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Impact of atrial fibrillation on cognitive decline and cortical atrophy in Alzheimer's disease

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ABSTRACT

Aims: This study aimed to evaluate the impact of atrial fibrillation (AF) on cognitive function and neuroradiological markers in patients with Alzheimer's disease (AD).

Methods: A total of 800 patients were screened at the Dementia Outpatient Clinic of Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital. Thirty-three AD patients with confirmed AF were included, alongside 31 age-, sex, and education-matched AD patients without AF as controls. Cognitive function was assessed using the Mini-Mental State Examination (MMSE), while mood symptoms were evaluated with the Geriatric Anxiety Scale and the Geriatric Depression Scale. Brain MRI scans were analyzed using Fazekas, Medial Temporal Atrophy (MTA), and Koedam scores. Due to the modest sample size, this study is best interpreted as exploratory in nature.

Results: After six months, the AF-positive group exhibited a significant decline in MMSE scores, whereas the AF-negative group showed no significant change. Koedam scores were significantly higher in the AF-positive group, indicating more advanced cortical atrophy. Regression analysis revealed that AF had a borderline significant negative impact on cognitive performance after adjusting for baseline MMSE scores.

Conclusion: AF may accelerate cognitive decline and is associated with greater cortical atrophy in patients with AD. These findings suggest that close cognitive monitoring and individualized care strategies may be beneficial in this population, though further research is needed to confirm this.

Keywords: Alzheimer disease, atrial fibrillation, cognitive decline, cortical atrophy, neuroimaging

INTRODUCTION

Atrial fibrillation (AF) is one of the most common cardiac arrhythmias, affecting millions of individuals worldwide. While AF is a well-established risk factor for stroke and other cardiovascular diseases, recent studies have also highlighted its strong association with cognitive decline and dementia.[1-3] The impact of AF on cognitive function extends beyond thromboembolic events and may involve additional mechanisms such as cerebral hypoperfusion, neuroinflammation, and microvascular damage.[4-6]

Meta-analyses have shown that individuals with AF have a 34% higher risk of developing dementia compared to those without AF.[7] This risk increases threefold in patients who have experienced a stroke; however, AF has also been linked to cognitive decline in individuals without a history of stroke.[8] Neuroimaging studies have demonstrated that patients with AF more frequently exhibit structural brain abnormalities, including silent cerebral infarcts, brain atrophy, and cortical thinning. Furthermore, AF has been implicated in Alzheimer's disease-specific pathological processes, such as increased beta-amyloid accumulation.[1,4] Some hypotheses also suggest a bidirectional relationship, where amyloid accumulation in the heart, as seen in Alzheimer's disease (AD), may contribute to the development of AF.[4]

Despite the detrimental effects of AF on cognitive function, certain therapeutic interventions may modify this trajectory. Rhythm control strategies, including catheter ablation, have been associated with improved cerebral blood flow and may help slow cognitive decline in patients with AF.[9] Additionally, oral anticoagulant therapy-particularly direct oral anticoagulants-has emerged as an effective approach to reducing the risk of cognitive impairment in individuals with AF.[10]

This exploratory study aims to evaluate whether AF is associated with greater cognitive decline and more advanced cortical atrophy in patients with AD. Cognitive change was assessed prospectively over a six-month follow-up period using the mini-mental state examination (MMSE), while cortical atrophy was evaluated cross-sectionally at baseline using validated visual rating scales. By comparing cognitive and neuroimaging findings between AD patients with and without

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AF, the study seeks to clarify the potential contribution of AF to neurodegenerative processes.

METHODS

Participants

The study was approved by the Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital Ethics Committee (Date: 09.04.2025, Decision No: 2025/107). All procedures were conducted in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments. Informed consent was obtained from all participants or their legal guardians.

This study included 64 patients diagnosed with AD who were followed at the Dementia Outpatient Clinic of Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital. The diagnosis of AD was made according to the National Institute on Aging and the Alzheimer's Association (NIA-AA) criteria.[11]

A total of 800 patients with AD were screened for the presence of AF, and 33 patients with a confirmed diagnosis of AF were included in the study. A control group of 31 AD patients without AF was formed, matched to the AF group in terms of age, sex, and years of education. Only participants with complete data on these demographic variables were included.

Patients were enrolled regardless of the stage of dementia. Written informed consent was obtained from all participants or their legal guardians.

