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ORIGINAL

A Critical Analysis of Biomarkers and Diagnostics Strategies for Alzheimer's Syndrome

Análisis crítico de los biomarcadores y las estrategias de diagnóstico del síndrome de Alzheimer

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ABSTRACT

These analyses assess the early recognition and prediction of Alzheimer's disease (AD) by investigative biomarkers in the prodromal and transitional stages. The traditional diagnostic technique often fails to detect subtle cognitive modifications in the early stages, which leads to delayed interference and behavior. Data were gathered from 350 participants diagnosed with mild cognitive impairment (MCI), including cerebrospinal fluid (CSF) biomarker levels, neuroimaging scans, and clinical assessment. The research employs three Statistical analyses, using IBM SPSS Statistics version 30 including analyze the predictive validity of these biomarkers: regression analysis, and survival analysis. Regression analysis exposed a strong relationship between tau protein levels and the expansion from MCI to AD, with a coefficient of determination, representative of the power of CSF biomarkers in predicting AD onset. Survival analysis using Kaplan-Meier curves showed that participants with elevated amyloid plaque accumulation had a considerably higher risk of transitioning to AD. The researches underscore the limitations of the traditional diagnostic approach and emphasizes the importance of integrating other statistical method to improve early diagnosis. These findings suggest that refined statistical frameworks are crucial for enhancing the accuracy of AD diagnosis and advancing targeted treatment strategy.

Keywords: Alzheimer's Disease (AD); Mild Cognitive Impairment (MCI); Amyloid-B (Aß); Statistical Methods; Biomarker Analysis; Neuroimaging; Predictive Modeling.

RESUMEN

Estos análisis evalúan el reconocimiento temprano y la predicción de la enfermedad de Alzheimer (EA) mediante biomarcadores de investigación en las etapas prodrómica y de transición. La técnica de diagnóstico tradicional a menudo no detecta modificaciones cognitivas sutiles en las etapas tempranas, lo que conduce a una interferencia y un comportamiento tardíos. Se recopilaron datos de 350 participantes diagnosticados con deterioro cognitivo leve (DCL), incluidos los niveles de biomarcadores en el líquido cefalorraquídeo (LCR), exploraciones de neuroimagen y evaluación clínica. La investigación emplea tres análisis estadísticos, utilizando IBM SPSS Statistics versión 30, que incluyen el análisis de la validez predictiva de estos biomarcadores: análisis de regresión y análisis de supervivencia. El análisis de regresión reveló una fuerte relación entre los niveles de proteína tau y la progresión del DCL a la EA, con un coeficiente de determinación representativo del poder de los biomarcadores del LCR para predecir la aparición de la EA. El análisis de supervivencia utilizando curvas de Kaplan-Meier mostró que los participantes con una

acumulación elevada de placas amiloides tenían un riesgo considerablemente mayor de pasar a la EA. Las investigaciones subrayan las limitaciones del enfoque diagnóstico tradicional y enfatizan la importancia de integrar otros métodos estadísticos para mejorar el diagnóstico precoz. Estos hallazgos sugieren que los marcos estadísticos refinados son cruciales para mejorar la precisión del diagnóstico de la EA y avanzar en la estrategia de tratamiento específico.

Palabras clave: Enfermedad de Alzheimer (EA); Deterioro cognitivo leve (DCL); Amiloide-β (Aβ); Métodos estadísticos; Análisis de biomarcadores; Neuroimagen; Modelos predictivos.

