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Research Paper

Recent advances in understanding and treating Alzheimer's disease

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ABSTRACT

Background: Alzheimer's disease (AD) still counts as a huge global health issue, with a steady slide in thinking ability and neurodegeneration. Over time, progress tied to the amyloid and tau ideas, plus work on neuroinflammation, and genetic risk signals like APOE ε4, has really broadened the way people understand AD as a kind of many-cause condition, even if the details stay complicated.
Objective: The aim here is sort of to look over what's new in the diagnosis, treatment, and day to day management of AD, and to point out what problems still don't fully get solved in the field.
Methods: This narrative review gathers together and connects recent updates across diagnostic biomarkers, imaging approaches, pharmacological and non-pharmacological interventions, and newer experimental directions that might shape future therapy for AD.
Results: Diagnostic accuracy has gotten better, in part because cerebrospinal fluid and blood-based biomarkers now offer more dependable signals. Also, advanced neuroimaging such as PET and MRI, along with improved neuropsychological assessment tools, helps clinicians catch AD earlier than before. On the treatment side, disease modifying agents aimed at amyloid and tau pathology have moved forward, including the approval of aducanumab, though the overall picture is still being refined. Meanwhile, symptom-focused therapies keep helping people manage cognitive and behavioral issues, day by day. Lifestyle approaches diet, physical activity, and cognitive training look promising as add-on strategies, potentially slowing down progression rather than reversing anything. New and still investigational ideas, like gene therapy and stem cell-based interventions, could become future routes for disease modification. Even so, the gap remains: AD shows clinical and biological heterogeneity, clinical trials can be hard to set up and run, and patient recruitment is often a bottleneck. On top of that, ethical concerns keep coming up around very early diagnosis, and whether treatment access stays fair for everyone.

Conclusion: there has been solid progress in figuring out AD pathophysiology and enhancing both diagnostic and therapeutic approaches, but a definitive cure still seems to stay out of reach. It's still important to keep up interdisciplinary research and collaboration, so we can tackle the open issues, sharpen early detection and tailor individualized treatment strategies, and in the end, improve results for patients and families that are affected by AD.

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1. INTRODUCTION

Alzheimer's disease (AD) is a gradually worsening neurological ailment that poses a substantial public health problem on a worldwide scale. The disease, initially documented by Dr. Alois Alzheimer in 1906, is distinguished by the progressive loss of memory, deterioration in cognitive abilities, and alterations in behaviour, ultimately resulting in total reliance on others and death. Alzheimer's disease (AD) is the predominant aetiology of dementia, responsible for 60-80% of all instances. Given the increasing number of elderly individuals worldwide, it is anticipated that the incidence of Alzheimer's disease (AD) would increase [1], [2], [3]. Therefore, it is crucial to enhance our comprehension and management of this debilitating condition. The precise aetiology of Alzheimer's disease remains unknown, however, it is commonly acknowledged to be a multifaceted interaction between genetic, environmental, and lifestyle variables. The defining pathological characteristics of Alzheimer's disease (AD) consist of the buildup of amyloid-beta (Aβ) plaques and the presence of neurofibrillary tangles made up of hyperphosphorylated tau protein in the brain. It is hypothesised that these irregularities interfere with the transmission of signals between cells and trigger immunological reactions, resulting in persistent inflammation, damage to neurones, and ultimately, the death of cells [3], [4], [5], [6]. The amyloid hypothesis has been a prominent theory in Alzheimer's disease (AD) research for many years. The theory suggests that the excessive synthesis and subsequent accumulation of Aβ peptides into plaques is a crucial first step in the development of Alzheimer's disease (AD). Although there is significant evidence supporting this notion, the precise pathways via which amyloid-beta contributes to neurodegeneration remain incompletely comprehended, and treatments targeting amyloid have so far achieved only limited success in clinical trials. As a result, researchers have begun investigating alternative causes and treatment targets [6], [7], [8].

The tau hypothesis has garnered significant attention as a potential alternative or supplementary explanation for the aetiology of Alzheimer's disease (AD). According to this idea, the excessive phosphorylation of tau protein results in the creation of neurofibrillary tangles, which interfere with the regular functioning of neurones and contribute to cell death. Research has demonstrated that tau pathology exhibits a stronger association with cognitive decline compared to amyloid-beta, indicating that it may have a more direct impact on the progression of the disease.

