Lifestyle Factors and Alzheimer's Disease Prevention

Samra Bashir[1], Manahil Farooq[2], Khadija Majeeb[2], Rao Zahid Abbas[3] and Saleha Tahir[1,3*]

1. Institute of Microbiology, University of Agriculture Faisalabad, Pakistan
2. Department of Microbiology, The University of Faisalabad, Pakistan
3. Department of Parasitology, University of Agriculture, Faisalabad, Pakistan

*Corresponding Author: **salehatahir999@gmail.com**

ABSTRACT

Alzheimer's disease (AD) has become much more common and concerning since it was first identified in the early 1900s. Alzheimer's disease is the most prevalent type of dementia. Several curable medical illnesses and lifestyle decisions are modifiable risk factors for AD development. The etiology and clinical effects of AD may be influenced by the molecular mechanisms of these risk factors. Among other factors, the aging of the population, which is somewhat observed globally, contributes to the rising prevalence of AD. Life expectancy continues to rise due to ongoing improvements in healthcare. The true frequency of AD is probably far lower than official figures show. Since its etiology is yet unknown, research into all possible risk factors that could contribute to the disease's development is essential.

Keywords: Alzheimer's disease, Dementia, Modifiable Risk factors, Prevalence, Prevention

To cite this article: Bashir S, M Farooq, K Majeeb, RZ Abbas & S Tahir. Lifestyle Factors and Alzheimer's Disease Prevention. Biological Times. 2024 November 3(11): 33-34.

Introduction

Dementia is a gradual, long-term illness marked by aberrant mental behavior and cognitive failure (1). It has emerged as the 21st century's biggest health and social care concern worldwide. Alzheimer disease (AD), a progressive, unrelenting, neurological disease, and vascular dementia (VD), primarily brought on by hypertension and arteriosclerosis, are two of the more prevalent forms of dementia (2). The primary form of dementia that has drawn attention from throughout the world is AD. Neuronal dysfunction, inflammatory processes, intracellular tangles of neurofibrillary cells (NFTs), and extracellular amyloid-β (Aβ) clusters, and neural death that results in cognitive impairment are some of the neuropathological indicators of Alzheimer's dementia, an aging neurological condition (3). This illness is terminal, degenerative, and incurable. A less common early-onset type may appear much sooner, even though it is often diagnosed in adults over 65 (4). It is estimated that by 2050, there will be 14 million AD sufferers in the US due to the population's progressive aging (5). There is currently no proven treatment for this illness. In both an emotional and financial sense, AD patients' daily care and treatment are expensive. The medications that are currently available to treat AD are costly and merely aim to reduce the symptoms, which are always incurable. The cause of intermittent AD, which affects over 95 percent of individuals, is not fully known (6). Determining risk factors and preventing the emergence of sporadic AD are extremely challenging due to the lack of understanding on its pathogenesis. Good lifestyle choices and comorbidity management may reduce the risk of dementia, according to a recent study, even though avoiding or preventing modifiable risk factors may not have a complete effect on the disease's future development (7). Identification of possible risk factors may therefore help to lessen the load on AD patients.

Pathophysiology of Alzheimer's disease

The pathophysiology of Alzheimer's disease (AD) can be broadly defined as the progressive loss of brain tissue. Over the course of the disease, neurons die in a certain pattern. In addition to subcortical nuclei like the serotonergic dorsal raphe, noradrenergic locus coeruleus, and cholinergic basal nucleus, neuronal loss and/or pathology can be mostly observed in the entorhinal cortex, hippocampus, amygdala, and cortical association areas of the frontal, temporal, and parietal cortices (8). The loss of memory, especially short-term recollection, is one of the first symptoms of AD. Patients with mild to moderate AD suffer from more noticeable memory loss (such as trouble remembering familiar names and locations), a reduction in their capacity to think clearly (such as trouble preparing meals or balancing the cheque book), and changes in their mood and personality (9).

