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Review Article

The role of MRI in assessing cognitive impairment changes

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ABSTRACT

Aging brings about various changes in the brain, leading to cognitive alterations that are increasingly relevant with extended life expectancy. Dementia, characterized by chronic cognitive impairment, is on the rise due to longer life expectancy, imposing a substantial burden on healthcare systems. Dementia encompasses conditions like Alzheimer's disease (AD), vascular dementia (VaD), Lewy body dementia (LBD), and frontotemporal dementia, each with its distinct symptoms and progression. Magnetic resonance imaging (MRI), especially 3T MRI, plays a crucial role in monitoring and diagnosing dementia, aiding in patient selection for emerging therapies. Study involves a comprehensive literature search without restrictions on date, language, age/publication type. Dementia can be divided into neurodegenerative and nondegenerative categories, with AD being the most prevalent. Diagnosis relies on clinical evaluation, supported by neuroimaging techniques like MRI. Various MRI findings, such as cerebral atrophy, microbleeds, white matter hyperintensities, lacunes, and strategic infarcts, offer insights into dementia-related brain changes. These findings facilitate early diagnosis, prognosis, and treatment monitoring, with standardized assessment tools and volumetric analysis enhancing diagnostic accuracy. As life expectancy continues to rise, MRI's role in assessing cognitive impairment changes becomes increasingly vital in addressing the growing challenge of dementia.

Keywords: Dementia, MRI, AD, Cerebral atrophy, Microbleeds, White matter hyperintensities, Lacunes, Strategic infarcts

INTRODUCTION

Aging brings about various changes in our body, including the brain. These changes range from molecular to structural levels, leading to alterations in brain size, blood vessel patterns, and, more frequently, cognitive functions.[1] It is important to note that biological aging and chronological aging aren't entirely synonymous. Projections suggest that by 2050, the global average life expectancy will increase by six years (currently standing at 72 years).[2] Consequently, it is essential to comprehend

how this extended lifespan will impact individuals' health, memory, and cognitive abilities. Aging affects both the structure and function of the brain, but we still lack a comprehensive understanding of these phenomena. Studies have stressed the importance of investigating the connection between brain structure and specific cognitive skills in the aging population.[3]

Dementia is a syndrome characterized by a chronic cognitive impairment that affects multiple domains and has a significant impact on a person's ability to perform daily activities (ADL).[4] With the rise in life expectancy, the global prevalence of dementia is on the upswing, placing a growing economic and social burden on healthcare systems worldwide.[5] It is crucial to note that dementia is the final stage of the disease, preceded by a condition known as mild cognitive impairment (MCI). MCI is characterized by subtle cognitive changes that do not significantly affect a person's ability to carry out daily activities and is a focal point of interest for researchers working on drug development.[6] Individuals with MCI can still maintain their independence, and interventions that can slow down or halt its progression have the potential to enhance their daily functioning.[7,8] Given the profound consequences of dementia, there is an urgent necessity to address it by developing improved diagnostic tools and disease-modifying treatments. Dementia can significantly impact an individual's quality of life and their ability to function independently. It often requires medical evaluation, diagnosis, and appropriate care and support. Common forms of dementia encompass AD, VaD, LBD, and frontotemporal dementia, among various others. The specific symptoms and progression of dementia can vary depending on the underlying cause. AD, for example, is characterized by memory loss and cognitive decline, while other forms of dementia may have different symptoms and patterns of progression.

MRI has become essential in the ongoing monitoring, diagnosis, and evaluation of treatment effectiveness in dementia, particularly with the advent of 3T MRI and its advanced techniques.[9] MRI can potentially assist in selecting suitable patients for emerging therapies aimed at reducing amyloid levels, such as aducanumab. Additionally, it can aid in the identification of amyloid-related imaging abnormalities (ARIA) associated with treatments that modify amyloid.

