

Visual rating of GCA and MTA scoring with deep white matter hyperintensities in relation to age and cognitive value

Ruaa Abdulkareem Salman^{1*}, Fedan Ihsan Hassan²

Abstract

Background: The cognitive dysfunction disorders are nowadays represented great health, social, and economic burden globally. Magnetic resonance imaging plays a major role in the evaluation of these disorders. This study aims to assess the relationship between the visual rating scale of global cerebral atrophy (GCA) & medial temporal lobe atrophy (MTA) scoring with age, white matter hyper-intensities, and cognitive value.

Methods: A cross-sectional study carried out from 1st of November 2022 to 28th of February 2023 at the Magnetic Resonance Imaging unit of the Radiology department in Baghdad Teaching Hospital at Medical Complex in Baghdad city, Iraq. Sixty patients aged over 45 years with suspected cognitive abnormalities were included, while younger patients, those with territorial infarction, watershed infarction, or unwillingness to participate were excluded. Data were collected through a semi-structured questionnaire covering sociodemographic factors, chronic illnesses, cognitive status, MRI findings, and visual rating scales (Fazekas, Global Cortical Atrophy [GCA], and Medial Temporal Lobe Atrophy [MTA]). MRI was performed using a 1.5T Philips Achieva Nova scanner, and cognitive assessment was conducted with the Mini-Mental State Examination (MMSE).

Results: The mean age of participants was 62.4 ± 8.8 years, with males slightly predominating (55%). Chronic diseases were common, particularly hypertension (57.2%). Cognitive assessment revealed 51.7% with normal cognition, 18.3% with mild cognitive impairment, and 30% with dementia. Higher Fazekas, GCA, and MTA scores were significantly associated with dementia ($p < 0.05$). MTA scores were significantly elevated in Alzheimer's disease ($p < 0.001$), while vascular etiologies were strongly associated with higher GCA, Fazekas, and MTA scores ($p < 0.05$). Non-strategic lacunar ischemia showed higher Fazekas scores compared to strategic types ($p = 0.006$). Increasing age was significantly linked to dementia, vascular pathology, and higher atrophy scores.

Conclusion: These findings suggest a strong correlation between structural brain changes observed on MRI and cognitive decline, highlighting the importance of visual rating scales in clinical assessment.

Keywords: Magnetic Resonance Imaging, Cognitive Disorders, Visual Rating Scales, White Matter Hyperintensities, Iraq

Correspondence: Ruaa Abdulkareem Salman
(dr.ruaa.salman@gmail.com)

¹Department of Radiology, Baquba Teaching Hospital, Diyala Health Directorate, 32001, Diyala, Iraq.

How to cite: Salman R, Hassan F. Visual rating of GCA and MTA scoring with deep white matter hyper-intensities in relation to age and cognitive value. J Ideas Health. 2025 Aug. 31;8(4):1313-1321
doi: 10.47108/jidhealth.Vol8.Iss4.422

Article Info: (Original Research)

Received: 03 July 2025

Revised: 23 August 2025

Accepted: 27 August 2025

Published: 31 August 2025

© The Author(s). **2025 Open Access** This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made.

The Creative Commons Public Domain Dedication waiver (<https://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article unless otherwise stated.

Journal Home Page: <https://www.jidhealth.com>

e ISSN: 2645-9248

Background

White matter hyperintensities (WMHs) are among the most frequent brain magnetic resonance imaging (MRI) abnormalities in older adults and represent a significant biomarker of cerebral small vessel disease (SVD). They are typically visualized as hyperintense lesions on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences, often distributed in the basal ganglia, periventricular, subcortical, or centrum semiovale regions. Pathologically, WMHs result from demyelination and axonal loss, commonly attributed to ischemic injury of small penetrating arteries [1]. They are a critical imaging feature in the study of cerebrovascular small vessel ischemic disease (SVID) [2], which is an established contributor to cognitive decline and the development of dementia [3]. Volumetric and visual rating assessments of WMHs have advanced our understanding of SVID and its impact on cognitive performance across the spectrum of normal aging and dementia [4]. Strong associations have been reported between WMH burden and deficits in both memory and executive function [5]. In older populations, WMHs are most frequently linked to vascular risk factors such as uncontrolled hypertension, hyperlipidemia, and diabetes mellitus [6]. Although volumetric quantification provides detailed longitudinal data [7,8], semi-quantitative visual rating scales remain widely used in clinical practice due to their feasibility, reproducibility, and correlation with clinical outcomes. Global brain atrophy (GBA), encompassing generalized cortical volume

loss and ventricular enlargement, is another common finding in neuroimaging of the elderly. The interpretation of moderate-to-severe GBA presents a diagnostic challenge for clinicians, as it may be attributed to normal aging, neurodegenerative disorders such as Alzheimer's disease (AD), or cerebrovascular insults [9]. Understanding the interplay between GBA, aging, AD, and cerebrovascular disease (CVD) is crucial for accurate diagnosis and management [7]. GBA has consistently been shown to correlate with cognitive and functional impairment [10], and can be evaluated on MRI or computed tomography (CT) scans using standardized visual rating scales such as the Global Cortical Atrophy (GCA) scale. Potential contributors to GBA include chronological aging, WMHs associated with CVD, and neurodegenerative pathology [7, 11]. While some studies show a direct association between GBA severity and age [12,13], others have found conflicting results [14]. Similarly, medial temporal atrophy (MTA) is a recognized imaging marker for AD, though it may also occur in normal aging [13]. Findings from the Alzheimer's Disease Neuroimaging Initiative (ADNI) indicate that over two years, medial temporal lobe volume may decrease by 1–7%, accompanied by a 10% increase in ventricular volume [15]. Because the annual rate of MTA may be subtle, standardized visual rating scales (e.g., MTA score) help distinguish normal from abnormal atrophy patterns [16,17]. WMHs and GBA frequently coexist in older individuals, and their relationship is of growing research interest. Some studies have shown strong associations between WMH severity and GBA, even in cognitively intact subjects [18,19], while others report no significant link [7,19]. WMHs are also closely related to vascular cognitive impairment and are considered hallmarks of cerebral small vessel disease [20,21]. Their presence increases steeply with age, and higher Fazekas scale scores (≥ 2) have been associated with greater dementia risk [16,17]. Dementia, or major neurocognitive disorder (MND) as defined in DSM-5, is a progressive condition marked by a significant decline in one or more cognitive domains, with resulting impairment in daily functioning [22]. Alzheimer's disease is the most common cause, accounting for up to 70–80% of cases [23], while vascular dementia represents approximately 15% [24]. Mixed dementia, commonly involving both Alzheimer's and vascular pathology, is also prevalent [25]. Neuroimaging plays an essential role in the evaluation of cognitive impairment, both to exclude reversible causes and to identify characteristic atrophy or vascular changes [26]. The differentiation between neurodegenerative and vascular contributions is critical for patient counseling, prognosis, and potential intervention. White matter, which constitutes approximately half of the brain volume, provides essential connectivity between cortical and subcortical regions [27]. Myelin integrity is crucial for efficient neural transmission [28,29], and damage due to ischemia or other insults manifests as WMHs on MRI [29]. The spatial distribution of WMHs may have distinct clinical implications [30,31,32], yet most studies focus on total lesion volume rather than regional patterns. In addition to WMHs and cortical atrophy, lacunar infarctions are frequently observed in older adults, often reflecting hypertensive small vessel disease [33,34,35]. These small subcortical infarcts can contribute to subtle cognitive impairment and, when multiple, may lead to vascular dementia [34,36]. MRI, particularly diffusion-weighted imaging (DWI), improves detection and characterization of such lesions [37,38,39]. Given the frequent coexistence of cortical atrophy, medial temporal