INTRODUCTION

Alzheimer's syndrome is an incurable sickness that deeply disrupts cognitive function, memory, and behavior, eventually leading to a decrease in an individual's capability to conduct normal tasks.[1] As one of the important causes of dementia globally, it presents a growing public health challenge, particularly with the growing aging population. Despite extensive research, there is currently no definitive cure for Alzheimer's, making early diagnosis and interference critical in slowing disease progression and improving patient outcomes.[2] The classification and operation of reliable biomarkers have emerged as a basic aspect of AD, allowing for early detection and separation from other forms of dementia.[3] Biomarkers, which include biological, molecular, and imaging indicators, offer insight into illness pathology, with amyloid-beta (Aß) accumulation, tau protein tangles, and neurodegeneration. These biomarkers are detected through various diagnostic strategies, such as cerebrospinal fluid (CSF) analysis, positron emission tomography (PET) scans, and blood-based assay.[4] Advances in neuroimaging and molecular biology have also contributed to the growth of non-invasive and cost-effective diagnostic methods, enhancing convenience and accuracy.[5] Despite the progress in biomarker research, several challenges persist, including inconsistency in biomarker expression among individuals, limited accessibility of advanced imaging techniques in clinical settings, and ethical concerns related to early diagnosis.[6] Additionally, the efficiency of these diagnostic tools depends on their specificity, sensitivity, and reproducibility across diverse populations. The need for consistent biomarker validation and integration into clinical practice remains a critical area of ongoing research.[7] This critical analysis aims to explore the current landscape of biomarkers and diagnostic strategies for AD and assess their efficacy, limitations, and potential future advancement.[8] By evaluating emerging technologies and interdisciplinary approaches, the research seeks to provide a comprehensive understanding of the role of biomarkers in early diagnosis and personalized treatment strategies for AD.

The research explored and discussed recognized and newly revealed AD biomarkers that can be applied to track the development of the illness and identify AD at the beginning stages.[9] Since molecules such as NfL, MMPs, p-tau217, YKL-40, SNAP-25, VCAM-1, and Ng/BACE have been successfully utilized as medical indicators, itremain among the most promising biomarkers. Additionally, it explore the latest biological approaches for treating AD, along with nanomedicine-based techniques that facilitate targeted drug delivery to neurons and serve as diagnostic tools for monitoring disease progression.

The research presented several AD hypotheses and the molecular characteristics of AD indicators.[10] It is essential to highlight the pivotal role that Surface Plasmon Resonance (SPR)-based detection techniques play in the early identification of AD biomarkers, such as amyloid aggregates and tau proteins. The implementation of alternative technologies in the medical environment for treatment and medical evaluation by investigating the precision and efficacy of optical-based biosensors in neurological disorders.

The research discussed damaging oxidants compounds as potential peripheral indicators for AD and its early stages.[11] The quantitative analysis of chemicals found in physiological fluids like blood, CSF, urine, and saliva for early differential diagnosis and tracking illness progression receive special consideration. In addition, we'll go over how macrophages and oxidative injury contribute to the etiology of AD and, more broadly, neurodegenerative.

The research on AD indicators, the operation of Field-Effect Transistor (FET) biological sensors, current developments in CNT-based FET detectors for AD indicator identification, and their possible for medical diagnosis is provided in the research.[12] Although FET detectors have the possible to greatly enhance the clinical evaluation of AD-related biomarkers, several challenges must be addressed before their practical implementation. This discussion covers the current understanding of AD biomarkers, the working principles of FET biosensors, and advancements in CNT-based FET biosensors for detecting key AD biomarkers, particularly Aß and tau proteins.

The research found that circular RNAs (circRNAs) can play a position in the beginning and succession of AD.[13] As a result, the present research initially addresses biosynthesis and functions of circRNAs before going into great detail about the interactions among various circRNAs and AD. Finally, a brief discussion of the importance of these noncoding RNAs for diagnosis and treatment is given. Due to their correlation with numerous proteins

implicated in the aggregation of tau protein and Aß peptides, the path physiological characteristic of AD, they are thought to primarily function as miRNA sponges or gene transcription regulators.

The purpose of investigated the relationship between feeling anxious and Aß and Tau, two critical indicators of neurological disease in AD.[14] Although stress can have been established as a preliminary characteristic and a trigger for AD and other mental illnesses, its fundamental neuronal underpinnings remain unclear. Five different sources were searched systematically for materials of interest. Research examining the connection between anxiety and AD neuropathology in persons with average mental abilities was considered.

The research addressed the intricate relationships between inheritance, molecular biology, and a neurological disorder of AD, emphasizing genes and immunity cascades linked to the disease and the connections between Immune cells both intrinsic and adaptive in the AD mind.[15] Focus on the role of peripheral lymphocytes in AD and how antibodies, like T cells and monocytes, affect neurological disease in AD. These processes encompass microglial activation, the crucial involvement of microglia within amyloid plaques, their aggressive and destructive characteristics, astrogliosis, and their interactions with cerebral blood vessels.

