Current knowledge regarding: Diagnosis and treatment of Alzheimer's disease.

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Abstract

The most prevalent cause of dementia in the world is Alzheimer's disease, and as the global population ages, its incidence is only increasing. The two signature diseases of this neurodegenerative disease process are the accumulation of amyloid plaques and neurofibrillary tangles of hyper phosphorylated tau. The clinical presentation must meet a number of criteria for a diagnosis, and fluid and imaging indicators are also considered. Although there are trials underway to lessen the generation and overall load of disease inside the brain, treatment is primarily focused on symptomatic treatments. Here, we review recent developments in our knowledge of the clinical diagnosis and management of Alzheimer's disease and provide updates on ongoing clinical trials.

Keywords: Alzheimer's disease, Dementia, Amyloid, Positron emission tomography.

Introduction

A clinical syndrome known as dementia is characterised by a steady decline in two or more cognitive functions, such as memory, language, executive function, visuospatial function, personality, and behaviour. This loss of function results in the inability to perform instrumental and/or fundamental daily activities. Up to 80% of dementia diagnoses are due to Alzheimer's Disease (AD), which is by far the most prevalent dementia cause. Although stroke and cardiovascular disease are still the leading causes of death in the US, the proportion of deaths due to AD is rising by 89%. Cerebrospinal Fluid (CSF) and Positron Emission Tomography (PET) biomarkers paired with a few relatively recent clinical criteria can help diagnose AD in people who are still alive, but a definite diagnosis needs post-mortem examination of brain tissue. For patients with AD dementia in any stage, cholinesterase inhibitors are an option, while memantine is an option for those with moderate-to-severe AD dementia. When taken at the right moment during the course of the illness, these drugs have been found to improve both patient and caregiver quality of life; but, they do not alter the course of the illness or the rate of decline [1].

Recently, a test that improves the diagnostic precision for AD 10 was created thanks to advancements in non-invasive diagnostic imaging. Patients are subjected to a customised PET scan that detects the deposition of amyloid-(A) peptides into plaques in the living brain after injection of a radio labelled tracer agent. Using this technique, doctors were able to detect the illness with up to 96% sensitivity and 100% specificity in 2012. The same test showed comparable outcomes over the course of the following year in patients with less severe illness [2]. The US Food and Drug Administration has approved the use of florbetapir for the detection of AD pathology, nearly ten years after University of Pittsburgh researchers developed the first tracer. Examining the CSF for the presence of A42, hyper phosphorylated tau peptide (p-tau), and total tau protein is a more invasive but less expensive kind of investigation. Because there aren't many labs that can perform the fluid analysis, this method has a little lower diagnostic accuracy, comes with the hazards and inconveniences of a lumbar puncture surgery, and frequently takes weeks to get findings. Although a head-to-head comparison of CSF A42:p-tau ratio and amyloid PET imaging biomarkers revealed no difference in diagnostic accuracy, it is possible that the optimum test for a given patient would depend on accessibility, cost, and patient/provider preferences [3].

In both cerebrovascular illness and neurodegenerative disease, the control of cardiovascular risk factors contributes to overall brain health. People who follow the Mediterranean diet had a lower chance of developing cognitive decline and AD, according to recent systematic evaluations. In addition to preserving function and lessening caregiver stress in AD patients, regular aerobic exercise has long been known to protect metabolic diseases like diabetes mellitus and coronary artery disease. Exercise not only keeps patients' strength and agility from deteriorating as they age, but it also lowers neuropsychiatric symptoms and the resulting rise in care needs. Regardless of age at the start of exercise, recreational physical activity improves cognitive function later in life. Patients with genetic risk factors for AD who consistently exercised showed less atrophy in their brains than those who

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did not, indicating that aerobic exercise slows the progression of neurodegeneration. Regardless of cognitive function, all healthcare professionals should advise patients to engage in regular exercise because of the inherent systemic benefits and lack of health risks, even though larger controlled studies are still required to examine the long-term effects of physical activity in patients with biomarker-proven AD pathology [4].

The hypothesis holds that, although the abnormal protein is implicated at the onset of AD, the progression of clinical symptoms is caused by more widespread neural network dysfunction. This hypothesis was made possible by the failure of some targeted therapies toward in large-scale clinical trials. In almost all brain networks, gamma oscillation, a high-frequency brainwave rhythm, is linked to inter-neuronal communication and may be used to discriminate between real and imagined memories. Recently, Massachusetts Institute of Technology researchers discovered that inducing gamma-frequency oscillations in an AD mice model resulted in decreased a deposition and improved cognitive performance. This was accomplished by entraining the desired frequency in the mouse cortex using a non-invasive 40 Hz photic stimulator. Early-stage tests for this approach are also ongoing right now [5].

Conclusion

Clinical symptoms that matched the pattern of memory impairment and loss of functional independence across many cognitive domains were used for the diagnosis and treatment of AD. The NIA-AA and DSM-5's reclassification approach has expanded the spectrum of AD to encompass pre-clinical disease and MCI, laying the groundwork for the early detection of patients who are at risk. Today, there are a few commonly used diagnostic tests that support clinical assessment for a more precise diagnosis of AD pathology. These tests include bodily fluids and imaging tests, which have high specificity. The AD therapy options, however, continue to be supportive and symptomatic without affecting the long-term outlook. Memantine and cholinesterase inhibitors are two examples of medications that enhance cognition and alertness, respectively, without affecting the lifespan or general course of AD dementia. The only therapies that have been shown to reduce the risk of AD and potentially prevent overall cognitive decline are lifestyle changes like diet and exercise, which are first-line recommendations for all patients regardless of cognitive function. The current targets for prospective treatments are the pathological traits linked to AD, A and p-tau; however, early success in comparative research and smaller clinical trials is not yet replicable in larger-scale administrations.

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