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on October 23, 2023, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the role of MRI in assessing cognitive impairment changes. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

Etiology

Dementia can be classified into two main categories based on its primary underlying pathophysiological mechanisms: neurodegenerative (or proteinopathies) and nondegenerative dementia, which encompasses various conditions (Table 1).[10]

Table 1: Neurodegenerative and nondegenerative causes of dementia and entity examples.[10]

Variables	Causes
Neurodegenerative causes	
AD;	Parkinson's disease;
Frontotemporal dementia;	Posterior cortical atrophy;
LBD;	Amyotrophic lateral sclerosis;
Progressive supranuclear palsy;	Multiple system atrophy;
Corticobasal degeneration;	Huntington's disease
Nondegenerative causes	
Structural brain lesions	Haemorrhagic and ischemic stroke; primary and secondary tumors; traumatic brain injury; diffuse axonal injury; normal pressure hydrocephalus
Infectious	Meningitis; encephalitis; acquired immunodeficiency syndrome; abscess; syphilis; Creutzfeldt–Jacob disease; Whipple's disease
Demyelination	Multiple sclerosis Metabolic Diabetes mellitus; mitochond
Metabolic	Diabetes mellitus; mitochondrial diseases; lysosomal diseases; obesity
Epilepsy	Therapy resistant; status epilepticus
Endocrine	Hyperthyroidism; hypothyroidism; Hashimoto's encephalitis
Deficiency	Vitamin (e.g., Korsakoff's syndrome) and mineral deficiencies
Intoxication	Alcohol; psychoactive substances; medication-induced (anticholinergic drugs); heavy metals, organic solvents; carbon monoxide
Psychiatric	Schizophrenia; depression; psychosis
Post-anoxia	Choking or strangulation; drowning; chronic smoke inhalation

Nondegenerative dementia is typically caused by structural brain abnormalities or systemic diseases, with VaD being the most common among them.[4] Identifying causes other than degeneration is important because it can lead to the initiation of specific treatments, and in some cases, it may even stabilize or reverse cognitive decline.

Neurodegenerative dementias constitute the predominant group. AD, the most common phenotype, accounts for 60-80% of late-onset dementias and is characterized by progressive memory loss. Another subtype is LBD, which presents with features such as parkinsonism, fluctuating cognition, rapid eye movement sleep behavior disorder, and visual hallucinations. LBD is distinguished from other types of dementia by significant visuospatial, executive, and attention deficits in the absence of major memory impairment.[4,7] Frontotemporal dementia (FTD) represents the next most prevalent type of neurodegenerative dementia and comprises three primary subtypes: behavioral variant FTD (bvFTD), nonfluent variant primary progressive aphasia (nfvPPA), and semantic variant primary progressive aphasia (svPPA).[10] In instances of early-onset dementia (occurring before the age of 65), the occurrence rates vary, with AD still being the most widespread, but a higher percentage of FTD and LBD cases are observed. Among the elderly population, as many as 30-40% of all dementia cases are linked to mixed pathology, typically involving a combination of AD and VaD.

Diagnosis

The diagnosis of dementia primarily relies on clinical assessment, with laboratory tests and neuroimaging serving as supplementary tools. The evaluation of a patient with suspected dementia typically begins with a thorough medical history.[4]

Neuroimaging plays a crucial role and includes both structural and functional imaging techniques. MRI, computed tomography (CT), and cerebral positron emission tomography (PET) scans are commonly used. MRI is preferred over CT due to its higher sensitivity in detecting vascular lesions and various forms of dementia. CT may be used initially to rule out secondary causes of dementia, especially when MRI is contraindicated or if no previous MRI scans are available for comparison.[10]

MRI dementia protocol

It is advisable to use a 3T field strength MRI scanner rather than a 1.5T scanner, as the former offers advantages such as enhanced image resolution.[11]

Additionally, the choice of MRI receive head coil should be carefully considered. The number of channels in the head coil can significantly vary, depending on the manufacturer's recommendation. For instance, Siemens recommends using a 64-channel head coil for anatomical studies.[12] Selecting the appropriate field strength and head coil is crucial to maintaining consistency and accuracy in research studies involving MRI.[10]