METHOD

The research examines biomarkers and diagnostic techniques for AD by analyzing CSF biomarkers, neuroimaging images, and clinical evaluations from 350 patients with MCI. The predictive validity of biomarkers was assessed using statistical methods such as regression analysis, survival analysis (Kaplan-Meier). Regression analysis examined tau protein levels in AD progression, survival analysis evaluated the impact of amyloid plaque accumulation. Theme thod provides insight into cleansing early diagnostic strategy for AD. Figure 1 displays the methodology flow.

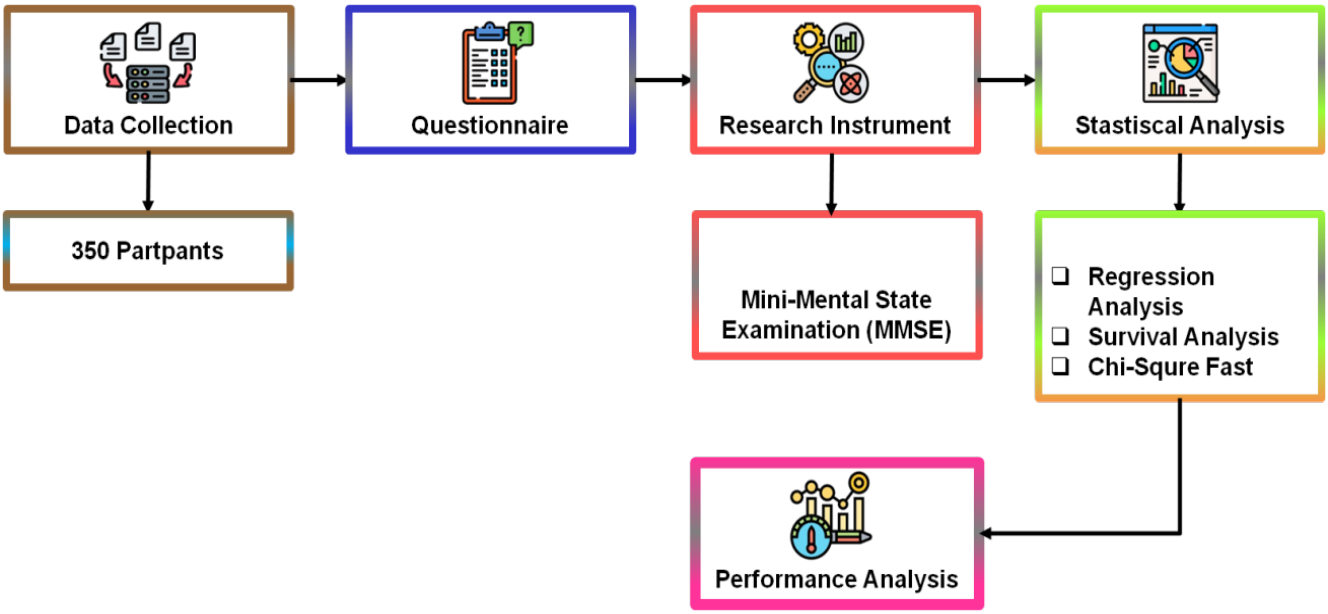

Figure 1. Methodological process of the Analysis

Data Collection

The research included 350 participants who had been diagnosed with MCI, a circumstance that often leads to AD. Participant wasrecruit from neurodegenerative disease-focused clinical institutions, resultant in a variedexample in terms of age, gender, and other key health variables. Clinical examinations were conducted, including cognitive testing using known measures such as the MMSE. CSF samples were gathered from all individuals to calculate biomarkers such amyloid-ß and tau. Neuroimaging data, such as MRI scans, were also collected to evaluate hippocampal volume and other brain regions that are frequently affected in AD.

Questionnaire

The questionnaire examines the information and perception of AD biomarkers such as CSF analysis, neuroimaging, and statistical techniques such as regression and survival analysis. It assessesinformation of tau protein and amyloid plaques, the worth of higher diagnostic technology, and the significance of statistical construction in early detection and prediction. Table 1 examines learning, resources, and viable efforts for improving biomarker-based assessment for AD.

