33 (66%) participants, DM in 17 (34%) participants, CVD/TIA in 5 (10%) participants, previous head trauma in 2 (4%) participants and previous cranial surgery in 2 (4%) participants.

Averaged right and left scores were used for Koedam and MTA scores because these have paired (right and left) scores. The median average Koedam score of the participants was 1.5 with a maximum score 3 and a minimum score of 0. Also, the median average MTA score of the participants was 2 with a minimum average score of 0.5 and a maximum average score of 4. The median GCA score of the participants was 2 with a minimum score of 0 and a maximum score of 3. The median Fazekas score of the participants was 2 with a minimum score of 1 and a maximum score of 3. MRI images in figure 2 shows GCA 1-3 scores.

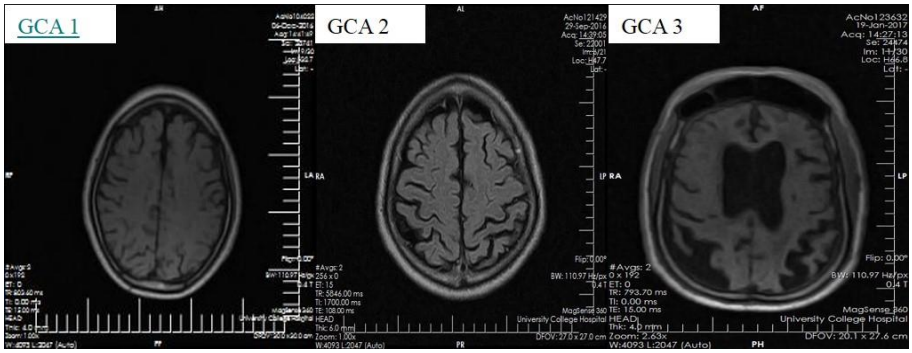

Figure 2a

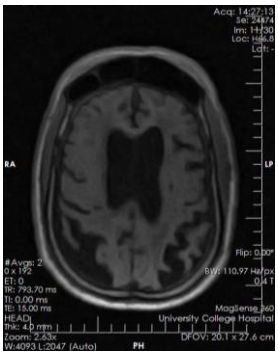

Figure 2b

Figure 2a: Axial T1W MR Images showing GCA scores 1-3.
Figure 2b: Axial T1W MR image showing right parieto-occipital association infarct.

Samples of the images obtained and their respective Koedam, MTA and Fazekas scores as depicted in Figures 3a-3c.