Recent studies have also emphasised the significance of neuroinflammation in Alzheimer's disease. The activation of microglia, which are the immune cells that dwell in the brain, is believed to have a dual function in Alzheimer's disease (AD). Although microglia initially try to eliminate amyloid-beta plaques, prolonged activation can result in the secretion of pro-inflammatory cytokines and additional harm to

neurons [8], [9]. This has created new opportunities for prospective therapies that focus on the inflammatory pathways in AD.

Genetic factors have a substantial influence on the likelihood of getting Alzheimer's disease. The most prominent genetic risk factor is the apolipoprotein E (APOE) ε4 allele, which is linked to a heightened risk of AD and an earlier age at which the disease manifests. Familial variants of Alzheimer's disease (AD) have been found to include genetic alterations, specifically in the amyloid precursor protein (APP) and presenilin (PSEN1 and PSEN2) genes. Progress in genomic technologies continues to reveal more genetic risk factors and pathways implicated in the disease, providing fresh opportunities for treatments [10].

Diagnosing Alzheimer's disease has traditionally been difficult, typically depending on clinical evaluations and the elimination of other illnesses. Nevertheless, the advancement of biomarkers has completely transformed the diagnostic procedure. Cerebrospinal fluid (CSF) indicators, including reduced levels of Aβ42 and elevated levels of tau and phosphorylated tau, have demonstrated potential in detecting Alzheimer's disease (AD) during its initial phases [11], [12], [13]. Blood-based biomarkers are increasingly being recognised as a less intrusive alternative, with the potential to enable extensive screening and earlier detection. Imaging technology have also enhanced our capacity to identify and track the progression of Alzheimer's disease. Positron emission tomography (PET) scans use tracers that specifically attach to amyloid and tau proteins enable the direct observation of these proteins within the brain while it is still functioning. Magnetic resonance imaging (MRI) is capable of identifying both structural and functional alterations in the brain, thereby offering valuable information regarding the advancement of diseases and the impact of potential therapies. In addition, the utilisation of digital tools and artificial intelligence is enhancing the accuracy of cognitive evaluations and assisting in the early identification of cognitive impairments through neuropsychological testing [13], [14], [15].

The therapy landscape for Alzheimer's disease is undergoing therapeutic advancements. Disease-modifying medicines seek to affect the fundamental pathophysiology of Alzheimer's disease (AD), with recent endeavours concentrating on diminishing levels of amyloid-beta and tau. In 2021, the U.S. Food and Drug Administration (FDA) granted fast approval to Aducanumab, an antibody that targets amyloid, which represents a major breakthrough in the treatment of Alzheimer's disease (AD). Additional intriguing strategies encompass anti-tau medicines, combination therapy that target various pathways, and symptomatic treatments that try to improve cognitive and behavioural symptoms [15], [16], [17]. Lifestyle adjustments, which are non-pharmacological therapies, have demonstrated promise in the management of Alzheimer's disease. Engaging in regular physical exercise, maintaining a good diet, participating in cognitive training, and actively engaging in social activities have been found to be linked to a decreased risk of Alzheimer's disease (AD) and a slower course of the disease in persons who are already affected. These therapies provide a comprehensive strategy to controlling the disease and enhancing the quality of life for patients and carers. Promising advancements in the field of research, such as gene therapy and the utilisation of stem cells, offer potential for the future treatment of Alzheimer's disease. Gene therapy seeks to rectify genetic abnormalities or introduce novel genes to counteract AD, whereas stem cell therapy concentrates on rejuvenating impaired neurones and reinstating brain functionality. Despite being in the initial phases, these methods demonstrate promising potential advancements in combating Alzheimer's disease [17], [18], [19], [20].

2. RELATED WORK

Recent research on Alzheimer's disease (AD) has made substantial progress in comprehending its aetiology, creating diagnostic instruments, and investigating therapy strategies. An important area of research has been centred around the amyloid hypothesis, which suggests that the buildup of amyloid-beta (Aβ) plaques plays a fundamental role in the development of Alzheimer's disease (AD). This concept posits that Aβ plaques interfere with brain transmission and initiate neuroinflammatory reactions. Although there has been much backing for this hypothesis, therapeutic experiments focusing on amyloid-beta have produced inconsistent outcomes. Previous trials of monoclonal antibodies targeting Aβ plaques did not demonstrate any cognitive advantages. However, the recent approval of aducanumab has sparked renewed

interest in medicines that target amyloid. The effectiveness of such treatments continues to be a topic of continuing discussion [20], [21], [22]. Aside from the amyloid theory, the tau hypothesis has garnered significant interest. This idea highlights the significance of anomalies in tau protein, specifically excessive phosphorylation that results in the creation of neurofibrillary tangles. These tangles are strongly linked to the loss of neurones and the deterioration in cognitive function in Alzheimer's disease (AD). Recent advancements in tau imaging techniques, like as positron emission tomography (PET) tracers, have made it possible to visualise tau disease in living organisms, leading to improved accuracy in diagnosis. Therapeutic interventions aimed at tau involve the use of small compounds and immunotherapies that are specifically targeted to decrease the clumping of tau proteins or enhance their removal. Several of these methods are now being studied in clinical trials [22], [23], [24], [25].