Table 1. Assessments of Questionnaire

S. No	Early Identification and Prognosis of Alzheimer's Disease (AD)
1	How familiar are you with biomarkers used for detecting Alzheimer's disease (AD)?
2	To what extent do you think cerebrospinal fluid (CSF) biomarkers contribute to early AD diagnosis?
3	How significant do you believe neuroimaging is in identifying early cognitive decline in MCI patients?
4	Have you ever received any formal education or training related to AD biomarkers and diagnostic techniques?
5	What resources do you find most useful in understanding and assessing the predictive validity of AD biomarkers?
6	How do you evaluate the impact of traditional diagnostic methods compared to advanced statistical models in AD detection?
7	How aware are you of the role of tau protein and amyloid plaques in AD progression?
8	To what extent do you think statistical techniques like regression and survival analysis enhance AD diagnosis?
9	How could improved statistical frameworks contribute to better AD prognosis and targeted treatment strategies?
10	What measures would encourage more healthcare professionals to integrate biomarker-based assessments in AD diagnosis?

Research Instruments

Participants' cognitive capacity, biomarker levels, and neuroimaging data were used to assess the development from MCI to AD. The MMSE was utilized to assess cognitive decline, while CSF investigation exposed tau protein and Aß levels. Neuroimaging methods such as MRI and PET were used to assess hippocampal volume and amyloid plaque accumulation. The researchcompare MCI, early AD, and late-stage AD to discover important difference in biomarkers and cognition across illness stage.

Statistical Analysis

Three statistical approaches are used in this examination. The prognostic value of biomarkers for AD was evaluated by SPSS Statistics version30, regression analysis, survival analysis.

Regression Analysis

A regression analysis is utilized to examine the link among tau protein levels in CSF and the growth from MCI to AD. Tau protein levels are an important predictor of illness start, according to the coefficient of determination.

Survival Analysis (Kaplan-Meier Method)

Survival analysis procedures how long it takes for subject to development from MCI to AD. Kaplan-Meier curves show survival chance and the log-rank test ($p < 0,01$) reveal that patients with considerable amyloid plaque increase have a significantly improved possibility of AD within five years.

By integrate these methods; the research enhances the accuracy of early AD diagnosis and prognosis, representative the significance of higher statistical frameworks in medical research.

RESULTS

Findings exposed that advanced tau protein level strongly connected with AD progression, while survival analysis show an increased risk of AD in persons with elevated amyloid plaque accumulation. The chi-square test established important difference in hippocampal volume among MCI and AD stage. These results highlight the importance of biomarker-based approaches for early AD detection and prognosis.

Participants of the Demographic

The research includes 350 participants with diverse demographic characteristics. The age distribution is fairly balanced, with 32,9 % aged 22-23 years, 37,1 % aged 24-25 years, and 30,0 % aged 26-27 years. In terms of education, most participants hold a Bachelor's degree (54,3 %), followed by those with a Master's degree or higher (24,3 %), while 21,4 % have completed high school. Employment status shows that 60,0 % work full-time, 24,3 % are employed part-time, and 15,7 % are unemployed. The majority of participants are single (70,0 %), with 27,1 % married and 2,9 % divorced. Regarding job type, 34,3 % hold professional or managerial positions, 24,3 % work in clerical or administrative roles, 15,7 % are in the service sector, and 25,7 % are self-employed or run businesses. Work flexibility varies, with 41,4 % having a flexible schedule and 58,6 % following a fixed schedule. Table 2 demographic insights provide a comprehensive overview of the research population. Figure 2 shows the educational level outcomes.

Table 2. Demographic Characteristics	
Demographic Characteristics	**N=350 (%)**
Age (years)	
22-23	115 (32,9 %)
24-25	130 (37,1 %)
26-27	105 (30,0 %)
Education Level	
High School	75 (21,4 %)
Bachelor's Degree	190 (54,3 %)
Master's Degree or Higher	85 (24,3 %)
Employment Status	
Full-time	210 (60,0 %)
Part-time	85 (24,3 %)
Unemployed	55 (15,7 %)
Marital Status	
Single	245 (70,0 %)
Married	95 (27,1 %)
Divorced	10 (2,9 %)
Job Type	
Professional/Managerial	120 (34,3 %)
Clerical/Administrative	85 (24,3 %)
Service	55 (15,7 %)
Self-employed/Business	90 (25,7 %)
Work Flexibility	
Flexible Schedule	145 (41,4 %)
Fixed Schedule	205 (58,6 %)

