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Modern methods for the instrumental detection of cognitive impairment in Alzheimer's and dementia patients

Aminath Samha[1,2], Dr. Ghassan Salibi MD.[3]
[1,3] Charisma University
[2] Kursk State Medical University

Abstract

Patients with cognitive impairment diseases are no longer fully oriented to time and space. The disease may be the root cause of all cognitive impairments (CI). Depending on the precise diagnosis, different cognitive disorders require different treatments. The three major goals of neuroimaging in Alzheimer's disease (AD) are to detect very early AD at a prodromal stage of moderate cognitive impairment, to differentiate AD from other dementia-causing illnesses, and to forecast the progression of MCI to AD. Consequently, this study's objective is to examine the most sensitive instrumental testing techniques for the early identification of cognitive deterioration in AD and dementia patients.

Keywords: Dementia, Alzheimer's disease, cognitive impairment

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Introduction

Before the 20th century, Dementia was a relatively uncommon condition since fewer individuals in pre-industrial societies lived to old age. As technology progresses, more people get access to healthcare and live longer. The number of seniors is rising. Population growth is now more rapid than in previous decades. According to WHO figures, "those 60 years and older now outnumber those under the age of five in terms of population. It is predicted that by 2050, there will be a 22% rise in the percentage of individuals over 60. This figure is anticipated to rise to 78 million in 2030, and 139 million in 2050 as the percentage of older people in the population rises in practically all nations". [1]

According to the Dementia Australia website, "Dementia was not first described as we understand it now until the middle of the 1970s. Around the world, more than 55 million people presently have Dementia, and an estimated 10 million new cases are diagnosed yearly. There are more than 100 conditions that can lead to Dementia. Alzheimer's disease, vascular Dementia, and Dementia with Lewy bodies are the three most prevalent causes of Dementia." [4] WHO states, "The most prevalent kind of dementia, Alzheimer's disease, can be a factor in 60–70% of cases."[2]

A controlled neuropathological investigation found and published in BMC medicine, it is said that "dementia with Lewy bodies (DLB) accounts for 10–25% of cases of dementia, Alzheimer's disease (AD) accounts for 60%–70%, and the pure vascular disease accounts for 8–10% of dementia cases." [3]

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Neuroimaging developments have significantly fueled this expansion. We discussed how structural and functional neuroimaging methods have contributed to understanding the neural underpinnings of cognitive impairment. Imaging has historically been used to rule out curable and reversible causes of Dementia rather than to use imaging to learn more about the pathogenesis of the different dementias. It is possible to examine the anatomy, biochemistry, metabolic condition, and functional capabilities of the brain using several imaging methods.

Currently, the usage of CT scans., structural MRI investigations, and PET scans in individuals accompanied by Alzheimer's disease (AD) and Dementia is very popular. To facilitate the early detection of Alzheimer's disease and moderate cognitive impairment, evaluate early structural and metabolic abnormalities, such as those in the entorhinal cortex, medial temporal lobe's gray matter components, and the hippocampus.

The study's goals and purpose

This study aims to estimate the sensitivity and specificity of different methods of instrumental CD examination in elderly patients.

Objectives: Modern Research methods of instrumental examination of older patients with CD in the practice of general practitioners according to evidence-based medicine to compare the best approach to diagnosing CD in Dementia and AD

Resources and research techniques

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A thorough literature of the last five years was searched on the NCBI database using the terms “cognitive impairment, dementia, Alzheimer's disease, and instrumental study of cognitive impairment.”

Alzheimer’s Dementia

“Alzheimer's disease” is diagnosed using a variety of different criteria. “The Diagnostic and Statistical Manual of Mental Disorders and the National Institute” on Aging-Association Alzheimer's Criteria are the two that are used most frequently. Both have defined parameters for Dementia with Alzheimer's and mild cognitive impairment caused by Alzheimer's disease “referred to in DSM-5”.

Hippocampal volume typically decreases by 10 to 15 percent in MCI individuals with an average MMSE score of 25 and by 20–25% in AD patients with an average MMSE score of 20. [23] Measuring these significant reductions in the medial temporal lobe (induced by AD) early on in AD and MCI diagnosis can be extremely beneficial.[23] [24]

The massive shrinking of the cortical tissue due to neuronal cell death is the most apparent aspect of Alzheimer's brain. At autopsy, the AD brain has significant atrophy. For those of us in middle age, the expansion of the ventricles and sulci in conjunction with the diminished tissue is readily apparent—and a little problematic. Amyloid plaques are the extracellular deposits that Alzheimer observed. A primary component of the plaques was found to be a small protein called amyloid-β or A-β.