Only patients who had undergone cognitive assessments using the Mini-Mental State Examination (MMSE), Geriatric Depression Scale (GDS), and Geriatric Anxiety Scale (GAS) at the time of diagnosis were included. To ensure valid cognitive and neuroradiological comparisons, only individuals who had undergone magnetic resonance imaging (MRI) were included.

Exclusion criteria were; the presence of neurodegenerative disorders other than AD, history of stroke or large-vessel disease, severe neurological or psychiatric conditions, inability to complete assessments due to significant visual or auditory impairments, history of alcohol or substance abuse, and any disease significantly affecting physical functioning within the last six months.

Demographic data and disease duration were recorded for all patients. The study population was divided into two groups based on the presence or absence of AF. Cognitive status was assessed using the MMSE, while depression and anxiety levels were evaluated using the GDS and GAS. MRI findings were recorded and evaluated using standardized atrophy rating scales.

Data Collection Tools

All assessment tools used in this study have validated Turkish versions.

Mini-Mental State Examination (MMSE): Developed by Folstein et al.[12] (1975) and validated in Turkish,[13] the MMSE assesses five domains: orientation, registration, attention and calculation, language, and recall. The maximum score is 30. Scores are interpreted as follows: 0-9 indicates severe, 10-19 moderate, 20-23 mild cognitive impairment, and 24-30 is considered normal.

Geriatric Depression Scale (GDS): The 30-item GDS is a self-report tool used to assess depressive symptoms in older adults.[14] Items are scored 0 or 1, with higher scores indicating more severe depression. Score interpretation: 0-4=normal, 5-8=mild, 9-11=moderate, ≥12=severe depression.

Geriatric Anxiety Inventory (GAI): The GAI evaluates anxiety symptoms in the elderly across somatic, cognitive, and emotional domains.[15] The Turkish version includes 28 items, of which the first 23 are scored on a 4-point Likert Scale (0-3), yielding a total score range of 0-69. Higher scores indicate greater anxiety. Items 24-28 are clinician-rated and not included in the total score.

Radiological Imaging Findings

All imaging was performed using 1.5 Tesla MRI units (GE Signa Explorer; GE Healthcare, Milwaukee, WI, USA). Non-contrast T1-weighted, T2-weighted, and FLAIR sequences were obtained. T1-weighted images (TR/TE: 543/24 ms) were acquired in axial and sagittal planes; T2-weighted images (TR/TE: 5724/102 ms) were acquired in axial and coronal planes. FLAIR images (TR/TE: 8000/86 ms) were obtained in the axial plane.

Koedam, medial temporal atrophy (MTA), and Fazekas scores were evaluated by a neurologist with over five years of clinical experience, who was blinded to patients' demographic and clinical data. Cortical atrophy was evaluated cross-sectionally at baseline, with no follow-up imaging performed; therefore, no conclusions regarding longitudinal progression can be drawn.

Cognitive decline was evaluated separately and prospectively over a six-month observational period using MMSE scores.

Fazekas Grading System: Vascular lesions were evaluated and recorded based on lesion burden.[16] Fazekas scores were used to assess the burden of white matter hyperintensities as a marker of small vessel disease.

- **Fazekas grading system:**
  - **Fazekas 0:** No lesions or a single punctate lesion (white matter hyperintensity).
  - **Fazekas 1:** Multiple punctate lesions.
  - **Fazekas 2:** Beginning to coalesce lesions (bridging).
  - **Fazekas 3:** Large, confluent lesions.

Medial temporal atrophy (MTA) score: The qualitative assessment of the MTA score was based on the degree of atrophy in the choroid fissure, temporal horn, and hippocampal volume in the coronal plane, as described by Frisoni et al.[17,18]

- **Medial temporal atrophy (MTA) scoring system:**
  - **MTA 0:** Normal choroid fissure, temporal horn, and hippocampal volume.
  - **MTA 1:** Mildly enlarged choroid fissure.

- **MTA 2:** Moderately enlarged choroid fissure, with mild enlargement of the temporal horn and a mild reduction in hippocampal volume.
- **MTA 3:** Significantly enlarged choroid fissure, with moderate enlargement of the temporal horn and a moderate reduction in hippocampal volume.
- **MTA 4:** Severely enlarged choroid fissure, significant enlargement of the temporal horn, and a marked reduction in hippocampal volume.