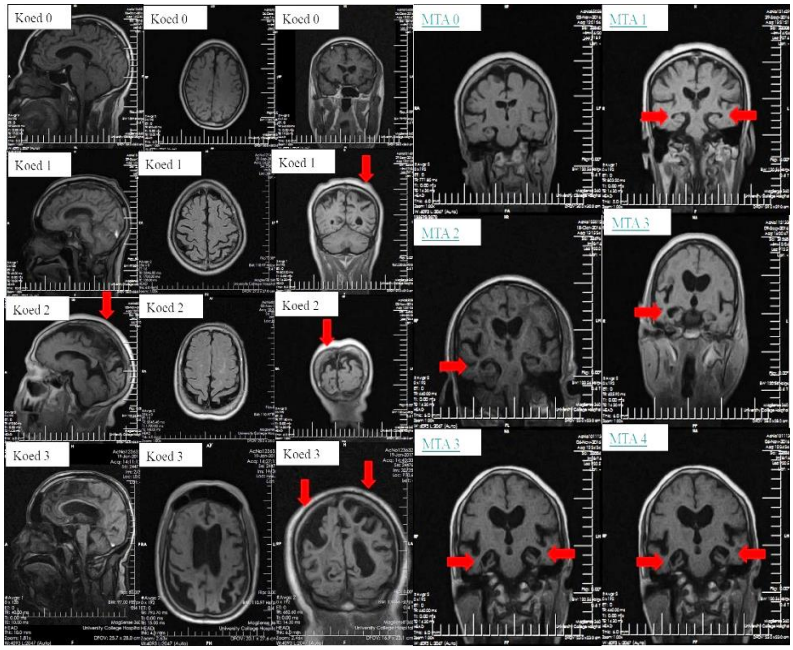

Figure 3a

Figure 3b

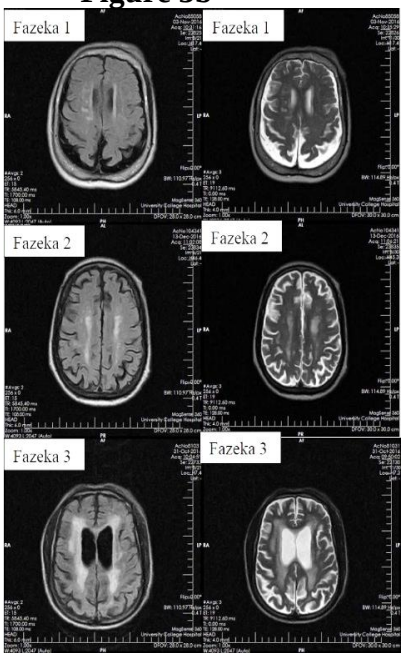

Figure 3c

Figure 3a: Sagittal, axial and coronal MR T1W images respectively showing posterior cerebral atrophy. The arrows show atrophy of the posterior brain regions.
Figure 3b: Coronal T1W MR Images showing MTA scores 0-4. The arrows show medial temporal lobe atrophy.
Figure 3c: Axial FLAIR MR Images showing Fazekas scores 1-3.

Strategic infarcts were detected in 22 (44%) participants with about 77.3% (17/22) located either on the left, right or both sides of the parieto-temporal association areas as shown in Table 2.

Table 2: Distribution of Strategic Infarcts

	Frequency	percent
Strategic Infarcts		
Present	22	44.0
Absent	28	56.0
Location of Strategic Infarcts (22)		
Left PT association area	8	36.4
Right PT association area	1	4.5
Bilateral PT association area	8	36.4
Left TO association area	2	9.1
Right TO association area	2	9.1
Bilateral TO association area	6	27.3

‡‡PT= parieto-temporal ††TO= temporo-occipital

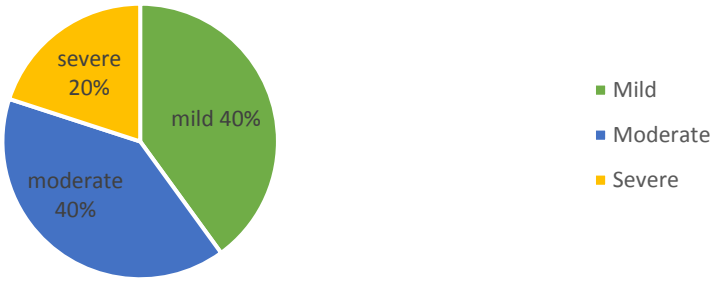

Figure 4: Distribution of MMSE Score in the study population

There was no statistically significant association between age and MMSE score, and sex and MMSE score of participants.

The Spearman's Rho test was used to test for association between the various visual rating scales as independent neuroimaging biomarkers of cognitive impairment. There was a moderate negative correlation between Fazekas score and MMSE score of participants with cognitive impairment ($r = -0.348$, $p < 0.05$). There was a strong positive correlation between average koedam score and GCA score of participants ($r = 0.854$, $p < 0.01$). Also, there was a moderate positive correlation between average MTA score and Fazekas score of participants ($r = 0.338$, $p < 0.05$) as shown in Table 3.

Table 3: Correlation between various visual assessment scales as independent neuroimaging biomarkers of cognitive impairment

		MMSE Score	GCA Score	Fazekas Score	Koedam score	*MTA score*
Spearman's Rho	MMSE Score	1.000				
	Global Cortical Atrophy Score	-0.119	1.000			
	Fazekas Score	-0.348*	0.182	1.000		
	Koed ave	-0.188	0.854**	0.255	1.000	
	MTA ave	-0.225	0.044	0.338*	0.209	1.000

*= $p < 0.05$, ** = $p < 0.01$, Koed ave = average Koedam score, MTA ave = average MTA score)

The association between the visual assessment scores and severity of cognitive impairment was tested with Kruskal-Wallis H test. It showed that there was no significant association between cerebral atrophy and severity of cognitive impairment.

However, there was significant association in the Fazekas score among the different levels of severity in patients with cognitive impairment (X^2 =7.489, p = 0.024) as shown in Table 4.

Table 4: Association between different visual rating scores and severity of cognitive impairment

	N	Mean rank	X^2	df	P value
GCA score					
Mild	20	22.55			
Moderate	20	28.15	1.793	2	0.408
Severe	10	26.10			
Koedam score					
Mild	20	21.85			
Moderate	20	27.53	2.262	2	0.323
Severe	10	28.75			
MTA score					
Mild	20	20.33			
Moderate	20	27.58	4.931	2	0.085
Severe	10	26.30			
Fazekas score					
Mild	20	19.35			
Moderate	20	29.60	7.489	2	0.024*
Severe	10	29.60			

‖*significant

Participants with low MTA score had statistically significantly higher MMSE scores than participants with high MTA score (U = 194.0, P = 0.034) as shown in Table 5.