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It is interesting to learn how AD proteins move throughout the brain and how some target certain brain areas but not others. It has been shown that, like prion disease, improperly folded A and tau can change the structure of structurally normal peptides. These might be trans-synaptically passed from one neuron to another.

To rule out structural issues and provide useful diagnostic information, structural imaging using computed tomography or, preferably, MRI is indicated for all patients being assessed for cognitive impairment.

Computer tomography (CT scan)

A head CT test for Alzheimer's disease has two objectives. First, the test allows your doctor to rule out circumstances that resemble Alzheimer's disease. These include strokes, hemorrhages, and malignancies. CT also scans aid in detecting the loss of brain tissue associated with Alzheimer's disease. Amyloid plaques are caused by abnormal protein levels called amyloid, typically in affected minds. Alzheimer's disease causes the brain to shrink by destroying crucial neurons and plaques that already exist inside the brain. [8]

Magnetic Resonance Imaging (MRI)

Since AD first manifests in the medial temporal lobe and hippocampus, these areas need to be our main areas of attention. As AD progresses, the remaining portions atrophy occurs in the temporal pole, para-hippocampal, and fusiform gyri of the medial temporal lobe (MTL).In more severe forms of Alzheimer's disease, the frontal, parietal, and temporal lobes eventually begin to atrophy along with the rest of the cortex. The hallmark of AD is extensive atrophy, which it shares with other end-stage dementias.[22] [25]

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A widely used technique for determining AD pathology is volumetric MRI scans of the hippocampus. Neuronal loss in the hippocampus manifests as decreased hippocampal volume. Hippocampal atrophy can be assessed using either manual or automatic segmentation from these scans, which are T1-weighted images. The anatomic validity of volumetric MRI measures is suggested by research by Babinski et al., which found that MRI was a useful tool for measuring hippocampal volume and projected volumes that were significantly associated with neuronal counts. Another study discovered that hippocampal volume deficits are quantifiable with MRI and are early signs of AD disease. [6]

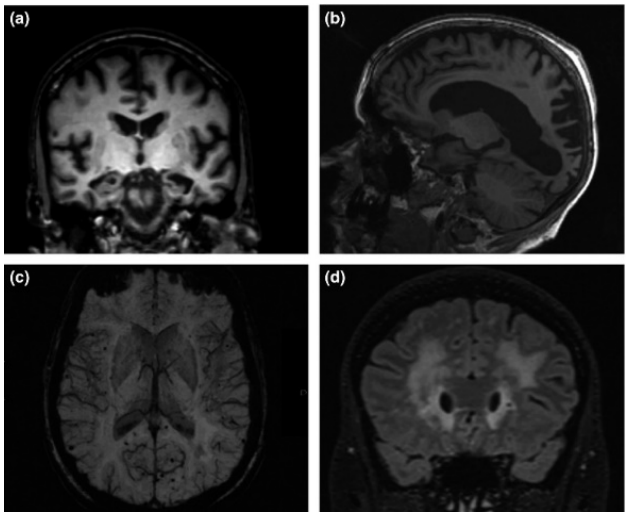

Figure1: “MRI images showing (a) characteristic hippocampal atrophy in a typical Alzheimer's disease case best visualized in the coronal plane on T1; (b) parieto-occipital atrophy in a posterior cortical atrophy case, here demonstrated in the sagittal plane on T1; (c) microbleeds which are best visualized on SWI (the posterior distribution seen on this axial image is characteristic of cerebral amyloid angiopathy); (d) extensive periventricular and subcortical white matter hyperintensities best visualized on FLAIR, seen here on a coronal image.”

"https://onlinelibrary.wiley.com/doi/10.1111/ene.13439 "

Another application of structural MRI, in addition to measuring volume decreases, is the detection of the cortical decrease in thickness in the brain's parietal, orbitofrontal, and temporal areas.

As atrophy progresses, the CSF spaces, such as the ventricles, cerebral sulci, and cerebellar folia, enlarge. Ventricle enlargement rates are higher in AD than ventricular dilation, which is typical of normal aging. The size of the lateral ventricles and AD progression is similarly correlated.