lobe atrophy, and WMHs in the aging brain and their overlapping yet distinct associations with cognitive decline, integrated assessment using visual rating scales may provide a practical and clinically relevant approach. The present study aimed to assess the relationship between visual rating scales of Global Cortical Atrophy (GCA) and Medial Temporal Atrophy (MTA) with age, white matter hyperintensities, and cognitive value.

Methods

Study design and settings

A cross-sectional study was carried out at the Magnetic Resonance Imaging unit of the Radiology department in Baghdad Teaching Hospital at Medical Complex in Baghdad city, Iraq, from 1st November 2022 to 28th February 2023.

Inclusion and exclusion criteria

All patients with suspicion of cognitive abnormality presented to the Magnetic Resonance Imaging unit of the Radiology department who were aged more than 45 years were included. While younger age patients with a history of vascular territory infarction, water shed area (infarction), and those who refused to participate were excluded.

Sampling technique and data collection

A convenient sampling technique was employed to collect data directly from 60 eligible patients. A semi-structured questionnaire was designed by the supervisor to collect the following information from each patient: Sociodemographic characteristics of patients including the age, gender and educational level; History of chronic diseases, types and duration; Cognitive diagnosis of patients which might be normal, mild cognitive impairment or dementia; Visual rating scales of patients including the Fazikas score, global cerebral atrophy (GCA) score and medial temporal lobe atrophy score (MTA); MRI diagnostic findings of patients including the vascular etiology, lacunar ischemia types and Alzheimer disease.

Procedures

The selected patients were referred by neurologists to the Magnetic Resonance Imaging unit of the Radiology department in Baghdad Teaching Hospital for suspicion of cognitive dysfunction. The researcher examined the selected patients for magnetic resonance imaging using MRI equipment (Philips, Germany) and assessed visual rating scales. The cognitive function of selected patients was assessed by using the mini-mental state examination (MMSE) (<https://mmse.neurol.ru/>). A 1.5T MR imaging scanner (Philips Achieva Nova, Dual 16 Channel) was used to perform brain MRI. All patients were scanned in a supine position (lying face-up) using a standard circularly polarized head coil. Standard T1-weighted, T2-weighted, and FLAIR (Fluid-Attenuated Inversion Recovery) sequences were performed in both axial (horizontal) and coronal (vertical) orientations to capture different tissue characteristics and structures of the brain. The parameters are summarized in Table 1. The visual rating scales included medial temporal lobe atrophy (MTA), global cortical atrophy (GCA), and the Fazekas scale, which were assessed by the researcher. Among them, the MTA was scored separately for the left and right sides, and the overall MTA score was obtained by calculating the average of both sides. Regarding the MTA scale, the scores of atrophies

were classified from "0 to 4" in the hippocampus, parahippocampal gyrus, entorhinal cortex, and the surrounding cerebrospinal fluid spaces. The scores were interpreted into grade 0 (no atrophy), grade 1 (widening of the choroid fissure), grade 2 (more widening of the temporal horn), grade 3 (moderate loss of hippocampal volume), and grade 4 (severe loss of hippocampal volume). Regarding the GCA scale, the scores of atrophies were categorized from "0 to 3" in cortex atrophy and sulcal dilatation. They are classified into: grade 0 (no cortical atrophy); grade 1 (mild atrophy), grade 2 (moderate atrophy), and grade 3 (severe atrophy). Regarding Fazekas scale, the scores were arranged from 0 to 3 to changes in white matter. The scores were classified into: grade 0 (no or single punctate lesion), grade 1 (multiple punctate lesions), grade 2 (bridging), and grade 3 (large confluent lesions).

Table 1: MRI sequence parameters.

Parameter	Ti FFE	FLAIR	T2w
TR (ms)	140	6000	1000
TE (ms)	1.63	120	110
Slice	5	5	5
FOV (mm)	210	250	230
Matrix size	510	288	255
Voxel size	0.776	0.9x1.2x5	1x1.2x5
Time (min)	1	1.12	1.14
NSA	2	1	2

TR Repetition Time, TE=Echo time, Ti=Inversion time, FOV=Field of view, NSA=Number of signal emerging, FLAIR=Fluid attenuation inversion recovery. T2w=T2 weighted image

Statistical analysis

Statistical Package for Social Sciences (SPSS) version 22 was used to analyze the data. Descriptive statistics presented as (mean \pm standard deviation) and frequencies as percentages. Multiple contingency tables were conducted, and appropriate statistical tests were performed. Chi-square and Fisher's exact tests were used for categorical variables. An independent sample t-test was used to compare two independent means, and a way ANOVA analysis was used to compare more than two means. Pearson correlation was used to assess the relationship between visual rating scales. In all statistical analyses, the level of significance (p-value) was set at ≤ 0.05 .