Risk factors of Alzheimer's disease

Some etiological ideas for the developments that have taken place have been published with reasonable to durable indications, based on neurological imaging techniques, epidemiological studies, and research on neuropathology.

1. Genetic risk factors

Neurodegenerative disease development and progression are facilitated by certain genetic risk factors that alter the transfer of pathogenic proteins (10). The lifetime risk of acquiring AD is higher for first-degree relatives of AD patients than for the general population. AD cases are more likely to occur in families and are influenced by a combination of hereditary and non-genetic variables. The existence of the apolipoprotein E (APOE) apoE4 allele may help with this risk in terms of genetic considerations (11).

2. Non-genetic risk factors

Several hereditary factors that cannot be changed have been studied in relation to neurological illnesses. Age is the most significant factors that responsible for the development of intermittent AD, according to population research (12). Furthermore, even among those who will not get dementia in their lifetime, neurodegenerative disease-related processes such as cognitive decline, cerebral shrinkage, white matter degradation, and neuropathological protein accumulation gradually appear throughout life. An increased chance of dementia, including Alzheimer's disease, is linked to vascular morbidity, including hypertension, diabetes mellitus, and asymptomatic cerebral infarction, as well as non-genetic risk factors include excessive total cholesterol levels, nicotine, and overweight.

3. Emerging risk factors

AD is widely recognized as a complicated neurodegenerative disease characterized by increased levels of external neuritic plaques and intracellular neurofibrillary tangles. Neuroinflammation can worsen the neurodegenerative process. There is mounting evidence that neurodegenerative illness and inflammasome activity are related (13). By disrupting function of lysosomes, compromising function of mitochondria, promoting misfolded protein dissemination, and possibly inducing an inflammatory reaction that ranges from causing acute lesions to accelerating distinct physiology of cells, such as, apoptosis, oxidative damage, and malfunctioning of proteins, heavy metals and pesticides harm cells. Inflammasomes may connect neuroinflammation and, eventually, cell death to cellular stress caused by environmental toxins (12).

Preventive strategies for Alzheimer’s disease

Wellness-focused prevention strategies are becoming more and more important for a long and healthy life. The focus on preventive and non-pharmacological treatments, particularly regarding the preliminary phase, is highlighted by an overview of recent research. Abeta-induced malfunctions in synapses and the enzymatic route blockage are examples of AD abnormalities that start early and worsen over decades, by which point the present treatments only slightly improve the condition. Diet as a preventive strategy can be useful in lowering cognitive disorders associated with aging and the danger of the preliminary phase AD because of the relevance of brain lipids development and functioning and the accessibility of determining lipids level can be modified by dietetic intercession (14).

1. Primary prevention of Alzheimer’s disease

Primary prevention strategies that address the non-genetic factors that contribute to the development of AD include measures to prevent obesity, high blood pressure, elevated glucose levels, middle-aged diabetes mellitus, heart failure, and extremely low blood pressure, which maintains adequate cerebral perfusion (15).

2. Secondary prevention of Alzheimer's disease

Even before classic clinical indications of AD appear, the brain undergoes gradual neurodegenerative alterations during a pre-clinical stage that lasts for several years. Even though certain diagnostic signs, neuroimaging biomarkers, and biochemical markers are accessible, it is challenging to detect this pre-clinical stage. In the preclinical stage of AD, neuroimaging techniques are thought to be a useful diagnostic tool (amyloid positron emission tomography can quantify beta amyloid in the brain in vivo) (14).

Current Treatment of Alzheimer's disease

The last ten years have seen the development of pharmacologic therapies for AD's main symptoms, memory loss and cognitive impairment.

- **Drug treatment**

To slow the advancement of cognitive, dementia's cognitive and psychological signs and symptoms, as well as the current drug therapies for AD are symptomatic rather than restorative. There are currently IV approved medicines on the market: galantamine, memantine, donepezil, and rivastigmine. The oral or transdermal routes are used to administer these medicines. These medications are used to regulate behavioral abnormalities, stabilize or improve cognitive skills, however briefly, and slow the disease's progression (16).