Table 5: Association between low and high MRI visual rating score and severity of cognitive impairment

	MMSE Score	Mean rank	U	W	Z	p-value
GCA score						
low	**20**	**26.3**				
high	**30**	**25.0**	**285.0**	**750.0**	**-0.300**	**0.764**
Koedam Ave						
low	**20**	**26.8**				
high	**30**	**24.6**	**274.0**	**739.0**	**-0.520**	**0.603**
MTA Ave						
low	**20**	**30.80**				
high	**30**	**21.97**	**194.0**	**659.0**	**-2.122**	**0.034****
Fazekas score						
low	**3**	**38.33**				
high	**47**	**24.68**	**32.000**	**1160.0**	**-1.590**	**0.125***

§* *Fisher's exact* **significant.

Association of socio-demographic and clinical data with cerebral atrophy was tested using the chi square test. Although male participants had higher Koedam scores than female participants, the difference was not significant. Same non-significant difference was observed for marital status, educational level, vocation, ethnicity, positive family history of cognitive impairment.

The independent student's t-test showed no statistical significant association between age, BMI and waist-hip ratio, and average Koedam score of participants. There was a statistical significant association between educational level of participants and participants average MTA scores (X^2= 6.294 p = 0.017). About 69.2% of participants with secondary and below educational level had high average MTA scores while about 27.3% of participants with tertiary education had high average MTA scores.

There was no statistical significant association between age, BMI, waist-hip ratio and the average MTA score of participants.

Multivariate analysis tested for an association between socio-demographic, clinical data and average MTA scores of participants, after adjusting for confounders. Level of education had statistically significant association with average MTA scores of participants. Participants with secondary and below level of education were about 15 times more likely to have high average MTA score than participants who had tertiary education (AOR = 14.6, 95% CI: 1.19; 179.4, P = 0.036) as shown in Table 6.

Table 6: Multivariate analysis of factors associated with participants' average MTA scores

Variables	Adjusted OR	95% confidence interval		p-value
		Lower	upper	
Sex				
Male	1			
Female	0.83	0.19	3.55	0.800
Educational level				
Tertiary	1			
Secondary and below	14.6	1.19	179.4	0.036*
Vocation				
No	1			
Yes	0.49	0.10	2.55	0.387
History of previous mild cognitive impairment				
No	1			
Yes	0.63	0.003	1.41	0.081
Significant smoking/alcohol intake				
No	1			
Yes	6.76	0.31	146.9	0.224

‡*significant

Discussion

Cerebral atrophy can be used as a neuroimaging biomarker in elderly patients with neurocognitive impairment.[20] Several studies have been conducted to determine the association between cerebral atrophy and cognitive impairment. However, these studies show contradictory findings with significant association between cerebral atrophy and cognitive impairment in some and not in others,[24] Cerebral atrophy is one of many factors that affect the cognitive status of individuals.[21,23,36] All the studies agreed that there is significant association of cerebral atrophy with cognitive impairment and increasing age.[24,25,26]

In this study, cerebral atrophy was measured using 3 visual rating scales- Koedam, MTA and GCA while small vessel disease was assessed using the Fazekas scale (white matter hyperintensities) and presence of strategic infarcts. An attempt was made to correlate cerebral atrophy with the cognition status of the participants and the study showed a significant association between MTA scores and severity of cognitive impairment but a non-significant association of Koedam score and GCA score with severity of cognitive impairment. This finding is similar to the findings by Pantano et al. and Shibamoto et al. who also reported no significant association between cerebral atrophy and cognitive impairment.[25,27]

There was statistically significant association between medial temporal lobe atrophy and severity of cognitive impairment and this agrees with the findings of Nihon et al. They reported a statistically significant association of hippocampal atrophy with cognitive impairment using the participant's MTA scores.[26]

Fjell et al. reported a significant correlation between cerebral atrophy and neurocognitive impairment. In their study, an automated method was used to assess cerebral atrophy.[24] Bilello et al. described a significant association between cerebral atrophy and cognitive impairment.[28] In their study, only Alzheimer's disease patients were recruited and the CERAD (Consortium to Establish a Registry for Alzheimer's Disease) neuropsychiatric battery instrument was employed for the study, not the MMSE assessment tool. This may explain the significant association between cerebral atrophy and cognitive impairment in their (Bilello et al.) study.[28]

Harper et al. also reported a statistically significant correlation between visual rating scales (MTA and GCA) and cognitive impairment.[30] This was a longitudinal study with pathologic (autopsy) correlation. A 1.5T MRI machine was used for the study.