Results

Sociodemographic characteristics

This study involved 60 patients with a mean age of 62.4 ± 8.8 years (range: 46–78 years). Nearly half of the participants (43.3%) were younger than 60 years, while 35% were aged 70 years and above. Males were slightly more represented than females (55% vs. 45%). Regarding educational status, secondary school level was the most common (35%), followed by primary (31.7%), while only 10% had attained college or institute education (Table 2).

History of chronic diseases

A positive history of chronic diseases was reported in 81.7% of patients, with hypertension being the most frequent condition (57.2%), followed by combined hypertension and diabetes (36.7%). The mean duration of chronic illness was 11.2 ± 6.6

years, and most patients (73.5%) had been affected for 10 years or longer (Table 3).

Table 2: General characteristics of patients (N=60)

Variable	Categories	N (%)
Age (mean \pm SD)	62.4 \pm 8.8 years	
Age range	<60 years	26 (43.3)
	60-69 years	13(21.7)
	≥ 70 years	21(35.0)
Gender	Male	33 (55.0)
	Female	27(45.0)
Educational level	Illiterate	14(23.3)
	Primary	19(31.7)
	Secondary	21(35.0)
	College/institute	6(10.0)

Table 3: History of chronic diseases of patients.

Variable	Categories	N (%)
Chronic diseases	Positive	49(81.7)
	Negative	11(18.3)
Type of chronic diseases	HT	28(57.2)
	DM	3(6.1)
	HT & DM	18(36.7)
Duration of chronic disease; mean \pm SD (11.2 \pm 6.6 years)	<10 years	13(26.5)
	≥ 10 years	36(73.5)

Cognitive status and visual rating scales

More than half of the patients (51.7%) had normal cognition, while 18.3% showed mild cognitive impairment and 30% had dementia. The most frequent Fazekas score was grade I (38.3%), followed by grade II (31.7%). The mean GCA score was 15.5 ± 10.7 , with mild atrophy observed in 38.3% of patients and moderate atrophy in 28.3%. Regarding MTA, the most common finding was score 0 (36.7%), followed by score II (31.7%) (Table 4).

Table 4: Cognitive diagnosis and visual rating scales of patients

Variable	Categories	N (%)
Cognitive diagnosis	Normal	31(51.7)
	MCI	11(18.3)
	Dementia	18(30.0)
Fazikas score	0	7(11.7)
	I	23(38.3)
	II	19(31.7)
	III	11(18.3)
GCA score: mean \pm SD	No atrophy	10(16.7)
	Mild atrophy	23(38.3)
	Moderate atrophy	17(28.3)
	Severe atrophy	10(16.7)
MTA score	0	(36.7)
	I	(23.3)
	II	(31.7)
	III	(8.3)
	IV	-

Diagnostic findings

More than half of the patients (56.7%) showed no vascular etiology. Among those with vascular changes, 11.7% had état criblé, and 31.6% presented with lacunar ischemia. Lacunar ischemia was more often strategic (63.2%) than non-strategic (36.8%). In addition, Alzheimer's disease was diagnosed in 8.3% of patients (Table 5).

Age-related distribution of cognitive and imaging findings.

Dementia was significantly more common in older patients, particularly those aged ≥ 70 years ($P=0.003$). Higher Fazekas scores were also associated with increasing age ($P=0.03$). Similarly, advanced GCA scores with severe atrophy were predominantly observed in older age groups ($p=0.002$).

MTA scores followed the same pattern, with higher grades significantly linked to advanced age ($P=0.003$) (Table 6).

Table 5: Diagnostic findings of patients.

Variable	Categories	N (%)
Vascular etiology	No	34(56.7)
	État criblé	7(11.7)
	Lacunar ischemia	19(31.6)
Lacunar ischemia	Strategic	12(63.2)
	Non-strategic	7(36.8)
Alzheimer disease	Positive	5(8.3)
	Negative	55(91.7)

Table 6: Distribution of cognitive diagnosis and visual rating scales according to age groups.

Variable	Categories	<60 years N (%)	60-69 years N (%)	≥ 70 years N (%)	P-value
Cognitive diagnosis	Normal	19(73.1)	8(61.5)	4(19.0)	0.003* ^S
	MCI	3(11.5)	3(23.1)	5(23.8)	
	Dementia	4(15.4)	2(15.4)	12(57.1)	
Fazikas score	0	7(26.9)	-	-	0.03*
	I	10(38.5)	4(30.8)	9(42.9)	
	II	5(19.2)	7(53.8)	7(33.3)	
	III	4(15.4)	2(15.4)	5(23.8)	
GCA score	No atrophy	7(26.9)	3(23.1)	-	0.002*
	Mild atrophy	15(57.7)	2(15.4)	6(28.6)	
	Moderate atrophy	2(7.7)	6(46.2)	9(42.9)	
	Severe atrophy	2(7.7)	2(15.4)	6(28.6)	
MTA score	0	16(61.5)	5(38.5)	1(4.8)	0.003*
	I	4(15.4)	4(30.8)	6(28.6)	
	II	6(23.1)	2(15.4)	11(52.4)	
	III	-	2(15.4)	3(14.3)	
	IV	-	-	-	

* Fisher's exact test

Age-related distribution of diagnostic findings

Lacunar ischemia showed a highly significant association with older age, being more frequent among patients aged ≥ 70 years ($P=0.03$). Similarly, strategic lacunar ischemia was significantly

more prevalent in older patients compared to younger groups ($P=0.03$). In contrast, no significant difference was found in the distribution of Alzheimer's disease across age groups ($P=0.3$) (Table 7).

Table 7: Distribution of diagnostic findings according to age groups.

Variable	Categories	<60 years N (%)	60-69 years N (%)	≥ 70 years N (%)	P-value
Vascular etiology	No	19(73.1)	8(61.5)	7(33.3)	0.03*
	État criblé	2(7.7)	-	5(23.8)	
	Lacunar ischemia	5(19.2)	5(38.5)	9(42.9)	
Lacunar ischemia	Strategic	3(60.0)	1(20.0)	8(88.9)	0.03*
	Non-strategic	2(40.0)	4(80.0)	1(11.1)	
Alzheimer disease	Positive	2(7.7)	-	3(14.3)	0.3
	Negative	24(92.3)	13(100.0)	18(85.7)	

Association of visual rating scales with cognitive status

Patients with dementia demonstrated significantly higher mean GCA, Fazikas, and MTA scores compared to those with normal

cognition or mild cognitive impairment ($p \leq 0.05$). These findings indicate a strong association between worsening cognitive status and increased structural brain changes (Table 8).