MRI findings

To achieve accurate MRI assessments, it is essential to have a comprehensive understanding of the anatomy and physiology of each brain region. This knowledge is crucial because the distribution of affected areas in different neurological conditions explains the variations in symptoms and imaging patterns observed.[4]

Cerebral atrophy

Cerebral atrophy, the shrinkage of brain tissue, can be observed even in the early and presymptomatic stages of various neurological conditions. Many diagnostic guidelines incorporate atrophy patterns as essential features, and there is substantial evidence supporting their use in diagnosing dementia.[13] One key indicator of several types of dementia, especially those linked to cognitive decline, is medial temporal lobe atrophy (MTA), which affects cognition-relevant regions such as the hippocampus (HC) and structures along the parahippocampal gyrus.[14] In AD, MTA is typically symmetric, but it can also present asymmetrically, as is often seen in frontotemporal dementia (FTD). The entorhinal cortex and the HC are early targets of atrophy, making HC volume analysis a potentially valuable marker for early diagnosis. Different patterns of HC atrophy may aid in distinguishing between different forms of mild dementia, such as LBD, FTD, and Creutzfeldt–Jakob disease (CJD).[15] However, the precision of distinguishing AD from other types of dementia, like LBD, is lower compared to distinguishing it from healthy individuals.[10]

Frontal lobe atrophy is predominantly associated with FTD, a condition characterized by behavioral or language changes. Atrophy initially occurs in the anterior cingulate, insula, and frontal lobes, with a typical pattern of bilateral and possibly asymmetric frontal and temporal atrophy, following an anterior-to-posterior gradient. Simultaneous atrophy of the caudate head can lead to a widening of the frontal horns of the lateral ventricles, which is disproportionate. Broadening of the orbitofrontal sulci may be an early sign. Atrophy patterns often correlate with clinical subtypes.

For instance, nonfluent variant primary progressive aphasia (nvPPA) primarily affects left perisylvian regions, including the inferior, opercular, and insular areas, while semantic variant primary progressive aphasia (svPPA) predominantly targets ventral and lateral regions of the anterior temporal lobes, along with the anterior HC and the amygdala. Both subtypes exhibit left-hemispheric dominance.[4] Posterior/parietal atrophy (PA) is characterized by early and significant visual and visuospatial impairments, with less apparent memory loss. It is associated with atrophy in the parieto-occipital and posterior temporal regions, typically presenting

asymmetrically, with the right side being more affected. PA is most commonly found in atypical AD and the logopenic variant of a primary progressive aphasia (PPA). In LBD, PA is less common than in AD, even though occipital hypometabolism is a characteristic pattern seen on PET scans.[16,17] Corticobasal degeneration, subcortical gliosis, and prion diseases are conditions less frequently associated with PA. In individuals with AD, some studies have found significant PA even in the absence of visible atrophy in the medial temporal lobe (MTL), especially in younger individuals. This suggests that relying solely on MTA for AD diagnosis may result in some patients going undiagnosed.[18]

Deep gray matter (GM) atrophy is another crucial imaging indicator. Similar to cortical atrophy, the patterns of deep GM involvement vary. The caudate nucleus and thalamus are commonly affected in behavioral variant FTD (bvFTD).[19] In LBD, the thalamus is also affected, particularly in the ventral–dorsal and pulvinar regions.[20] In AD, thalamic involvement includes asymmetric atrophy in the ventrolateral and ventromedial nuclei, correlating with disease severity. Asymmetric caudate nucleus atrophy can also be found in AD patients.[19,21] Although some patterns of deep GM atrophy have been identified, there are still discrepancies between studies, necessitating further research.