- **Nonpharmacological therapies**

Neurodegenerative illnesses can also be treated using non-pharmacological therapies in addition to medication. The multidomain intervention, which employs a variety of activities, is being studied in AD through a number of completed or ongoing international trials (7). Increased exercise, psychological instruction, healthier diet, and a slower extent of dementia and behavioral decline, based on the investigation. Due to the short duration of such endeavors, there is a dearth of data on ongoing study (16).

Future perspective

Numerous longitudinal studies have found a number of protective and risk factors for AD, some of which may be treated to lower the chance of AD or postpone its beginning; appropriate preventative measures may also help slow the disease's progression (17). The availability of treatments that modify the underlying pathology of AD will make early diagnosis more crucial, especially since it is anticipated that these therapies will be further successful in maintaining cognitive function and extending individuality when administered early in the course of the illness. With the approval of such medicines, the general public and non-specialists, including those working in primary care settings, will probably become more aware of cognitive impairment and other symptoms linked to AD. In contrast to community practice, this might motivate more patients and family members to seek assistance sooner in the course of their illness. Patients who are suspected of having early AD will be identified with the aid of more sensitive screening methods that proactively check for the presence of AD symptoms. Additionally, protecting the environment particularly against air pollutants would be crucial to preventing AD. Since daily preventions and neuroprotective interventions are expected to have higher positive impacts on people at high risk of AD or in the prodromal stage of AD, more research should, if at all possible, concentrate on these individuals (7).

Conclusion

Even after more than a century of research into AD, there is still no treatment that can stop, reduce, or inverse the neurodegenerative disease's development. Promising medicines for AD don't work out when tested on AD patients, despite the scientific community's enormous efforts to identify successful treatments. The issue of Alzheimer's disease is a significant public health concern. Since its exact cause is still unknown, research into every risk factor that could be involved in its development is essential. Genetic variables are the most significant from the perspective of early diagnosis and possible primary or secondary preventative application.

References

[1] Perrotta, G. (2020). General Overview of "Human Dementia Diseases": Definitions, Classifications, Neurobiological Profiles and Clinical Treatments. Gerontology & Geriatrics Studies, 6(1). https://doi.org/10.31031/ggs.2020.06.000626

[2] Porsteinsson, A. P., Isaacson, R. S., Knox, S., Sabbagh, M. N., & Rubino, I. (2021). Diagnosis of Early Alzheimer's Disease: Clinical Practice in 2021. Journal of Prevention of Alzheimer's Disease, 8(3), 371–386. https://doi.org/10.14283/jpad.2021.23

[3] Karantzoulis, S., & Galvin, J. E. (2011). Distinguishing Alzheimer's disease from other major forms of dementia. Expert Review of Neurotherapeutics, 11(11), 1579–1591. https://doi.org/10.1586/ern.11.155

[4] Ayodele, T., Rogaeva, E., Kurup, J. T., Beecham, G., & Reitz, C. (2021). Early-Onset Alzheimer's Disease: What Is Missing in Research? Current Neurology and Neuroscience Reports, 21(2). https://doi.org/10.1007/s11910-020-01090-y

[5] Lazaros, K., Gonidi, M., Kontara, N., Krokidis, M. G., Vrahatis, A. G., Exarchos, T., & Vlamos, P. (2024). Exploring the Association between Pro-Inflammation and the Early Diagnosis of Alzheimer's Disease in Buccal Cells Using Immunocytochemistry and Machine Learning Techniques. Applied Sciences (Switzerland), 14(18). https://doi.org/10.3390/app14188372

[6] Breijyeh, Z., & Karaman, R. (2020). Comprehensive Review on Alzheimer's Disease: Causes and Treatment. Molecules, 25(24). https://doi.org/10.3390/MOLECULES25245789