Amyloid PET imaging

The primary components of abnormally folded amyloid plaques, extracellular accumulations, are two by-products of APP metabolism (A40 and A42). A42 is more prevalent than A40 inside because it is insoluble and fibrillates more quickly than other materials; it forms plaques. Even though the progression of amyloid deposition is not always predictable, it usually starts in the exocortex and only later affects the subcortical areas. The bulk of the paired helical filaments that make up neurofibrillary tangles is hyperphosphorylated tau. Tau illness frequently begins in the allocortex of the medial temporal lobe before progressing to the associative exocortex (which includes the hippocampus and entorhinal cortex). [6]

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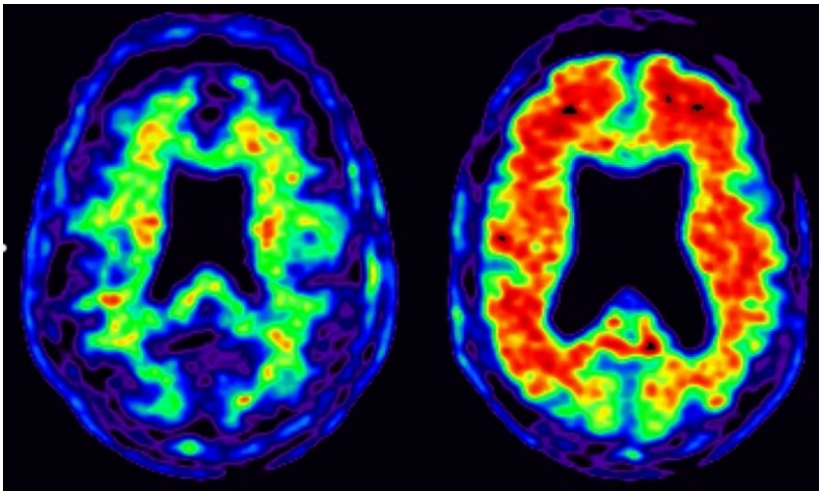

Figure 2: "Florbetapir amyloid positron-emission tomography scan in healthy control (left) and an Alzheimer's disease patient (right). Warm colors indicate high amyloid accumulation. For clinical purposes, florbetapir scans are read on a grey scale."

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According to research presented in "EFNS guidelines for the diagnosis and management of Alzheimer's disease," a PET scan may reliably predict a pathologic diagnosis of AD with a sensitivity of 93% and a specificity of 63%. With a specificity of more than 95% in cases of early-onset AD, a PET scan is the distinction between AD and other dementias and is especially useful in differential diagnosis [5]. Amyloid PET, which is not regularly reimbursed in most countries, is currently the subject of several pieces of research investigating its clinical efficacy and economic sustainability.

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Vascular Dementia

One of the most typical causes of Dementia is vascular etiology. In the "Rochester Epidemiology Project, 419 senior dementia patients had a postmortem diagnosis of AD in 51% of cases; other diagnoses included pure vascular dementia in 12% of cases, mixed vascular-Alzheimer dementia in 12% of cases, and "other" in the remaining instances."[12]

Computer tomography (CT scan)

Computed tomography (CT) is used for brain imaging in most studies. CT is, therefore, typical of the entire clinical group in acute stroke patients. The primary purpose of a CT scan in a clinical setting is to eliminate stroke imitators, such as brain tumors and bleeding. Early ischemia symptoms can commonly be seen on CT scans, such as edema, hypodensity, hyperdense arteries, and old stroke lesions. [11]

Magnetic Resonance Imaging (MRI)

The primary neuroimaging technique used in VCI is still MRI. MRI has better sensitivity and specificity than CT for detecting pathological changes in research and ordinary clinical usage, so it is preferred unless it is restricted.

Traditional Spin echo T1- and T2-weighted imaging shows macro-hemorrhages connected to cognitive impairment (such as venous infarcts), but micro-hemorrhages can be precisely identified utilizing T2*-weighted gradient echo images.[11]

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High-field MRI reveals the location and extent of brain atrophy and identifies lesions that may be chosen for histological analysis. It is possible to quantify small cerebrovascular lesions and assess the iron burden.

High-resolution 7.0-T MRI allows for in vivo cerebral micro-infarct detection. Detecting tiny cortical bleeds has a 96 percent reliability because cortical microbleeds vary in prevalence in the frontal areas of all neurodegenerative disease groups compared to the controls.[11].

Vascular dementia individuals' brains are more prone to have lacunes and white matter alterations. In addition, rather than being caused by cerebrovascular disease, the latter is generally seen in frontotemporal lobar degeneration brought on by Wallerian degeneration. The subpial layer's hemosiderin deposition causes superficial siderosis associated with an underlying cortical lesion, either a hemorrhage or an infarct following a hemorrhagic change. Frontotemporal lobar degeneration is associated with a substantial increase in iron accumulation in the basal ganglia.

Diffusion-weighted imaging can spot microstructural alterations in the white matter that seems to be normal, which is also influenced by local pathology and Wallerian degeneration from remote lesions in VCI. Compared to traditional MRI, the diffusion MRI indicators of normal white matter have a stronger relationship with cognition.

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Functional transcranial Doppler (fTCD)

Many studies have used fTCD to show poor neurovascular function in patients at high risk of cerebrovascular injury before the development of MRI indicators of clinical signs of a stroke or small artery disease.