Additionally, the presence of iron deposition in the basal ganglia (BG) is noteworthy. Some data suggest that iron accumulation in the BG can affect verbal memory and executive function. While iron depositions are commonly associated with Parkinson's disease, if detected, they should be reported in radiological findings as another potential cause of dementia.[22]

Microbleeds

Cerebral microbleeds (CMBs) are characterized by the persistent presence of blood breakdown products, particularly hemosiderin. These tiny spots, typically less than 10 mm in diameter, appear as areas of signal loss on T2-weighted images and show a characteristic "blooming" effect on T2*-weighted gradient echo images (Figure 1).[23]

A more sensitive imaging sequence for detecting CMBs is susceptibility-weighted angiography (SWAN), which enhances the "blooming" effect, making CMBs more apparent, although they may appear unevenly formed. Filtered phase SWAN is used to differentiate between CMBs and calcifications. On SWAN, both may appear hypointense, but on filtered phase images, calcifications (diamagnetic) can appear hyperintense while CMBs (paramagnetic) are seen as hypointense lesions (Figure 1).

However, the appearance of CMBs on a phase image can vary depending on whether filtered phase SWAN is used in a right- or left-handed coordinate system.[10]

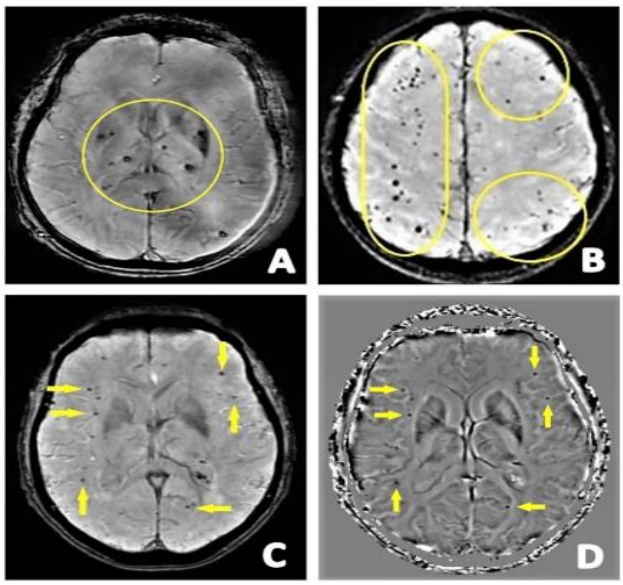

Figure 1 (A-D): The first row presents axial SWAN images showing: multiple CMBs in the deep GM (circle) indicating probable hypertensive disease; multiple lobar CMBs (circle) indicating probable CAA. Second row shows: axial SWAN images with multiple lobar CMBs (arrows) corresponding with filtered phase images for confirming CMBs (arrows).
SWAN: Susceptibility-weighted angiography; CMBs: Cerebral microbleeds; GM: Gray matter; CAA: Cerebral amyloid angiopathy.[23]

CMBs have been linked to various underlying pathologies, including cerebral small vessel disease (cSVD), stroke, brain injury, radiation exposure, and VaD. While CMBs may be asymptomatic, early detection is crucial for assessing the risk of future cerebrovascular conditions and cognitive decline.[24] CMBs are classified into three categories based on their location: deep (in areas like the basal ganglia, thalamus, internal and external capsule, and corpus callosum), lobar (in the frontal, temporal, parietal, and occipital lobes), and infratentorial (in the brainstem and cerebellum). The localization of CMBs can aid in differentiating between cerebral amyloid angiopathy (CAA) and hypertensive disease.[10] Lobar CMBs are more commonly associated with CAA, with a preference for distribution in the parietooccipital region. On the other hand, deep CMBs are more often associated with small penetrating artery disease related to hypertension.

A solitary CMB in any brain region is typically not considered clinically significant. However, the presence of several lobar CMBs in an individual over the age of 55 without other explanations may suggest CAA. Multiple lobar CMBs are frequently detected in individuals who meet the clinical criteria for AD due to the common co-occurrence of CAA and AD. The presence of superficial siderosis, a condition characterized by iron deposition on the brain's surface, can further strengthen the diagnosis of CAA. Cerebellar CMBs can occur in both CAA and

hypertension and therefore do not significantly contribute to the diagnosis.