Table 8: Distribution of visual rating scales according to cognitive diagnosis.

Variable	Normal Mean \pm SD	MCI Mean \pm SD	Dementia Mean \pm SD	P-value
GCA score	9.5 \pm 7.1	18.2 \pm 12.3	24.6 \pm 7.2	<0.001*
Fazicas score	1.1 \pm 0.8	1.7 \pm 0.6	2.1 \pm 0.9	0.002*
MTA score	0.5 \pm 0.3	1 \pm 0.8	2.1 \pm 0.6	<0.001*

* One-way ANOVA analysis

Association of visual rating scales with Alzheimer s-positive and -Negative Patients

Table 9 shows that among visual rating scales, only the MTA

The score was significantly higher in Alzheimer s-positive patients compared to negatives (2.6 ± 0.5 vs 0.98 ± 0.93 , $P < 0.001$), while GCA and Fazicas scores showed no significant differences.

Table 9: Distribution of visual rating scales according to the presence of Alzheimer's disease.

Variable	Positive Mean \pm SD	Negative Mean \pm SD	P-value
GCA score	22 \pm 8.5	15.1 \pm 10.6	0.1*
Fazicas score	1.2 \pm 0.4	1.6 \pm 0.9	0.3*
MTA score	2.6 \pm 0.5	0.98 \pm 0.93	<0.001*

* Independent sample t-test

Association of visual rating scales with vascular etiology.

Patients with vascular etiologies showed significantly higher mean scores across all visual rating scales. Both GCA and Fazicas scores were markedly elevated in those with état criblé

and lacunar ischemia compared to patients without vascular changes ($P < 0.001$). Similarly, the mean MTA score was also significantly higher among patients with vascular pathology ($P = 0.02$) (Table 10).

Table 10: Distribution of visual rating scales according to the presence of vascular etiology

Variable	No Mean \pm SD	État criblé Mean \pm SD	Lacunar Mean \pm SD	P-value
GCA score	11.2 \pm 10	28 \pm 3.9	18.6 \pm 8.4	<0.001*
Fazicas score	1.1 \pm 0.8	2.8 \pm 0.4	1.9 \pm 0.6	<0.001*
MTA score	0.8 \pm 0.7	2 \pm 0.5	1.2 \pm 0.7	0.02*

* One-way ANOVA analysis

Association of visual rating scales with types of lacunar ischemia

Table 11 shows the distribution of visual rating scale scores according to lacunar ischemia type. The mean of Fazicas' score was significantly increased in patients with non-strategic lacunar

ischemia ($P = 0.006$), while the means of GCA and MTA scores were not significantly different in regard to types of lacunar ischemia ($P > 0.05$).

Table 11: Distribution of visual rating scales according to types of lacunar ischemia.

Variable	Strategic Mean \pm SD	Non-strategic Mean \pm SD	P-value
GCA score	18.8 \pm 9.4	18.2 \pm 7.3	0.9*
Fazicas score	1.6 \pm 0.5	2.4 \pm 0.5	0.006*
MTA score	1.3 \pm 0.6	1.1 \pm 0.9	0.5*

* One-way ANOVA analysis

Discussion

Neurodegenerative diseases represent nowadays a great health, social, and economic challenge facing health institutes [40,41]. Magnetic resonance imaging (MRI) is helpful in the recognition of cerebral microvascular [42] in addition to lesion predictors of microvascular integrity [43]. The current study showed a significant association between dementia and older age groups of patients ($p = 0.003$). This finding is consistent with much of the literature, such as El-Metwally et al. [44] and Wolters et al. [45], who reported that the prevalence of dementia is higher in the older age population. Stephan et al. [46] study stated that, in addition to the effect of chronological age on dementia incidence,

feeling older is associated with the risk of incident dementia. In our study, a significant association was observed between increased Fazicas score and older age patients ($P = 0.03$). This finding coincides with the results of Muñoz Maniega et al. [47] study, which revealed that white matter integrity changes (Fazicas score) were associated with older age of patients. The white matter changes are progressive and highly prevalent in the elderly. It was shown that highly extensive white matter changes are associated with poor clinical outcomes. However, the exact mechanisms of these changes are still uncertain, although they are related to older age, small vessel disease, and other vascular risk factors [48]. Our study found that advanced GCA score and

severe atrophy were significantly prevalent in older age patients ($P=0.002$). This finding is similar to the results of Al-Janabi et al. [49] study, which showed that the GCA score of patients was increased with an increase in their age, and the moderate to severe global cerebral atrophy was more prominent in older age patients. A study by Blinkouskaya and Weickenmeier stated that healthy and pathological brain aging are manifested with different degrees of cognitive decline, which are all associated with morphological changes known as cerebral atrophy. Cortical thinning, white and gray matter changes, ventricular enlargement, and loss of gyrfication are the main morphological changes that are related to aging; however, the mechanism of brain aging is still vague [50]. In our study, a significant association was observed between increased MTA score and older age patients ($P=0.003$). This finding is parallel to the results of Claus et al. [51] study, which revealed that the MTA score was significantly increased in the advanced age population, and the cutoff value of MTA was limited at the age of 85 years. Pereira et al. [17] study found that MTA was commonly affected by elderly age, earlier disease, and genetic factors. The present study showed a highly significant association between lacunar ischemia and older age patients ($P=0.03$). Similarly, Arboix et al. [52] reported that lacunar infarcts are predominant in the elderly population. Inconsistently, Cai et al. [53] study reported that the lacunar infarct prevalence was decreasing with aging among the elderly population, while the prevalence of lacunar infarct increases with aging among the young and adult age population. Our study found a significant association between strategic lacunar ischemia and older age patients ($P=0.03$). This finding is consistent with the results of Yu et al.'s study [54], which revealed that the strategic lacunae infarct incidence was increased among elderly patients, especially among diabetic patients, and was accompanied by white matter changes. Our study found no significant differences were observed between patients with different age groups regarding Alzheimer's disease ($P=0.3$). This finding is inconsistent with the results of Farfel et al. [55] study, which reported that Alzheimer's disease onset increased with advancing age and reached the peak at the age of 95 years. This inconsistency might be attributed to the fact that the onset of Alzheimer's disease starts at the age of 65 years, and a low proportion of enrolled patients in our study were 65 years of age or older. In the present study, a strong and significant correlation was observed between the Fazikas score and the GCA score of patients ($r=0.76$, $P<0.001$). This finding is similar to the results of Kaushik et al.'s [56] study. Our study found a strong and significant correlation between MTA score and GCA score of patients ($r=0.8$, $p<0.001$). This finding is consistent with the results of Mårtensson et al. [57] study. Our study showed a strong and significant correlation between MTA score and Fazikas score of patients ($r=0.8$, $P<0.001$). This finding is parallel to the results of Molinder et al.'s [58] study. In the current study, the means of GCA, Fazikas, and MTA scores were significantly increased in patients with dementia ($P\leq 0.05$). These findings are in agreement with the results of Wahlund et al. [59] study and Agrawal et al. [60] study, which all documented that visual rating scales of MRI were increased with cognitive decline, reaching a maximum with dementia. Our study found that the mean of the MTA score was significantly increased in patients with Alzheimer's disease ($p=0.002$). Consistently, Visser et al. [61] study stated that a high risk of Alzheimer's disease was prevalent with increasing age and