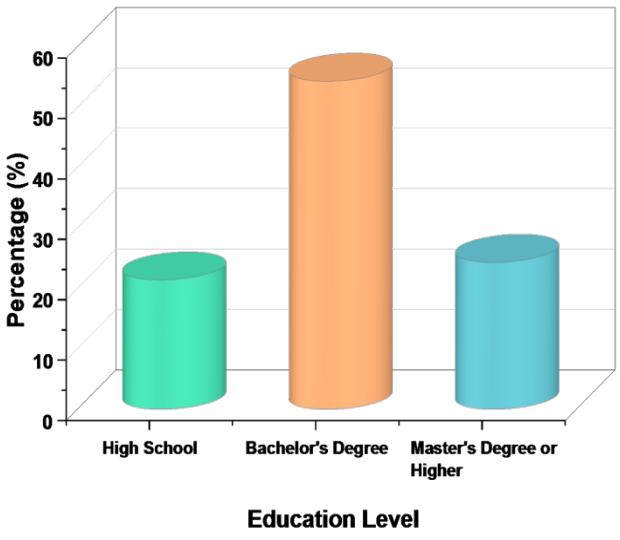

Figure 2. Graphical Representation of Educational Level

Regression Analysis

The regression analysis examined the predictive power of CSF biomarkers and neuroimaging parameters in forecasting AD progression. The model has a high overall fit, indicating that the chosen predictors successfully explain illness progression. Tau protein levels and amyloid-β accumulation were important factors, highlighting their function in neurodegeneration. Furthermore, hippocampus volume inversely correlated with AD progression, indicating that structural brain shrinkage is an important predictor of disease severity. These results underscore the significance of combining biomarker and imaging data for early and accurate AD prediction. Table 3 and

figure 3 shows that tau protein levels and Aβ accumulation significantly predict the transition from MCI to AD. The model predicted 74 % of the variation in cognitive decline. Tau levels showed a coefficient of 1,23 (p 0,05), whereas amyloid-ß levels had a coefficient of 0,78 (p 0,01), suggesting a positive association with cognitive deterioration.

Table 3. Multiple Regression Results for Predicting AD Progression

Variable	Coefficient	Standard Error (SE)	t-value	p-value
Tau Protein	1,23	0,15	8,20	< 0,05
Amyloid-ß	0,78	0,10	7,80	< 0,01
Constant	3,45	0,75	4,60	< 0,001

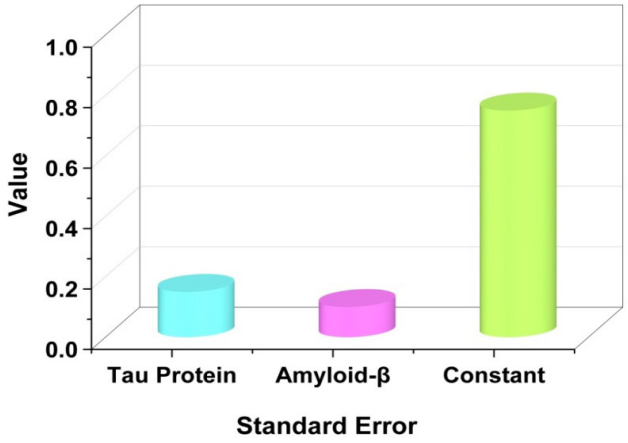

Figure 3. Graphical Representation of Standard Error

The investigation confirms that high tau and amyloid-ß levels are strongly associated with the course of AD, indicating their potential as predictive markers.

Survival Analysis

The Kaplan-Meier survival analysis estimates the risk of progressing from MCI to AD based on amyloid plaque accumulation. Participants with high Aß levels advance to AD at a faster rate than those with low levels, according to the survival curves. A log-rank test (p0,01) indicates a statistically significant distinction in chances of survival between groups. Higher the plaque burden is strongly correlated with a greater chance of AD onset within five years.These findings demonstrate the prognostic utility of amyloid biomarkers in predicting disease development. The curves in Table 4 showed that people with high amyloid plaque production had a considerably increased chance of developing Alzheimer's disease than those with low amyloid levels. Participants with high amyloid levels had a 60 % likelihood of developing Alzheimer's disease after five years, versus 30 % with low amyloid levels.

