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Opinion

Alzheimer's Disease is a Type of Dementia

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OPINION

Alzheimer's disease is one of the most debilitating brain diseases that affect the elderly. It's an illness that's undertreated and underappreciated, but it's quickly becoming a big public health issue. Efforts to uncover the origin of the condition and create pharmacological treatments have progressively increased during the last decade. Improvements in clinical diagnostic standards and treatment of both cognitive and behavioural issues have been made in recent years. Symptomatic treatment, primarily cholinergic therapy, has been clinically tested in randomised, double-blind, placebo-controlled, parallel-group studies assessing performance-based cognitive functions. Cholinesterase inhibitors such as donepezil, tacrine, rivastigmine, and galantamine are advised for people with Alzheimer's disease who are experiencing cognitive difficulties. The role of oestrogen replacement therapy, anti-inflammatory drugs, and antioxidants is debatable and requires more research. Behavioral disturbances are treated with antidepressants, antipsychotics, mood stabilisers, anxiolytics, and hypnotics. Future directions in Alzheimer's disease research and treatment include: using functional brain imaging techniques in early diagnosis and treatment efficacy evaluation; developing new classes of medications that target different neurotransmitter systems (cholinergic, glutamatergic, etc), both for cognitive deficits and behavioural disturbances; and developing preventive methods (amyloid p-peptide immunisation). Because of increased life expectancy in the general population and a greater understanding of the disease's socioeconomic effects, Alzheimer's Disease (AD) has become a significant public health problem. Alzheimer's disease was first identified in 1906 by Alois Alzheimer, who used criteria such as gradual memory loss, disorientation, and pathology indicators to identify it (senile plaques and neurofibrillary tangles). Initially, Alzheimer's disease was thought to be a rare ailment, and later, it was thought to be a natural part of ageing. The stigma connected to ageing and other causes has stifled vigorous study into and treatment of persons with Alzheimer's disease, but these misunderstandings are dispelling, and medicines, albeit ineffective at first, are becoming available.

Senile plaques and neurofibrillary tangles appear to be the key neuropathological hallmarks of Alzheimer's disease. The senile plaques appear to form first in cognition-related parts of the brain, then extend to other cortical areas as the disease advances. Insoluble deposits of amyloid p-peptide (A), a portion of the amyloid precursor protein, are among the components of senile plaques (APP). Two cleavage events generate a peptide from APP: proteolytic activity by -secretase generates one end of the Ap peptide, while proteolytic activity by -secretase generates the other end. There appear to be two types of A: A42, which is a longer species, and A40, which is a shorter species. A42 appears to be deposited first, and it may play a role in triggering the mechanisms that lead to amyloid deposition. It's still unclear whether senile plaques are a cause or a symptom of Alzheimer's disease, despite mounting evidence suggesting a malfunction in APP metabolism, leading to a rise in insoluble A, is to blame for the disease. A seems to be harmful to neurons, either directly or indirectly, by inducing inflammation or boosting free radical generation. A second defining hallmark of Alzheimer's disease is the buildup of neurofibrillary tangles in neurons. Chemically altered tau protein, a protein involved in microtubule formation, is primarily responsible for neurofibrillary tangle formation. Tangle production is linked to disease severity; the more advanced the disease stage, the more tau tangles in the brain. Despite the existence of neurofibrillary tangles in AD, there have been no cases of AD caused by mutations in the tau gene on chromosome 174, while frontotemporal dementias with parkinsonism have been observed in some families with that mutation.

The majority of Alzheimer's patients suffer from a variety of dementia-related behavioural problems, with depression and psychosis being the most commonly investigated from a therapy standpoint. Depression in Alzheimer's patients should be treated aggressively, with cognitive function closely monitored. Due to a lack of clinical trial evidence, the treatment of depression in Alzheimer's disease is still empirical, consisting of starting an antidepressant at a low dose and gradually increasing it. In depressed patients without dementia, a sufficient dosage and therapy time are required for clinical response. Patients with dementia may take up to 6 weeks to respond to antidepressant medication, while patients with depression may take up to 6 weeks to respond to antidepressant treatment. Brofaromine and moclobemide, reversible monoamine oxidase inhibitors, appear to be beneficial in people with depression and dementia, without the severe potential side effects of traditional monoamine oxidase inhibitors (phenelzine, tranylcypromine).

Current dementia therapy options are based on varying degrees of scientific evidence, indicating a lack of understanding of the core

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biology of Alzheimer's disease. Cholinergic deficiencies have been well documented, and the evidence is strong enough to recommend cholin-esterase inhibitors (donepezil, tacrine, rivastigmine, and galantamine) as the first-line treatment for cognitive impairment in Alzheimer's disease patients. Randomized, double-blind, placebo-controlled, parallel-group studies evaluating performance-based assessments of cognitive function, activities of daily living, and behaviour have been used to evaluate symptomatic treatment, which mostly focuses on cholinergic therapy. Cholinesterase inhibitors may be used to address behavioural problems. Antioxidants, anti-inflammatories, and hormone replacement therapy are remaining contentious, despite the fact that clinical research investigating their efficacy is underway. Symptomatic treatment of behavioural disturbances includes antidepressants, antipsychotics, mood stabilisers, anxiolytics, and hypnotics.