related to medial temporal lobe atrophy. In our study, the means of GCA, Fazikas, and MTA scores were significantly increased in patients with État criblé ($p\leq 0.05$). These findings are in agreement with the results of Kaltoft et al. [62] study, which reported that the visual are highly increased in patients with État criblé. Our study showed that the mean of Fazikas score was significantly increased in patients with non-strategic lacunar ischemia ($p=0.006$). This finding is parallel to the results of Benjamin et al. [63] study, which reported that white matter changes are commonly related to lacunar infarcts with more prevalence in non-strategic type. This study was limited by its small sample size and single-center design, which may restrict the generalizability of the findings. The cross-sectional nature precluded causal inference. Furthermore, reliance on convenient sampling and lack of biomarker validation (such as CSF or PET imaging) may limit diagnostic accuracy for Alzheimer's disease.

Conclusion

The magnetic resonance imaging visual rating scales (global cerebral atrophy, medial temporal atrophy, and white matter hyper-intensity scores) are increased with advancing age. The cognitive function declines with the elderly age population. The global cerebral atrophy, medial temporal atrophy, and white matter hyper-intensity scores are increased with the decline of cognitive function. The incidence of lacunar ischemia, especially the strategic type, is increased with elderly age. The medial temporal atrophy score is predictive of Alzheimer's disease. The global cerebral atrophy, medial temporal atrophy, and white matter hyper-intensity scores are increased with État criblé. The white matter hyperintensity is related to non-strategic lacunar ischemia. Encouraging neurologists to refer older age patients for magnetic resonance imaging to assess the white matter changes and predict dementia and Alzheimer's disease. The lacunar ischemia should be taken in consideration during the assessment of cognitive function. Further national multi-centers studies on the role of magnetic resonance imaging and visual rating scales in the assessment of cognitive function.

Abbreviation

GCA: Global Cerebral Atrophy; MTA: Medial Temporal Lobe Atrophy; MMSE: Mini-Mental State Examination; WMHs: White Matter Hyperintensities; FLAIR: Fluid-Attenuated Inversion Recovery; SVID: Small Vessel Ischemic Disease; AD: Alzheimer's Disease; CVD: Cardiovascular Disease; CT: Computed Tomography; ADNI: Alzheimer's Disease Neuroimaging Initiative; MRI: Magnetic Resonance Imaging; MND: Major Neurocognitive Disorder; DWI: Diffusion-Weighted Imaging; CSF: Cerebrospinal Fluid; PET: Positron Emission Tomography.

Declaration

Acknowledgment

None.

Funding

None.

Availability of data and materials

Data will be available by emailing dr.ruaa.salman@gmail.com.

Authors' contributions

Ruaa Abdulkareem Salman (RAS) conceived and designed the analysis; collected the data; contributed data or analysis tools; performed the analysis; and wrote the manuscript. Fedan Ihsan Hassan (FIH) conceived and designed the analysis; collected the data; contributed data or analysis tools; and wrote the manuscript. All Authors approved the final version to be published, agreeing to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All authors read and approved the final manuscript.

Ethics approval and consent to participate

We conducted the research following the Declaration of Helsinki. The protocol was approved by the Arab Board of Health Specializations. An agreement was taken from hospital authorities, Iraq A, signed, and an oral informed consent was taken from patients enrolled in the study.

Consent for publication

Not applicable

Competing interest

The authors declare that they have no competing interests.

Open Access

This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article unless otherwise stated.

Author Details

¹Department of Radiology, Baquba Teaching Hospital, Diyala Health Directorate, 32001, Diyala, Iraq

²Department of Radiology Specialists, Baghdad Medical City Complex, Baghdad Teaching Hospital, Baghdad, Iraq