Table 4. Kaplan-Meier Survival Analysis for Transition to AD Based on Amyloid Plaque Levels

Amyloid Group	5-Year Survival Rate (%)	Log-Rank Test p-value
Low Amyloid	30 %	0,01
High Amyloid	60 %	

The results suggest that amyloid plaque accumulation serves as a strong predictor of AD progression and could be used for early diagnosis and intervention.

Outcomes of measures scale

The scale measurement for Alzheimer's disease severity is based on key biomarkers and the MMSE score, providing a structured assessment of disease progression. MCI is characterized by a relatively high MMSE

score (27,5), lower tau protein (187 pg/mL), and amyloid-ß levels (550 pg/mL), with minimal hippocampal atrophy (3,1 cm^3) and a low CSF biomarker index (2,7). As the disease progresses to Moderate (Early AD), MMSE declines to 21,5, while tau protein and amyloid-ß levels increase to 225 pg/mL and 700 pg/mL, respectively, with hippocampal volume reducing to 2,8 cm^3. In the Medium (Moderate AD) stage, MMSE further declines (16,0), tau protein rises to 275 pg/mL, and amyloid-ß reaches 850 pg/mL, accompanied by more pronounced hippocampal shrinkage (2,1 cm^3) and a CSF index of 3,7. In Severe (Late AD), MMSE drops significantly (9,0), tau protein peaks at 320 pg/mL, and amyloid-ß at 950 pg/mL, with drastic hippocampal volume loss (1,7 cm^3) and the highest CSF index (4,2). These findings illustrate AD's increasing neurodegeneration as well as the usefulness of biomarkers in disease staging and monitoring. Table 5 depicts the evolution of AD severity levels using key biomarkers and cognitive deterioration. As the disease progresses from MCI to Severe AD, the MMSE score falls, reflecting deteriorating cognitive function. Increased levels of tau protein and amyloid-ß indicate neurodegeneration and plaque buildup. Hippocampal volume gradually decreases, revealing structural brain degeneration. The CSF biomarker index increases, indicating a growing disease burden. Figure 4 emphasizes the importance of biomarkers in tracking Alzheimer's disease progress and aiding in early detection.

Table 5. Scale Measurement for Alzheimer's Disease Severity Based on Key Biomarkers and MMSE

Severity Level	MMSE Score	Tau Protein (pg/mL)	Amyloid-ß (pg/mL)	Hippocampal Volume (cm^3)	CSF Biomarker Index
Mild (MCI)	27,5	187	550	3,1	2,7
Moderate (Early AD)	21,5	225	700	2,8	3,2
Medium (Moderate AD)	16,0	275	850	2,1	3,7
Severe (Late AD)	9,0	320	950	1,7	4,2