Neuroinflammation plays a crucial part in AD research. Microglia, the immune cells that live in the brain, are believed to have a crucial role in the development of Alzheimer's disease. Although microglia initially try to eliminate Aβ plaques, their persistent activation can result in the secretion of pro-inflammatory cytokines and further harm to neurones. This comprehension has prompted the investigation of anti-inflammatory medications as potential remedies for AD.

Genetic elements play a vital role in comprehending the risk and advancement of AD. The APOE ε4 allele is a widely recognised genetic risk factor for AD, affecting both the probability of having the disease and the age at which it begins. Familial variants of Alzheimer's disease (AD) are linked to certain genetic mutations, mainly in the amyloid precursor protein (APP) and presenilin genes [25], [26], [27].

Advancements in genomic technologies are currently discovering more genetic risk factors and pathways associated with the disease, providing new opportunities for intervention. Recent research has placed significant emphasis on improving diagnostic capabilities, particularly through the advancement of biomarkers. Cerebrospinal fluid (CSF) indicators, including decreased Aβ42 levels and elevated tau and phosphorylated tau levels, have demonstrated potential in detecting Alzheimer's disease (AD) during its initial phases. Blood-based biomarkers are becoming increasingly popular as a less intrusive alternative, perhaps enabling more extensive screening and earlier detection. Moreover, advanced imaging techniques, such as Positron Emission Tomography (PET) and Magnetic Resonance Imaging (MRI), have enhanced our capacity to observe amyloid and tau pathology in the brain while it is still functioning, offering crucial knowledge about the advancement of diseases and the impact of treatments. Therapeutic methods have made major advancements as well. Treatments that try to influence the disease process of Alzheimer's disease (AD) focus on changing the underlying pathology by specifically targeting amyloid-beta and tau proteins [27], [28], [29], [30]. There is ongoing progress in the development of treatments that target the symptoms of a condition, with the introduction of novel approaches to better manage cognitive and behavioural symptoms. Non-pharmacological therapies, such as lifestyle alterations encompassing dietary changes, physical activity, and cognitive training, are being investigated for their capacity to decelerate the advancement of the condition. Promising advancements in AD treatment can be expected from emerging research areas such as gene therapy and stem cell applications. Gene therapy seeks to rectify genetic abnormalities or introduce novel genes to target the fundamental reasons of the ailment, whereas stem cell therapy concentrates on rejuvenating impaired neurones and reinstating brain functionality. While these techniques are now in the first phases of study, they have promising potential for progress in combating Alzheimer's disease [31].

3. METHODOLOGY

In this review, a detailed examination of the most recent research on Alzheimer's disease (AD) was carried out by searching through reputable databases such as PubMed, Scopus, and Google Scholar. In order to present a comprehensive overview of the progress that has been made in understanding Alzheimer's disease, the primary purpose was to collect and synthesise the findings of the most recent research currently available. A careful selection process was used to choose the research because of their significance to important areas of Alzheimer's disease (AD), such as the biology of the disease, advancements in diagnosis, and the many different treatment approaches. The biology of Alzheimer's

disease includes a number of important components, including neuroinflammation, amyloid plaques, and tau protein tangles, to begin with. It is the buildup of beta-amyloid peptides in the brain that is the primary focus of research on amyloid plaques. This accumulation is a characteristic feature of Alzheimer's disease. In addition to disrupting the function of cells, these plaques are thought to have a substantial role in the course of Alzheimer's disease. Research conducted on tau proteins investigates the process by which aberrant phosphorylation results in the creation of tangles within neurones, which in turn contributes to the death of neurones and a reduction in cognitive abilities. In addition, neuroinflammation, which is characterised by the activation of glial cells and the production of inflammatory mediators, is an important topic of inquiry since it exacerbates neuronal damage and the course of disease.