References

- Qi X, Tang H, Luo Q, Ding B, Chen J, Cui P, Chen S, Ling H, Ma J. White Matter Hyperintensities Predict Cognitive Decline: A Community-Based Study. *Can J Neurol Sci*. 2019 Jul;46(4):383-388. doi: 10.1017/cjn.2019.47.
- Wang Y, Yang Y, Wang T, Nie S, Yin H, Liu J. Correlation between White Matter Hyperintensities Related Gray Matter Volume and Cognition in Cerebral Small Vessel Disease. *J Stroke Cerebrovasc Dis*. 2020 Dec;29(12):105275. doi: 10.1016/j.jstrokecerebrovasdis.2020.105275.
- Brugulat-Serrat A, Salvadó G, Operto G, Cacciaglia R, Sudre CH, Grau-Rivera O, et al; ALFA Study. White matter hyperintensities mediate gray matter volume and processing speed relationship in cognitively unimpaired participants. *Hum Brain Mapp*. 2020 Apr 1;41(5):1309-1322. doi: 10.1002/hbm.24877.
- d'Arbeloff T, Elliott ML, Knodt AR, Melzer TR, Keenan R, Ireland D, et al. White matter hyperintensities are common in midlife and already associated with cognitive decline. *Brain Commun*. 2019;1(1):fcz041. doi: 10.1093/braincomms/fcz041.
- Valdés Hernández MDC, Chappell FM, Muñoz Maniega S, Dickie DA, Royle NA, Morris Z, et al. Metric to quantify white matter damage on brain magnetic resonance images. *Neuroradiology*. 2017 Oct;59(10):951-962. doi: 10.1007/s00234-017-1892-1.
- Murray ME, Vemuri P, Preboske GM, Murphy MC, Schweitzer KJ, Parisi JE, Jack CR Jr, Dickson DW. A quantitative postmortem MRI design sensitive to white matter hyperintensity differences and their relationship with underlying pathology. *J Neuropathol Exp Neurol*. 2012 Dec;71(12):1113-22. doi: 10.1097/NEN.0b013e318277387e.
- Al-Janabi OM, Bauer CE, Goldstein LB, Murphy RR, Bahrani AA, Smith CD, Wilcock DM, Gold BT, Jicha GA. White Matter Hyperintensity Regression: Comparison of Brain Atrophy and Cognitive Profiles with Progression and Stable Groups. *Brain Sci*. 2019 Jul 19;9(7):170. doi: 10.3390/brainsci9070170.
- Ramirez J, McNeely AA, Berezuk C, Gao F, Black SE. Dynamic Progression of White Matter Hyperintensities in Alzheimer's Disease and Normal Aging: Results from the Sunnybrook Dementia Study. *Front Aging Neurosci*. 2016 Mar 24;8:62. doi: 10.3389/fnagi.2016.00062.
- Appelman AP, Exalto LG, van der Graaf Y, Biessels GJ, Mali WP, Geerlings MI. White matter lesions and brain atrophy: more than shared risk factors? A systematic review. *Cerebrovasc Dis*. 2009;28(3):227-42. doi: 10.1159/000226774.
- Schmidt R, Ropele S, Enzinger C, Petrovic K, Smith S, Schmidt H, Matthews PM, Fazekas F. White matter lesion progression, brain atrophy, and cognitive decline: the Austrian stroke prevention study. *Ann Neurol*. 2005 Oct;58(4):610-6. doi: 10.1002/ana.20630.
- Faldu H, Surana D, Patel C. Clinical spectrum of demyelinating disease of central nervous system and frequency of anti AQP4 and anti MOG among them: one-year single-center retrospective study. *J Ideas Health*. 2024 Aug. 31 ;7(4):1100-5. DOI: 10.47108/jidhealth.Vol7.Iss4.353
- Gattringer T, Enzinger C, Ropele S, Gorani F, Petrovic KE, Schmidt R, Fazekas F. Vascular risk factors, white matter hyperintensities and hippocampal volume in normal elderly individuals. *Dement Geriatr Cogn Disord*. 2012;33(1):29-34. doi: 10.1159/000336052.
- Park M, Moon WJ. Structural MR Imaging in the Diagnosis of Alzheimer's Disease and Other Neurodegenerative Dementia: Current Imaging Approach and Future Perspectives. *Korean J Radiol*. 2016 Nov-Dec;17(6):827-845. doi: 10.3348/kjr.2016.17.6.827.
- Resnick SM, Goldszal AF, Davatzikos C, Golski S, Kraut MA, Metter EJ, Bryan RN, Zonderman AB. One-year age changes in MRI brain volumes in older adults. *Cereb Cortex*. 2000 May;10(5):464-72. doi: 10.1093/cercor/10.5.464.
- Hua X, Ching CRK, Mezher A, Gutman BA, Hibar DP, Bhatt P, Leow AD, Jack CR Jr, Bernstein MA, Weiner MW, Thompson PM; Alzheimer's Disease Neuroimaging Initiative. MRI-based brain atrophy rates in ADNI phase 2: acceleration and enrichment considerations for clinical trials. *Neurobiol Aging*. 2016 Jan;37:26-37. doi: 10.1016/j.neurobiolaging.2015.09.018.
- Vernooij MW, van Buchem MA. Neuroimaging in Dementia. 2020 Feb 15. In: Hodler J, Kubik-Huch RA, von Schulthess GK, editors. *Diseases of the Brain, Head and Neck, Spine 2020–2023: Diagnostic Imaging* [Internet]. Cham (CH): Springer; 2020. Chapter 11. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK554327/>
- Pereira JB, Cavallin L, Spulber G, Aguilar C, Mecocci P, Vellas B, Tsolaki M, Kłoszewska I, Soininen H, Spenger C, Aarsland D, Lovestone S, Simmons A, Wahlund LO, Westman E;