Koedam score: The qualitative assessment of the Koedam score was based on the degree of parietal atrophy in the sagittal plane, as outlined by Koedam et al.,[19] and further supported by subsequent studies.[20,21]

- **Koedam (posterior/parietal) atrophy score:**
  - **Koedam 0:** No sulcal widening or precuneus atrophy.
  - **Koedam 1:** Mild sulcal widening and mild precuneus atrophy.
  - **Koedam 2:** Marked sulcal widening and moderate parietal cortical atrophy.
  - **Koedam 3:** Severe parietal atrophy with a "knife-edge" sulcal appearance.

Study Design

This study employed a combined design approach: cognitive function was assessed longitudinally over a six-month follow-up period using the MMSE, while structural brain imaging data were obtained and analyzed cross-sectionally at baseline. The primary aim was to explore whether AF is associated with greater cognitive decline and/or more advanced cortical atrophy in patients with AD.

Statistical Analysis

Data were analyzed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were first calculated for participants' demographic characteristics using frequency analyses. The normality of continuous variables was assessed using the Shapiro-Wilk test, along with evaluations of skewness and kurtosis (acceptable range: -3 to +3).

For group comparisons, the independent samples t-test was used for normally distributed variables, while the Mann-Whitney U test was applied to non-normally distributed data. Categorical variables were analyzed using the Pearson chi-square test or Fisher's exact test, as appropriate. Descriptive statistics for numerical data included the mean, standard deviation, median, minimum, and maximum values.

Spearman's correlation coefficient was calculated for bivariate correlation analyses. For multivariate analysis, linear regression models were constructed to evaluate the independent effect of atrial fibrillation on cognitive function. A p-value of <0.05 was considered statistically significant for all analyses.

RESULTS

Participant Characteristics

A total of 64 patients with Alzheimer's disease were included in the study. Of these, 33 patients were diagnosed with atrial fibrillation [AF (+)], while 31 patients without AF comprised the control group [AF (-)].

The mean age was 77.21±6.78 years in the AF (+) group and 74.39±9.25 years in the AF (-) group; the difference was not statistically significant (p > 0.05). The proportion of female participants was 54.5% (n=18) in the AF (+) group and 45.2% (n=14) in the AF (-) group, with no significant difference in sex distribution (p>0.05). The mean duration of education was 3.88±3.45 years in the AF (+) group and 3.77±3.01 years in the AF (-) group, also showing no significant difference (p>0.05).

These findings indicate that the two groups were comparable in terms of key demographic characteristics (Table 1).

Table 1. Demographic characteristics of the participants

Characteristic	AF (+) (n=33)	AF (-) (n=31)	Total (n=64)	p-value
Age (years)	77.21±6.78	74.39±9.25	75.83±8.17	>0.05
Sex (female)	18 (54.5%)	14 (45.2%)	32 (50.0%)	>0.05*
Education (years)	3.88±3.45	3.77±3.01	3.83±3.25	>0.05

AF: Atrial fibrillation

At baseline, vascular risk factors were similarly distributed between AF (+) and AF (-) patients. Diabetes mellitus was present in 24.2% of AF (+) patients and 22.6% of AF (-) patients. Hypertension was observed in 66.7% and 58.1%, hyperlipidemia in 18.2% and 25.8%, and current smoking in 3.0% and 12.9% of AF (+) and AF (-) patients, respectively. Regarding body-mass index (BMI), in the AF (+) group 12.5% were normal weight, 37.5% overweight, 45.8% obese, and 4.2% severely obese, while in the AF (-) group the proportions were 25.0%, 37.5%, 29.2%, and 8.3%, respectively.

Cognitive Assessment (MMSE)

At baseline, the difference in MMSE scores between the groups was at the threshold of statistical significance (AF [+]: 20.07±5.80; AF [-]: 17.73 ± 4.78; p=0.052). After six months of follow-up, MMSE scores were 19.64±6.51 in the AF (+) group and 17.64±6.07 in the AF (-) group, with no statistically significant difference between the groups (p=0.28).

Within-group changes over time: In the AF (+) group, MMSE scores significantly declined over six months (from 20.58 to 18.55; p=0.0004), while no significant change was observed in the AF (-) group (from 18.94 to 18.55; p=0.5215). This comparison is illustrated in Figure 1.