White matter hyperintensities

White matter hyperintensities (WMHs) appear as regions with a high-intensity signal on T2-weighted (T2W) and FLAIR sequences in brain imaging. They are a prominent feature of cSVD and are commonly observed in the elderly.[23]

Lacunes and dilated perivascular spaces

A lacune is a brain lesion that typically ranges in size from 2 to 20 mm in diameter and commonly affects the subcortical white matter (WM) and deep gray matter (GM). These lesions are fluid-filled cavities surrounded by gliotic tissue and often precede infarcts in the deep perforating arteries of the brain. On imaging, lacunes appear with cerebrospinal fluid (CSF) signal intensity on all sequences and are encircled by a T2 hyperintensity rim. It is important not to confuse lacunes with dilated perivascular spaces (PVS). Dilated PVS are spaces filled with CSF that surround perforating arterioles and venules as they traverse through the brain parenchyma from the subarachnoid space. Dilated PVS typically result from volume loss in the surrounding tissue and are more commonly found in the basal ganglia (BG).[10] Both lacunes and dilated PVS are considered manifestations of cSVD. Differentiating between the two can be aided by the size and location of the lesions. Lesions smaller than 2 mm, located in the lower third of the corpus striatum and exhibiting an intensified CSF signal, are indicative of dilated PVS, as opposed to lacunes.[23]

There is evidence suggesting that lacunes may be associated with cognitive decline, increased mortality, and poor clinical outcomes. Independent of other factors, lacunes have been linked to cognitive impairment and have been found to be strong predictors of rapid global functional decline in older individuals living independently.[25]

Strategic infarcts

In addition to lacunar infarcts, another type of infarction that can significantly impact cognition, especially in cases of VaD, is strategic infarcts (Figure 2).[10] While large cortico-subcortical ischemic lesions can lead to acute cognitive deterioration, dementia progression resulting from a stroke may be driven by infarcts occurring in "strategic" brain regions crucial for normal cognitive functioning. Research exploring the relationship between the location of brain lesions and cognitive function has revealed that the degree of cognitive impairment following a stroke may depend on various lesion features, including type, size, number, location, and severity.[26] Interestingly, a study employing multivariate lesion-symptom mapping demonstrated that smaller-sized strategic infarctions were associated with compromised cognition.

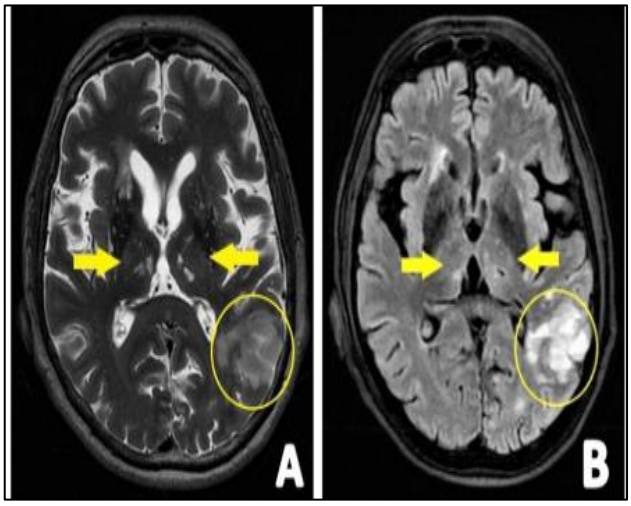

Figure 2 (A and B): Axial T2W and axial FLAIR images showing strategic infarctions in both thalami and in the parieto-occipital watershed area. Images were obtained in a male patient, age 71, with clinical manifestations of VaD. FLAIR: Three-dimensional T2-weighted fluid-attenuated inversion-recovery imaging.[10]