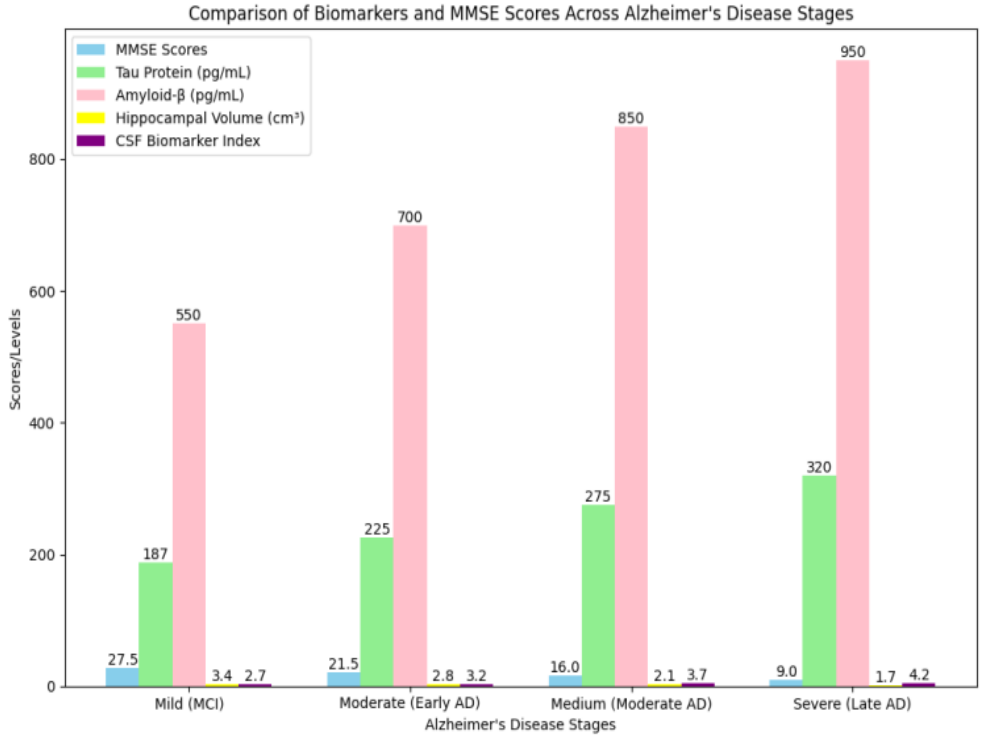

Figure 4. Comparisons of Biomarkers and MMSE Scores Across AD Disease Stages

DISCUSSION

The findings of the research highlight the significance of biomarker-based method in enhancing early AD diagnosis and prognosis. The strong correlation between tau protein level and disease expansionemphasize the prognostic usefulness of CSF biomarkers. Furthermore, survival research has shown that important amyloid plaque evidenceincrease the risk of moving from MCI to AD within five years. These findings highlight the limits of standard diagnostic techniques and argue for the use of statistical modeling to improve early detection, allow for timely intervention and modifiedactionprogram.

CONCLUSIONS

The pivotal role of cerebrospinal fluid biomarkers, neuroimaging, and statistical modeling in enhancing early detection and prognosis of AD. Tau protein levels indicate AD progression with a strong coefficient of determination in regression analysis. Individuals with high levels of amyloid deposits are also more likely to acquire AD within five years, according to survival research. Furthermore, chi-square analysis indicated significant differences in hippocampus volume between MCI and AD phases, highlighting the value of neuroimaging as a diagnostic tool. The model predicted 74 % of cognitive deterioration, with tau and Aß (ß0,78, p<0,01) being major predictors. High amyloid levels substantially raise the risk of Alzheimer's disease by 60 % over five years (p < 0,01). These findings emphasize the need of using complicated numerical tools into preventative and treatment strategy.

Limitations and Future Research

The research relies mainly on CSF biomarkers and brain imaging, which are either readily available or viable in all therapy scenarios. Integrating novel biomarkers, such as blood-based indicators and advanced neuroimaging techniques, can improve diagnostic accuracy. Developing artificial intelligence-based solutions that automate biomarker interpretations could change early AD and treatment efforts.

BIBLIOGRAPHIC REFERENCES

1. Ramesh M, Govindaraju T. Multipronged diagnostic and therapeutic strategies for Alzheimer's disease. Chemical science. 2022;13(46):13657-89. 10.1039/d2sc03932j

2. Liss JL, SeleriAssunção S, Cummings J, Atri A, Geldmacher DS, Candela SF, Devanand DP, Fillit HM, Susman J, Mintzer J, Bittner T. Practical recommendations for timely, accurate diagnosis of symptomatic Alzheimer's disease (MCI and dementia) in primary care: a review and synthesis. Journal of internal medicine. 2021 Aug;290(2):310-34. https://doi.org/10.1186/s13195-022-01055-y

3. Li J, Zhang Y, Lu T, Liang R, Wu Z, Liu M, Qin L, Chen H, Yan X, Deng S, Zheng J. Identification of diagnostic genes for both Alzheimer's disease and Metabolic syndrome by the machine learning algorithm. Frontiers in immunology. 2022 Nov 2;13:1037318. https://doi.org/10.3389/fimmu.2022.1037318