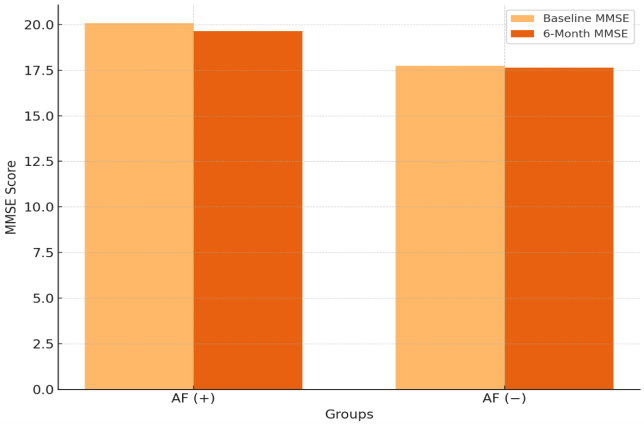

Figure 1. Comparison of MMSE scores at baseline and after 6 months in patients with atrial fibrillation [AF (+)] and without atrial fibrillation [AF (-)]

MMSE: Mini-Mental State Examination, AF: Atrial fibrillation

Radiological Findings

There were no statistically significant differences between the AF (+) and AF (-) groups in terms of Fazekas and Medial Temporal Atrophy (MTA) scores (p=0.935 and p=0.898, respectively). However, the Koedam score was significantly higher in the AF (+) group (1.85±0.67) than in the AF (-) group (1.55±0.57; p=0.027), suggesting a possible increase in cortical atrophy in patients with atrial fibrillation (Table 2 and Figure 2).

Table 2. Comparison of radiological atrophy scores between AF (+) and AF (-) groups. A significantly higher Koedam score was observed in the AF (+) group, indicating increased cortical atrophy

MRI Scale	AF (+)	AF (-)	p-value
Fazekas	1.85±1.23	1.74±1.12	0.935
MTA	1.85±1.23	1.74±1.12	0.898
Koedam	1.85±0.67	1.55±0.57	0.027

AF: Atrial fibrillation, MRI: Magnetic resonance imaging, MTA: Medial temporal atrophy

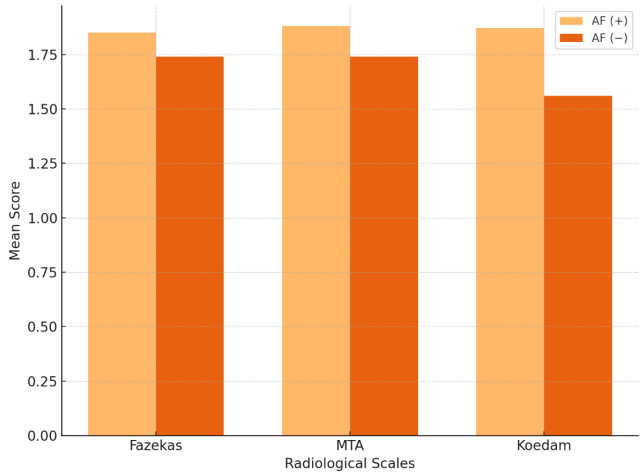

Figure 2. Comparison of fazekas, MTA, and Koedam scores between AF (+) and AF (-) groups in patients with Alzheimer's disease

MTA: Medial temporal atrophy, AF: Atrial fibrillation

Correlation Analyses

No significant correlation was found between MMSE scores and the presence of AF (p>0.05).

A weak but statistically significant positive correlation was observed between Koedam scores and the presence of AF.

Both MTA and Fazekas scores demonstrated significant negative correlations with MMSE scores:

- MTA vs. MMSE: r=-0.500, p<0.001
- Fazekas vs. MMSE: r=-0.380, p=0.002

Regression Analysis

Multiple linear regression models were constructed to assess the independent effect of AF on cognitive function. After adjusting for baseline MMSE, AF demonstrated a borderline negative association with 6-month cognitive performance, which did not reach conventional levels of statistical significance. Educational level emerged as a positive and statistically significant predictor in all three models (p<0.05 in models 1 and 2) (Table 3 and Figure 3).

Table 3. When controlling for baseline MMSE, AF showed a borderline significant negative effect on 6-month cognitive performance. Educational level emerged as a positive and statistically significant predictor in all three models (p<0.05 in models 1 and 2)

Model	AF (β)	p-value	Significance
Baseline MMSE	+2.32	0.22	Not significant
6-month MMSE	+0.77	0.68	Not significant
6-month MMSE (adjusted for baseline MMSE)	-1.36	0.108	Borderline significance