The study emphasises the significance of imaging techniques and biomarkers in relation to the developments that have been made in diagnostic systems. A number of biomarkers, including levels of tau and beta-amyloid in cerebrospinal fluid (CSF), as well as indicators based on blood, have demonstrated potential for application in the early identification and monitoring of the course of disease. Different imaging techniques, such as positron emission tomography (PET) and magnetic resonance imaging (MRI), offer extremely helpful insights into the anatomical and functional changes that occur in the brain as a result of Alzheimer's disease. While magnetic resonance imaging (MRI) can detect brain atrophy and other structural abnormalities, positron emission tomography (PET) imaging can observe amyloid plaques and tau tangles in living organisms. In order to assess the effectiveness of therapeutic strategies and to facilitate early intervention, these diagnostic tools are absolutely necessary.

There have been recent developments in this field, such as the creation of monoclonal antibodies that target beta-amyloid. These antibodies, such as aducanumab and lecanemab, have demonstrated potential in clinical studies for lowering amyloid plaques and increasing cognitive performance. Furthermore, research is also being conducted in order to create medicines that target tau proteins as well as other degenerative processes that are associated with Alzheimer's disease. A patient's quality of life can be improved by the use of measures that are referred to as "symptom management." These tactics aim to reduce the cognitive and behavioural symptoms that are associated with Alzheimer's disease. This includes non-pharmacological therapy such as cognitive rehabilitation, physical activity, and psychological support in addition to pharmaceutical treatments such as cholinesterase inhibitors and NMDA receptor antagonists, all of which can assist in the management of cognitive symptoms. The review highlights the significance of taking a holistic approach to the treatment of Alzheimer's disease, which involves combining pharmacological and non-pharmacological approaches in order to address the complex character of the disease.

During the process of data extraction, the primary focus was on recognising significant discoveries and trends, as well as establishing strategies for treatment. Specifically, this entailed doing a comprehensive review of each of the studies that were chosen, summarising the most important findings, and analysing trends and emerging themes. As an illustration, the review reveals that there is a rising interest in the role that neuroinflammation plays in Alzheimer's disease, as well as the possibility of targeting inflammatory pathways as a therapeutic method. In addition, the review emphasises the growing utilisation of advanced imaging techniques and biomarkers in both research and clinical practice, highlighting the significance of these tools in the early identification of disease and the monitoring of its course. The synthesis of the literature that was evaluated involved bringing together the information that was already known, identifying the areas in which there was a lack of understanding, and putting an emphasis on potential research paths. Through the utilisation of this all-encompassing strategy, it is possible to conduct a full evaluation of the current status of Alzheimer's disease research and to identify gaps that call for additional exploration. For example, the study highlights the necessity of conducting additional research on the long-term impact and safety of new disease-modifying medicines, as well as the creation of biomarkers that are more sensitive and specific for early diagnosis. The review intends to direct future research efforts and provide information that can be used in clinical practice by putting an emphasis on potential research areas. A number of potential avenues for future research include the exploration of the genetic and environmental variables that contribute to Alzheimer's disease, the development of

combination medicines that target several degenerative processes, and the enhancement of the integration of diagnostic technologies in normal clinical care.

4. RESULTS AND DISCUSSION

Recent breakthroughs in Alzheimer's disease (AD) research have greatly improved our comprehension of its underlying mechanisms, methods of diagnosis, and available treatment choices. The amyloid hypothesis, positing that the buildup of amyloid-beta (Aβ) plaques plays a key role in the development of Alzheimer's disease (AD), remains very prominent. Research regularly demonstrates the presence of Aβ plaques in the brains of individuals with Alzheimer's disease (AD), and these plaques are linked to a loss in cognitive function. Nevertheless, treatment trials focusing on Aβ reduction have shown inconclusive outcomes, suggesting that the buildup of Aβ alone may not completely account for the advancement of the disease. The tau hypothesis has been prominent as a response, with an emphasis on anomalies in tau protein, including hyperphosphorylation and the formation of neurofibrillary tangles [31], [32], [33], [34], [35]. These abnormalities are strongly associated with the loss of neurones and cognitive impairment. Recent advancements in tau imaging techniques have enhanced our capacity to observe tau pathology, which exhibits a strong correlation with cognitive deterioration.