The lack of an acoustic window restricts usage to patients by a slight proportion. It offers low spatial resolution, i.e., the evaluation will be restricted to the Willis circle's vascular regions, which is a general restriction of all TCD procedures. The observed values in individuals with considerable cardiac cycle oscillations (such as atrial fibrillation caused by BFV oscillations) were adjusted by averaging the activity across a single cardiac cycle.

To identify individuals with a heightened risk of vascular brain damage before permanent impairment occurs, fTCD may be a valuable pre-clinical screening technique. [13]

Positron emission tomography (PET)

By displaying cerebral functioning in often damaged brain areas, a PET scan can assist in clinical diagnosis.

When the association areas are damaged by significant hypometabolism, FDG-PET detects regions of localized cortical and subcortical hypometabolism in VCI, an alternate metabolic pattern to that found in AD. In several cortical areas, there was a significant decrease in rCMRglc "middle frontal cortex, temporoparietal cortex, basal ganglia, cerebellum, and brainstem."

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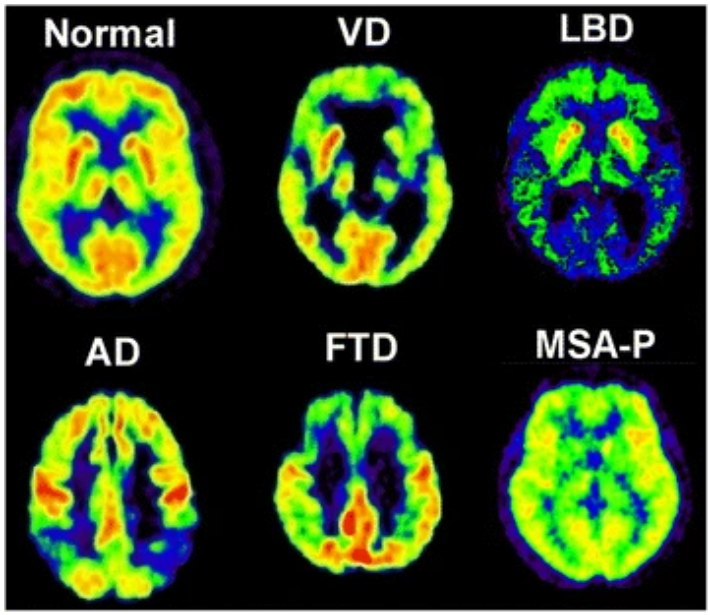

Figure 3: "Typical metabolic patterns for different types of Dementia compared to normal controls and Vascular Dementia (VD). Alzheimer's disease (AD), frontotemporal Dementia (FTD), and Lewy-Body Dementia (LBD) show distinct cortical patterns of decreased metabolism, while multisystem atrophy type P (MSD-P) shows a decreased metabolism in the putamen on both sides. In contrast, a typical feature of VD is the simultaneous occurrence of patchy, often asymmetrical cortical and subcortical areas of decreased glucose metabolism."

"https://bmcmedicine.biomedcentral.com/articles/10.1186/s12916-016-0725-0#Sec1"

Conclusion

To rule out Dementia with a curable etiology, CT and MRI scans may be utilized. Hippocampal atrophy can be evaluated using multi-slice CT, and coronal MRI can assist in making a clinical AD diagnosis (Level B). When a diagnosis is uncertain, FDG PET and perfusion SPECT are useful ancillaries. (level B). To discriminate between AD and DLB,

dopaminergic SPECT is helpful (level A). In a clinical context, follow-up using serial MRI helps track the development of the condition.

An accurate diagnosis will aid doctors in avoiding administering potentially harmful drugs to patients and will enable better evaluation of the treatment's effectiveness. Additionally, because neurodegenerative illnesses are linked to the emergence of pathologic alterations years before the onset of functional impairment, neuroimaging may be useful in identifying dementing disorders in their earliest presymptomatic stages.

Neuroimaging will continue to be a key component of dementia patient diagnosis. While most centers have access to and employ MRI, PET allows for the differentiation of Dementia caused by vascular and neurodegenerative conditions.

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Abbreviations

CI- Cognitive impairment

CD- cognitive dysfunction

MCI- Mild Cognitive Impairment

AD- Alzheimer's disease

GP- General Practice

CT- Computed tomography

MRI- Magnetic resonance imaging

MRS- magnetic resonance spectroscopy

PET- positron emission tomography

MSI- magnetic source imaging

SPECT- single photon emission computed tomography

ETI- electrical impedance tomography

TCD- transcranial doppler ultrasound

FTLD - frontotemporal lobar degeneration

VaD- vascular dementia

MFV - mean flow velocity

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