- AddNeuroMed consortium and for the Alzheimer's Disease Neuroimaging Initiative. Influence of age, disease onset and ApoE4 on visual medial temporal lobe atrophy cut-offs. *J Intern Med*. 2014 Mar;275(3):317-30. doi: 10.1111/joim.12148.
18. Aribisala BS, Valdés Hernández MC, Royle NA, Morris Z, Muñoz Maniega S, Bastin ME, Deary IJ, Wardlaw JM. Brain atrophy associations with white matter lesions in the ageing brain: the Lothian Birth Cohort 1936. *Eur Radiol*. 2013 Apr;23(4):1084-92. doi: 10.1007/s00330-012-2677-x.
 19. Zheng L, Vinters HV, Mack WJ, Weiner MW, Chui HC; IVD program project. Differential effects of ischemic vascular disease and Alzheimer's disease on brain atrophy and cognition. *J Cereb Blood Flow Metab*. 2016 Jan;36(1):204-15. doi: 10.1038/jcbfm.2015.152.
 20. Prins ND, Scheltens P. White matter hyperintensities, cognitive impairment and dementia: an update. *Nat Rev Neurol*. 2015 Mar;11(3):157-65. doi: 10.1038/nrneurol.2015.10.
 21. Østergaard L, Engedal TS, Moreton F, Hansen MB, Wardlaw JM, Dalkara T, Markus HS, Muir KW. Cerebral small vessel disease: Capillary pathways to stroke and cognitive decline. *J Cereb Blood Flow Metab*. 2016 Feb;36(2):302-25. doi: 10.1177/0271678X15606723.
 22. Sachdev PS, Blacker D, Blazer DG, Ganguli M, Jeste DV, Paulsen JS, Petersen RC. Classifying neurocognitive disorders: the DSM-5 approach. *Nat Rev Neurol*. 2014 Nov;10(11):634-42. doi: 10.1038/nrneurol.2014.181.
 23. Reitz C, Mayeux R. Alzheimer disease: epidemiology, diagnostic criteria, risk factors and biomarkers. *Biochem Pharmacol*. 2014 Apr 15;88(4):640-51. doi: 10.1016/j.bcp.2013.12.024.
 24. O'Brien JT, Thomas A. Vascular dementia. *Lancet*. 2015 Oct 24;386(10004):1698-706. doi: 10.1016/S0140-6736(15)00463-8.
 25. Custodio N, Montesinos R, Lira D, Herrera-Pérez E, Bardales Y, Valeriano-Lorenzo L. Mixed dementia: A review of the evidence. *Dement Neuropsychol*. 2017 Oct-Dec;11(4):364-370. doi: 10.1590/1980-57642016dn11-040005.
 26. McKhann GM, Knopman DS, Chertkow H, Hyman BT, Jack CR Jr, Kawas CH, et al. The diagnosis of dementia due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimers Dement*. 2011 May;7(3):263-9. doi: 10.1016/j.jalz.2011.03.005.
 27. Filley CM, Fields RD. White matter and cognition: making the connection. *J Neurophysiol*. 2016 Nov 1;116(5):2093-2104. doi: 10.1152/jn.00221.2016.
 28. Muzio MR, Cascella M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 19, 2021. Histology, Axon.
 29. Nave KA, Werner HB. Myelination of the nervous system: mechanisms and functions. *Annu Rev Cell Dev Biol*. 2014;30:503-33. doi: 10.1146/annurev-cellbio-100913-013101.
 30. Habes M, Sotiras A, Erus G, Toledo JB, Janowitz D, Wolk DA, et al. White matter lesions: Spatial heterogeneity, links to risk factors, cognition, genetics, and atrophy. *Neurology*. 2018 Sep 4;91(10):e964-e975. doi: 10.1212/WNL.00000000000006116.
 31. Smith CD, Johnson ES, Van Eldik LJ, Jicha GA, Schmitt FA, Nelson PT, et al. Peripheral (deep) but not periventricular MRI white matter hyperintensities are increased in clinical vascular dementia compared to Alzheimer's disease. *Brain Behav*. 2016 Feb 16;6(3):e00438. doi: 10.1002/brb3.438.
 32. Sotiras A, Resnick SM, Davatzikos C. Finding imaging patterns of structural covariance via Non-Negative Matrix Factorization. *Neuroimage*. 2015 Mar;108:1-16. doi: 10.1016/j.neuroimage.2014.11.045.
 33. Blanco-Rojas L, Arboix A, Canovas D, Grau-Olivares M, Oliva Morera JC, Parra O. Cognitive profile in patients with a first-ever lacunar infarct with and without silent lacunes: a comparative study. *BMC Neurol*. 2013 Dec 16;13:203. doi: 10.1186/1471-2377-13-203.
 34. Wardlaw JM, Smith C, Dichgans M. Small vessel disease: mechanisms and clinical implications. *Lancet Neurol*. 2019 Jul;18(7):684-696. doi: 10.1016/S1474-4422(19)30079-1.
 35. Nelson RF, Pullicino P, Kendall BE, Marshall J. Computed tomography in patients presenting with lacunar syndromes. *Stroke*. 1980 May-Jun;11(3):256-61. doi: 10.1161/01.str.11.3.256.
 36. Noguchi K, Nagayoshi T, Watanabe N, Kanazawa T, Toyoshima S, Morijiri M, Shojaku H, Shimizu M, Seto H. Diffusion-weighted echo-planar MRI of lacunar infarcts. *Neuroradiology*. 1998 Jul;40(7):448-51. doi: 10.1007/s002340050621.
 37. Schonewille WJ, Tuhim S, Singer MB, Atlas SW. Diffusion-weighted MRI in acute lacunar syndromes. A clinical-radiological correlation study. *Stroke*. 1999 Oct;30(10):2066-9. doi: 10.1161/01.str.30.10.2066.
 38. Tan MY, Singhal S, Ma H, Chandra RV, Cheong J, Clissold BB, Ly J, Srikanth V, Phan TG. Examining Subcortical Infarcts in the Era of Acute Multimodality CT Imaging. *Front Neurol*. 2016 Dec 5;7:220. doi: 10.3389/fneur.2016.00220.
 39. Zhao L, Biesbroek JM, Shi L, Liu W, Kuijff HJ, Chu WW, Abrigo JM, Lee RK, Leung TW, Lau AY, Biessels GJ, Mok V, Wong A. Strategic infarct location for post-stroke cognitive impairment: A multivariate lesion-symptom mapping study. *J Cereb Blood Flow Metab*. 2018 Aug;38(8):1299-1311. doi: 10.1177/0271678X17728162.
 40. El-Hayek YH, Wiley RE, Khoury CP, Daya RP, Ballard C, Evans AR, Karran M, Molinuevo JL, Norton M, Atri A. Tip of the Iceberg: Assessing the Global Socioeconomic Costs of Alzheimer's Disease and Related Dementias and Strategic Implications for Stakeholders. *J Alzheimers Dis*. 2019;70(2):323-341. doi: 10.3233/JAD-190426.
 41. Han EJ, Lee J, Cho E, Kim H. Socioeconomic Costs of Dementia Based on Utilization of Health Care and Long-Term-Care Services: A Retrospective Cohort Study. *Int J Environ Res Public Health*. 2021 Jan 6;18(2):376. doi: 10.3390/ijerph18020376.
 42. Doulal FN, MacLulich AM, Ferguson KJ, Dennis MS, Wardlaw JM. Enlarged perivascular spaces on MRI are a feature of cerebral small vessel disease. *Stroke*. 2010 Mar;41(3):450-4. doi: 10.1161/STROKEAHA.
 43. Zdanovskis N, Platkājis A, Kostiks A, Šneidere K, Stepens A, Naglis R, Karelis G. Combined Score of Perivascular Space Dilatation and White Matter Hyperintensities in Patients with Normal Cognition, Mild Cognitive Impairment, and Dementia. *Medicina (Kaunas)*. 2022 Jul 1;58(7):887. doi: 10.3390/medicina58070887.
 44. El-Metwally A, Toivola P, Al-Rashidi M, Nooruddin S, Jawed M, AlKanhal R, Razzak HA, Albawardi N. Epidemiology of Alzheimer's Disease and Dementia in Arab Countries: A Systematic Review. *Behav Neurol*. 2019 Oct 29;2019:3935943. doi: 10.1155/2019/3935943.
 45. Wolters FJ, Chibnik LB, Waziry R, Anderson R, Berr C, Beiser A, et al. Twenty-seven-year time trends in dementia incidence in Europe and the United States: The Alzheimer Cohorts Consortium. *Neurology*. 2020 Aug 4;95(5):e519-e531. doi: 10.1212/WNL.0000000000010022.