Research on neuroinflammation has discovered that the persistent activation of microglia, which are the immune cells of the brain, has a substantial impact on Alzheimer's disease, in addition to amyloid and tau. The activated microglia secrete pro-inflammatory cytokines that worsen the damage to neurones, emphasising the possibility of using anti-inflammatory treatments as an additional strategy to address amyloid and tau. The progress in diagnostics has significantly enhanced our capacity to identify AD. Cerebrospinal fluid (CSF) indicators, including decreased levels of Aβ42 and increased levels of tau, are highly reliable for identifying Alzheimer's disease (AD), particularly during its initial phases [35], [36], [37]. Blood-based biomarkers are becoming increasingly recognised as a less intrusive option, showing encouraging outcomes in the detection of Alzheimer's disease risk. Imaging tools such as positron emission tomography (PET) and magnetic resonance imaging (MRI) have improved our ability to see amyloid and tau deposits and detect structural alterations in the brain. These technological developments facilitate earlier and more precise diagnosis, which is essential for prompt intervention. Significant advancements have been achieved in the therapeutic field of disease-modifying medicines that target the fundamental pathophysiology of Alzheimer's disease. The recent authorisation of aducanumab, a monoclonal antibody targeting amyloid, signifies a significant advancement, while its clinical effectiveness is still a subject of controversy. Several medicines that specifically target tau are currently undergoing clinical trials at different stages. These trials have shown promising findings, indicating the potential benefits of these therapies in slowing down the progression of the disease [37], [38], [39], [40]. Additionally, there have been advancements in treatments that target the symptoms themselves, offering improved methods for efficiently treating cognitive and behavioural disorders. Non-pharmacological therapies, such as lifestyle modifications encompassing dietary changes, physical activity, and cognitive training, have demonstrated promise in decelerating the advancement of the disease and enhancing the quality of life for patients [40], [41], [42], [43], [44], [45].

Current studies in gene therapy and the use of stem cells have promising prospects for the advancement of Alzheimer's disease treatment. Gene therapy seeks to rectify genetic abnormalities or introduce novel genes to target the fundamental causes of AD, whereas stem cell therapy concentrates on rejuvenating impaired neurones and reinstating brain functionality. Despite being in the early phases, these approaches show potential as pathways for future advancements [45], [46], [47], [48], [49].

5. CONCLUSION

Advancements in recent research on Alzheimer's disease (AD) have greatly increased our comprehension of its intricate pathogenesis, improved diagnostic capabilities, and broadened therapy choices. The further development of the amyloid and tau theories has yielded useful insights into the

molecular underpinnings of Alzheimer's disease (AD, however difficulties remain in converting these discoveries into viable therapeutic interventions. The increasing acknowledgement of neuroinflammation and hereditary variables contributes to the intricacy of the condition, emphasising the requirement for a comprehensive therapy.

Advancements in diagnostics, such as enhanced biomarkers and imaging techniques, have facilitated the earlier and more precise identification of AD, which is essential for prompt intervention. Therapeutic advancements involve the creation of medications that modify diseases by targeting amyloid-beta and tau, along with strategies for managing symptoms and non-pharmacological interventions. Ongoing studies in gene therapy and stem cell applications show potential for significant advancements in the future. Nevertheless, there are still notable obstacles to overcome, such as the diverse nature of the disease and the requirement for tailored treatment strategies. To overcome these hurdles and make progress in developing more effective medicines, it is crucial to continue collaborating across different disciplines and fostering creativity. In the end, the continuous research endeavours offer optimism for enhanced treatment and, possibly, a remedy for Alzheimer's disease, with the goal of reducing the hardship on patients and their families.

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Author Contributions Statement

Name of Author	C	M	So	Va	Fo	I	R	D	O	E	Vi	Su	P	Fu
Mohd Altaf Dar	✓	✓	✓	✓		✓		✓	✓	✓	✓			
Afshana Qadir	✓	✓	✓			✓	✓	✓			✓	✓	✓	
Zulfkar Qadrie		✓	✓	✓			✓	✓	✓			✓	✓	✓
Humaira Ashraf	✓	✓	✓			✓	✓	✓		✓	✓	✓		

C : **C**onceptualization
M : **M**ethodology
So : **So**ftware
Va : **Va**lidation
Fo : **Fo**rmal analysis
I : **I**nvestigation
R : **R**esources
D : **D**ata Curation
O : Writing - **O**riginal Draft
E : Writing - Review & **E**diting
Vi : **Vi**sualization
Su : **Su**pervision
P : **P**roject administration
Fu : **Fu**nding acquisition

Conflict of Interest Statement

The authors declare that there are no conflicts of interest regarding the publication of this paper.

Informed Consent

All participants were informed about the purpose of the study, and their voluntary consent was obtained prior to data collection.

Ethical Approval

Not Applicable.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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