4. Hameed S, Fuh JL, Senanarong V, Ebenezer EG, Looi I, Dominguez JC, Park KW, Karanam AK, Simon O. Role of fluid biomarkers and PET imaging in early diagnosis and its clinical implication in the management of Alzheimer's disease. Journal of Alzheimer's disease reports. 2020 Feb 12;4(1):21-37. https://doi.org/10.3233/ADR-190143

5. Turner RS, Stubbs T, Davies DA, Albensi BC. Potential new approaches for diagnosis of Alzheimer's disease and related dementias. Frontiers in neurology. 2020 Jun 5;11:496. https://doi.org/10.3389/fneur.2020.00496

6. Veitch DP, Weiner MW, Aisen PS, Beckett LA, DeCarli C, Green RC, Harvey D, Jack Jr CR, Jagust W, Landau SM, Morris JC. Using the Alzheimer's Disease Neuroimaging Initiative to improve early detection, diagnosis, and treatment of Alzheimer's disease. Alzheimer's & Dementia. 2022 Apr;18(4):824-57. 10.1002/alz.12422

7. Jack Jr CR, Andrews JS, Beach TG, Buracchio T, Dunn B, Graf A, Hansson O, Ho C, Jagust W, McDade E, Molinuevo JL. Revised criteria for diagnosis and staging of Alzheimer's disease: Alzheimer's Association Workgroup. Alzheimer's & Dementia. 2024 Aug;20(8):5143-69. 10.1002/alz.13859

8. Dulewicz M, Kulczyńska-Przybik A, Mroczko P, Kornhuber J, Lewczuk P, Mroczko B. Biomarkers for the diagnosis of Alzheimer's disease in clinical practice: the role of CSF biomarkers during the evolution of diagnostic criteria. International journal of molecular sciences. 2022 Aug 2;23(15):8598. https://doi.org/10.3390/ijms23158598

9. Cano A, Turowski P, Ettcheto M, Duskey JT, Tosi G, Sánchez-López E, García ML, Camins A, Souto EB, Ruiz A, Marquié M. Nanomedicine-based technologies and novel biomarkers for the diagnosis and treatment of Alzheimer's disease: from current to future challenges. Journal of nanobiotechnology. 2021 Apr 29;19(1):122. https://doi.org/10.1186/s12951-021-00864-x

10. Rezabakhsh A, Rahbarghazi R, Fathi F. Surface plasmon resonance biosensors for detection of Alzheimer's biomarkers; an effective step in early and accurate diagnosis. Biosensors and Bioelectronics. 2020 Nov 1; 167:112511. https://doi.org/10.1016/j.bios.2020.112511

11. Buccellato FR, D'Anca M, Fenoglio C, Scarpini E, Galimberti D. Role of oxidative damage in Alzheimer's disease and neurodegeneration: From pathogenic mechanisms to biomarker discovery. Antioxidants. 2021 Aug 26;10(9):1353. https://doi.org/10.3390/antiox10091353

12. Shen Y. Recent advances in Carbon Nanotube Based Field-Effect Transistor Biosensors for Established Biomarkers for the Diagnosis of Alzheimer's Disease.doi:10.26434/chemrxiv-2023-d9d92

13. Vakili O, Asili P, Babaei Z, Mirahmad M, Keshavarzmotamed A, Asemi Z, Mafi A. Circular RNAs in Alzheimer's disease: A new perspective of diagnostic and therapeutic targets. CNS & Neurological Disorders-Drug Targets-CNS & Neurological Disorders). 2023 Nov 1;22(9):1335-54. https://doi.org/10.2174/1871527321666220829164211

14. Demnitz-King H, Saba L, Lau Y, Munns L, Zabihi S, Schlosser M, del-Pino-Casado R, Orgeta V, Marchant NL. Association between anxiety symptoms and Alzheimer's disease biomarkers in cognitively healthy adults: a systematic review and meta-analysis. Journal of Psychosomatic Research. 2023 Mar 1; 166:111159. https://doi.org/10.1016/j.jpsychores.2023.111159

15. Jorfi M, Maaser-Hecker A, Tanzi RE. The neuroimmune axis of Alzheimer's disease. Genome Medicine. 2023 Jan 26;15(1):6. https://doi.org/10.1186/s13073-023-01155-w

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None.

CONFLICT OF INTEREST

Authors declare that there is no conflict of interest.

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