Most studies that reported a significant correlation between cerebral atrophy and cognitive impairment used at least a 1.5T machine and larger sample size of at least 101 participants.[24,26,28]

There was a significant association between Fazekas score and severity of cognitive impairment of participants. This is in agreement with the findings of Nihon et al. who reported similar association between Fazekas score and severity of cognitive impairment of participants.[54] This suggests that Fazekas score is useful and relevant in measuring cognitive impairment of patients in this environment. As a measure of small vessel disease, it correlates significantly with the severity of cognitive impairment.

There was no significant association between strategic infarcts and severity of

cognitive impairment. This may be attributable to the tesla strength of the MRI machine used. Most of the infarcts were noted in the left parieto-temporal association area. This is in agreement with the findings of Akinyemi et al. who noted the region as the most commonly affected by brain infarcts.[29] This implies strategic infarcts does not directly correlate to worsening cognitive impairment in this environment.

There was a strong positive correlation of Koedam score with GCA score. There was also a strong positive correlation of average MTA score with Fazekas score. This is in agreement with Pantoni et al. who reported a strong positive correlation among the visual rating scales, especially between koedam and GCA and between MTA and Fazekas.[30] It also agrees with Vasconcellos et al. who reported positive correlation among MTA and Fazekas scores.[31] There were other positive associations among the visual rating scales but these were not significant. This may be attributable to the tesla strength of the MRI machine used and the study sample size.

There was no significant association between age and the visual rating scores, and severity of cognitive impairment (MMSE scores of participants). However, hypothetically if there were more participants older than eighty years of age, the result might have been significant. This agrees with Ge Yulin et al.[32] who reported an association of age with MMSE scores of participants.

Apart from age, other factors such as gender, educational qualification, vocation, positive family history of cognitive impairment, significant smoking, and alcohol intake may influence cognitive impairment.[7]

There was no significant association of gender with cerebral atrophy in this study with respect to the Koedam and MTA scores of participants. Although males had a higher Koedam and MTA scores, these were not statistically significant. This is in agreement with Ge Yulin et al.[32] and Allen et al.[33] They reported no sex prevalence with respect to cerebral atrophy. Bilello et al.[28] also reported no correlation of age and sex of study participants with cerebral atrophy. However, this is at variance with Bromiley et al.[34] who documented that cerebral atrophy is significantly higher and developing faster in men than women. This may be attributable to the fact that Bromiley's study was a longitudinal study in contrast to this cross-sectional descriptive study.

Participants with low MTA scores had statistically significant higher MMSE scores than participants with high MTA scores. This implies that the MTA score is very relevant as a neuroimaging biomarker in this environment. The significant association between participants' average MTA scores and educational level of participants would suggest that individuals with tertiary education were about 15 times more likely to have a low MTA score. This is in agreement with the findings of Ogunniyi et al.[7] who reported low educational attainment as a significant risk factor for neurocognitive impairment. There was no significant association between MTA scores of participants and vocational involvement. However, a trend was noted. The relatively lower educational level and poor vocational inclination of the participants in this study also explains the significant association of MTA scores with

severity of cognitive impairment as higher educational level and being involved in a vocation confers protection against neurocognitive impairment because of continuous brain stimulation.[7]

Conclusion

The significant association between medial temporal lobe atrophy and severity of cognitive impairment suggests medial temporal lobe atrophy is a useful marker of the severity of cognitive impairment. White matter disease is significantly associated with presence and severity of cognitive impairment. This underscores the effect of small vessel disease in neurocognitive impairment. Both medial temporal lobe atrophy and white matter disease can serve as useful neuroimaging biomarkers of neurocognitive impairment.

Recommendation

1) Routine MTA score and Fazekas score, among other visual rating scores, should be included in the neuroimaging work up of patients with neurocognitive impairment.

2) A higher tesla machine (at least 1.5T) will delineate cerebral atrophy and white matter changes better. This is recommended for future studies.

3) There should be research into other neuroimaging biomarkers using structural, functional and metabolic neuroimaging biomarkers.

4) A long term prospective, preferably multicenter study with larger sample size and serial imaging of patients will better test the association between cerebral atrophy and cognitive impairment.

Study limitations

1) The number of patients in the study is relatively smaller, compared with other similar studies, which might have reduced the power of the study to detect associations of cerebral atrophy and small vessel diseases with some of the variables. This is largely due to lack of funds to undertake cranial MRI evaluation and poor attitude of the public to care of the elderly.

2) Some essential data were either not volunteered by the patients because of their cognitive status or were not known to the care givers.

3) A 0.36 tesla MRI machine was used for this study. This might have limited the resolution of details of some cerebral atrophy and small vessel diseases.

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