46. Stephan Y, Sutin AR, Luchetti M, Terracciano A. Subjective age and risk of incident dementia: Evidence from the National Health and Aging Trends survey. *J Psychiatr Res.* 2018 May;100:1-4. doi: 10.1016/j.jpsychires.2018.02.008.
47. Muñoz Maniega S, Chappell FM, Valdés Hernández MC, Armitage PA, Makin SD, Heye AK, Thrippleton MJ, Sakka E, Shuler K, Dennis MS, Wardlaw JM. Integrity of normal-appearing white matter: Influence of age, visible lesion burden and hypertension in patients with small-vessel disease. *J Cereb Blood Flow Metab.* 2017 Feb;37(2):644-656. doi: 10.1177/0271678X16635657.
48. Xiong YY, Mok V. Age-related white matter changes. *J Aging Res* 2011; 2011:617927.
49. Al-Janabi OM, Panuganti P, Abner EL, Bahrani AA, Murphy R, Bardach SH, Caban-Holt A, Nelson PT, Gold BT, Smith CD, Wilcock DM, Jicha GA. Global Cerebral Atrophy Detected by Routine Imaging: Relationship with Age, Hippocampal Atrophy, and White Matter Hyperintensities. *J Neuroimaging.* 2018 May;28(3):301-306. doi: 10.1111/jon.
50. Blinkouskaya Y, Weickenmeier J. Brain Shape Changes Associated With Cerebral Atrophy in Healthy Aging and Alzheimer's Disease. *Front Mech Eng* 2021; 7:705653.
51. Claus JJ, Staekenborg SS, Holl DC, Roorda JJ, Schuur J, Koster P, Tielkes CEM, Scheltens P. Practical use of visual medial temporal lobe atrophy cut-off scores in Alzheimer's disease: Validation in a large memory clinic population. *Eur Radiol.* 2017 Aug;27(8):3147-3155. doi: 10.1007/s00330-016-4726-3.
52. Arboix A, García-Eroles L, Massons J, Oliveres M, Targa C. Lacunar infarcts in patients aged 85 years and older. *Acta Neurol Scand.* 2000 Jan;101(1):25-9. doi: 10.1034/j.1600-0404.2000.00005.x.
53. Cai Z, He W, Peng CY, Zhou J, Xu QL, Wu ZS. The prevalence of lacunar infarct decreases with aging in the elderly: a case-controlled analysis. *Clin Interv Aging.* 2016 May 26;11:733-8. doi: 10.2147/CIA.S108166.
54. Yu L, Yang L, Zhang X, Yuan J, Li Y, Yang S, Gu H, Hu W, Gao S. Age and recurrent stroke are related to the severity of white matter hyperintensities in lacunar infarction patients with diabetes. *Clin Interv Aging.* 2018 Dec 7;13:2487-2494. doi: 10.2147/CIA.S184463.
55. Farfel JM, Yu L, Boyle PA, Leurgans S, Shah RC, Schneider JA, Bennett DA. Alzheimer's disease frequency peaks in the tenth decade and is lower afterwards. *Acta Neuropathol Commun.* 2019 Jul 3;7(1):104. doi: 10.1186/s40478-019-0752-0.
56. Kaushik S, Vani K, Chumber S, Anand KS, Dhamija RK. Evaluation of MR Visual Rating Scales in Major Forms of Dementia. *J Neurosci Rural Pract.* 2021 Jan;12(1):16-23. doi: 10.1055/s-0040-1716806.
57. Mårtensson G, Ferreira D, Cavallin L, Muehlboeck JS, Wahlund LO, Wang C, Westman E; Alzheimer's Disease Neuroimaging Initiative. AVRA: Automatic visual ratings of atrophy from MRI images using recurrent convolutional neural networks. *Neuroimage Clin.* 2019;23:101872. doi: 10.1016/j.nicl.2019.101872.
58. Molinder A, Ziegelitz D, Maier SE. Validity and reliability of the medial temporal lobe atrophy scale in a memory clinic population. *BMC Neurol* 2021; 21: 289. Available from: <https://doi.org/10.1186/s12883-021-02325-2>.
59. Wahlund LO, Westman E, van Westen D, Wallin A, Shams S, Cavallin L, Larsson EM; From the Imaging Cognitive Impairment Network (ICINET). Imaging biomarkers of dementia: recommended visual rating scales with teaching cases. *Insights Imaging.* 2017 Feb;8(1):79-90. doi: 10.1007/s13244-016-0521-6.
60. Agarwal P, Panda AK, Jena S, Mohapatra S. Correlation of Cerebral Atrophy and White Matter Hyperintensity Burden in MRI with Clinical Cognitive Decline. *Siriraj Med J* 2022; 74(5):323-329. Available from: <https://he02.tci->
61. Visser PJ, Verhey FR, Hofman PA, Scheltens P, Jolles J. Medial temporal lobe atrophy predicts Alzheimer's disease in patients with minor cognitive impairment. *J Neurol Neurosurg Psychiatry.* 2002 Apr;72(4):491-7. doi: 10.1136/jnnp.72.4.491.
62. Kaltoft NS, Marnier L, Larsen VA, Hasselbalch SG, Law I, Henriksen OM. Hybrid FDG PET/MRI vs. FDG PET and CT in patients with suspected dementia - A comparison of diagnostic yield and propagated influence on clinical diagnosis and patient management. *PLoS One.* 2019 May 2;14(5):e0216409..
63. enjamin P, Trippier S, Lawrence AJ, Lambert C, Zeestraten E, Williams OA, Patel B, Morris RG, Barrick TR, MacKinnon AD, Markus HS. Lacunar Infarcts, but Not Perivascular Spaces, Are Predictors of Cognitive Decline in Cerebral Small-Vessel Disease. *Stroke.* 2018 Mar;49(3):586-593. doi: 10.1161/STROKEAHA.117.017526.