[7] Chen, X., Sun, G., Tian, E., Zhang, M., Davtyan, H., Beach, T. G., Reiman, E. M., Blurton-Jones, M., Holtzman, D. M., & Shi, Y. (2021). Modeling Sporadic Alzheimer's Disease in Human Brain Organoids under Serum Exposure. Advanced Science, 8(18), 1–16. https://doi.org/10.1002/advs.202101462

[8] Abubakar, M. B., Sanusi, K. O., Ugusman, A., Mohamed, W., Kamal, H., Ibrahim, N. H., Khoo, C. S., & Kumar, J. (2022). Alzheimer's Disease: An Update and Insights Into Pathophysiology. Frontiers in Aging Neuroscience, 14(March), 1–16. https://doi.org/10.3389/fnagi.2022.742408

[9] Mirakhori, F., Moafi, M., Milanifard, M., Asadi, A., & Tahernia, H. (2022). Diagnosis and Treatment Methods in Alzheimer's Patients Based on Modern Techniques: The Orginal Article. Journal of Pharmaceutical Negative Results, 13(S01), 1889–1907. https://doi.org/10.47750/pnr.2022.13.s01.226

[10] Boyd, R. J., Avramopoulos, D., Jantzie, L. L., & McCallion, A. S. (2022). Neuroinflammation represents a common theme amongst genetic and environmental risk factors for Alzheimer and Parkinson diseases. Journal of Neuroinflammation, 19(1), 1–20. https://doi.org/10.1186/s12974-022-02584-x

[11] Sierksma, A., Escott-Price, V., & De Strooper, B. (2020). Translating genetic risk of Alzheimer's disease into mechanistic insight and drug targets. Science, 370(6512), 61–66. https://doi.org/10.1126/science.abb8575

[12] Zhang, X. X., Tian, Y., Wang, Z. T., Ma, Y. H., Tan, L., & Yu, J. T. (2021). The Epidemiology of Alzheimer's Disease Modifiable Risk Factors and Prevention. Journal of Prevention of Alzheimer's Disease, 8(3), 313–321. https://doi.org/10.14283/jpad.2021.15

[13] Rahman, M. A., Rahman, M. S., Uddin, M. J., Mamum-Or-Rashid, A. N. M., Pang, M. G., & Rhim, H. (2020). Emerging risk of environmental factors: insight mechanisms of Alzheimer's diseases. Environmental Science and Pollution Research, 27(36), 44659–44672. https://doi.org/10.1007/s11356-020-08243-z

[14] De la Rosa, A., Olaso-Gonzalez, G., Arc-Chagnaud, C., Millan, F., Salvador-Pascual, A., García-Lucerga, C., Blasco-Lafarga, C., Garcia-Dominguez, E., Carretero, A., Correas, A. G., Viña, J., & Gomez-Cabrera, M. C. (2020). Physical exercise in the prevention and treatment of Alzheimer's disease. Journal of Sport and Health Science, 9(5), 394–404. https://doi.org/10.1016/j.jshs.2020.01.004

[15] Insel, P. S., Mormino, E. C., Aisen, P. S., Thompson, W. K., & Donohue, M. C. (2020). Neuroanatomical spread of amyloid β and tau in Alzheimer's disease: implications for primary prevention. Brain Communications, 2(1), 1–11. https://doi.org/10.1093/braincomms/fcaa007

[16] Pardo-Moreno, T., González-Acedo, A., Rivas-Domínguez, A., García-Morales, V., García-Cozar, F. J., Ramos-Rodríguez, J. J., & Melguizo-Rodríguez, L. (2022). Therapeutic Approach to Alzheimer's Disease: Current Treatments and New Perspectives. Pharmaceutics, 14(6), 1–20. https://doi.org/10.3390/pharmaceutics14061117

[17] Zhang, Z., Yang, X., Song, Y. Q., & Tu, J. (2021). Autophagy in Alzheimer's disease pathogenesis: Therapeutic potential and future perspectives. Ageing Research Reviews, 72(June), 101464. https://doi.org/10.1016/j.arr.2021.101464.