Strategic infarct locations that can result in dementia include specific brain regions such as the angular gyrus, both sides of the anterior cerebral artery territory, the paramedian thalamic area, the inferior medial temporal lobe (MTL), parieto-temporal and temporo-occipital association areas, superior frontal region, and parietal watershed areas in the dominant hemisphere. Additionally, dementia has been observed in cases of infarctions in the hippocampus (HC) or in bilateral/unilateral thalamic regions.[10] In a study focused on mapping these areas, it was discovered that the angular gyrus, basal ganglia (BG), and the white matter surrounding the BG, particularly on the left side, showed the strongest correlation with a decline in overall cognitive function.[27]

Standardized assessment

Standardized assessment tools play a crucial role in the evaluation of dementia-related brain changes. These tools offer efficient, reproducible, and cost-effective ways to assess volume loss, cerebral microbleeds (CMBs), and white matter hyperintensities (WMHs). Commonly used scales include the MTA scale for medial temporal atrophy, the GCA scale for global cortical atrophy, the Koedam score for posterior atrophy, and the Fazekas scale for WMH severity. These scales enhance diagnostic precision, although they may have limitations in certain cases. Advanced techniques like arterial spin labeling (ASL) further contribute to dementia evaluation. Volumetric analysis, performed by specialized software like volBrain and FreeSurfer, aids in identifying volume

loss and differentiating various dementia causes, improving diagnostic accuracy. Despite their potential, these methods are predominantly utilized in research.[10]

CONCLUSION

MRI has become indispensable in assessing cognitive impairment changes, particularly in dementia. Various MRI findings, including cerebral atrophy, microbleeds, white matter hyperintensities, lacunes, and strategic infarcts, provide valuable insights into dementia-related brain alterations. These imaging markers aid in early diagnosis, prognosis, and monitoring of treatment effectiveness. Standardized assessment tools and volumetric analysis further enhance diagnostic precision. While primarily utilized in research settings, utilization of advanced MRI techniques is crucial for improving early dementia diagnosis and patient care. As global life expectancy continues to rise, MRI's role in assessing cognitive impairment changes becomes increasingly vital in addressing the growing challenge of dementia.

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REFERENCES

1. Peters R. Ageing and the brain: This article is part of a series on ageing edited by Professor Chris Bulpitt. Postgraduate Med J. 2006;82(964):84-8.
2. Dartora CM, Borelli WV, Koole M, Marques da Silva AM. Cognitive Decline Assessment: A Review From Medical Imaging Perspective. Frontiers Aging Neurosci. 2021;13:704661.
3. Oschwald J, Guye S, Liem F. Brain structure and cognitive ability in healthy aging: a review on longitudinal correlated change. Rev Neurosci. 2019;31(1):1-57.
4. Álvarez-Linera Prado J, Jiménez-Huete A. Neuroimaging in dementia. Clinical–radiological correlation. Radiología (English Edition). 2019;61(1):66-81.
5. Jongsiriyanyong S, Limpawattana P. Mild Cognitive Impairment in Clinical Practice: A Review Article. Am J Alzheimer's Dis Other Dementias®. 2018;33(8):500-7.
6. Petersen RC, Lopez O, Armstrong MJ, Thomas SDG, Mary G, David G et al. Practice guideline update summary: Mild cognitive impairment. Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology. Neurology. 2018;90(3):126-35.
7. NA. 2021 Alzheimer's disease facts and figures. Alzheimer's Dementia. 2021;17(3):327-406.
8. Jack Jr. CR, Albert MS, Knopman DS, Guy MMcK, Reisa AS, Maria CC et al. Introduction to the recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimer's Demen. 2011;7(3):257-62.
9. Barakos J, Purcell D, Suhy J, Chalkias S, Burkett P, Marsica CG et al. Detection and Management of Amyloid-Related Imaging Abnormalities in Patients with Alzheimer's Disease Treated with Anti-Amyloid Beta Therapy. J Prevention Alzheimer's Dis. 2022;9(2):211-20.
10. Živanović M, Aracki Trenkić A, Milošević V. The role of magnetic resonance imaging in the diagnosis and prognosis of dementia. Biomol Biomed. 2023;23(2):209-24.
11. Ismail Z, Black SE, Camicioli R. Recommendations of the 5th Canadian Consensus Conference on the diagnosis and treatment of dementia. Alzheimer Demen. 2020;16(8):1182-95.
12. Schmitt T, Rieger JW. Recommendations of Choice of Head Coil and Prescan Normalize Filter Depend on Region of Interest and Task. Frontiers Neurosci. 2021;15.
13. Harper L, Bouwman F, Burton EJ. Patterns of atrophy in pathologically confirmed dementias: a voxelwise analysis. J Neurol Neurosur Psychiatr. 2017;88(11):908-16.
14. Van Someren EJW, Oosterman JM, Van Harten B. Medial temporal lobe atrophy relates more strongly to sleep-wake rhythm fragmentation than to age or any other known risk. Neurobiol Learning Memory. 2019;160:132-8.
15. Huang L, Chen K, Hu X, Guo Q. Differential Atrophy in the Hippocampal Subfield Volumes in Four Types of Mild Dementia. Frontiers in Neurosc. 2020;14.
16. Mak E, Su L, Williams GB, O'Brien JT. Neuroimaging characteristics of dementia with Lewy bodies. Alzheimer's Res Therapy. 2014;6(2):18.
17. Holden SK, Bettcher BM, Pelak VS. Update on posterior cortical atrophy. Curr Opinion Neurol. 2020;33(1):68-73.
18. Lehmann M, Koedam ELGE, Barnes J, Jonathan WB, Natalie SR, Yolande ALP et al. Posterior cerebral atrophy in the absence of medial temporal lobe atrophy in pathologically-confirmed Alzheimer's disease. Neurobiol Aging. 2012;33(3):627-7.
19. Oldan JD, Jewells VL, Pieper B, Wong TZ. Complete Evaluation of Dementia: PET and MRI Correlation and Diagnosis for the Neuroradiologist. Am J Neuroradiol. 2021;42(6):998-1007.
20. Watson R, Colloby SJ, Blamire AM, Wesnes KA, Wood J, O'Brien JT. Does attentional dysfunction and thalamic atrophy predict decline in dementia with Lewy bodies? Parkinsonism Rel Disord. 2017;45:69-74.
21. Low A, Mak E, Malpetti M. Asymmetrical atrophy of thalamic subnuclei in Alzheimer's disease and amyloid-positive mild cognitive impairment is associated with key clinical features. Alzheimer's Dementia: Diagnosis, Assessment Dis Monitoring. 2019;11(1):690-9.

22. Biel D, Steiger TK, Bunzeck N. Age-related iron accumulation and demyelination in the basal ganglia are closely related to verbal memory and executive functioning. Scient Rep. 2021;11(1):9438.
23. Stojanov D, Vojinovic S, Aracki-Trenkic A, Aleksandar T, Daniela B-S, Srdjan L et al. Imaging Characteristics of Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leucoencephalopathy (CADASIL). Biomole Biomed. 2015;15(1):1-8.
24. Chesebro AG, Amarante E, Lao PJ, Meier IB, Mayeux R, Brickman AM. Automated detection of cerebral microbleeds on T2*-weighted MRI. Scientific Rep. 2021;11(1):4004.
25. Ghaznawi R, Geerlings MI, Jaarsma-Coes MG. The association between lacunes and white matter hyperintensity features on MRI: The SMART-MR study. J Cerebral Blood Flow Metabol. 2019;39(12):2486-96.
26. Kalaria RN, Akinyemi R, Ihara M. Stroke injury, cognitive impairment and vascular dementia. Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease. 2016;1862(5):915-25.
27. Zhao L, Biesbroek JM, Shi L Wenyan L, Hugo JK, Winnie WC et al. Strategic infarct location for post-stroke cognitive impairment: A multivariate lesion-symptom mapping study. J Cerebral Blood Flow Metabol. 2018;38(8):1299-311.

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