AF: Atrial fibrillation; MMSE: Mini-Mental State Examination

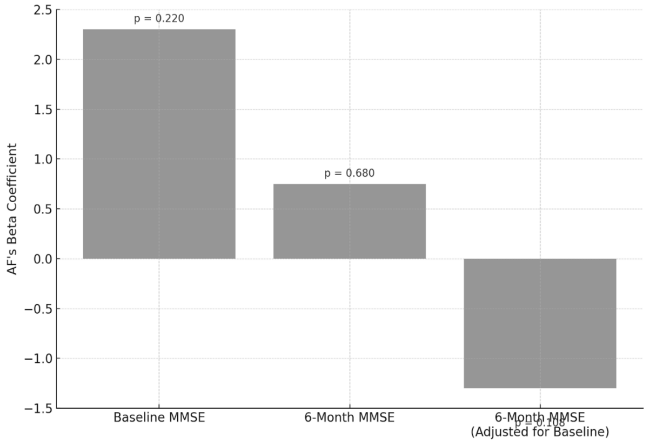

Figure 3. Beta coefficients and corresponding p-values from three regression models showing the independent effect of atrial fibrillation on cognitive performance as measured by the MMSE

MMSE: Mini-Mental State Examination

Adjusted Analysis Including Vascular Risk Factors

In the fully adjusted model including vascular risk factors, the previously significant association between AF and parietal atrophy (Koedam score) was no longer observed. In contrast, the short-term decline in MMSE scores remained borderline significant (β=-1.85; p=0.086), indicating a trend that may warrant further investigation regarding the possible role of AF in cognitive decline. These findings highlight the need for longer-term follow-up to determine whether this trend becomes statistically significant over time.

DISCUSSION

In this study, patients with AD and AF exhibited more pronounced cognitive decline than those without AF, particularly reflected in increased cortical atrophy, as indicated by higher Koedam scores. Based on the MMSE, a significant decline in cognitive performance was observed over six months in the AF (+) group, whereas no such change was detected in the AF (-) group. However, this decline was modest, and regression analyses indicated a borderline association after adjusting for baseline MMSE scores. These findings should be interpreted with caution, in line with the exploratory nature of the study.

Although the initially significant association between AF and parietal atrophy disappeared after adjusting for vascular risk factors, a borderline association with short-term MMSE decline persisted. This trend suggests a possible relationship between AF and cognitive deterioration, but causality cannot be inferred from the current data. Given the small sample size

and short follow-up duration, further longitudinal studies are needed to determine whether AF contributes independently to cognitive decline in Alzheimer's disease.

Our findings raise the possibility that, beyond traditional vascular risk factors, AF may play a role in neurodegeneration; however, this hypothesis requires confirmation in larger samples. These results may support closer monitoring of cognitive function in AD patients with AF, but clinical recommendations should await further evidence.

These findings suggest that AF may contribute to neurodegenerative processes in Alzheimer's disease not only through cardiovascular pathways but also via direct cerebral mechanisms, although confirmation through longer follow-up with serial imaging is needed. In the literature, the link between AF and cognitive impairment has been attributed to several factors, including cerebral hypoperfusion, neuroinflammation, microvascular damage, and beta-amyloid accumulation.[7,22-24] Silent brain infarcts and microemboli are also recognized as major contributors to cognitive decline in individuals with AF.

In our study, the significant association between AF and cortical atrophy (as measured by the Koedam score) supports the involvement of these neurodegenerative mechanisms. However, this association was no longer observed after adjusting for vascular risk factors, suggesting it may be confounded by comorbidities. Previous studies have reported a higher frequency of posterior cortical atrophy in patients with AF, which correlates with greater cognitive decline.[25] Additionally, large-scale cohort studies have demonstrated strong associations between AF, silent brain lesions, and lower cognitive performance.[26]

Our results also revealed a positive and significant relationship between educational level and MMSE scores, supporting the theory of "cognitive reserve." Higher educational attainment may confer resilience against cognitive decline in the presence of risk factors such as AF.[27,28]

An important aspect in the literature regarding AF and cognitive outcomes is the role of treatment strategies. In a meta-analysis conducted by Chen et al.,[29] catheter ablation was shown to reduce the risk of dementia by 40% and significantly improve MMSE scores in patients with AF. While these findings are promising, their generalizability to patients with comorbid AD remains unclear. Similarly, studies have demonstrated that oral anticoagulant (OAC) therapy-particularly with direct oral anticoagulants (DOACs)-significantly reduces the risk of cognitive impairment.[30,31] In our study, only one patient received ablation therapy, and data regarding OAC or DOAC use did not reach statistical significance due to the small sample size. Future studies should more robustly investigate the cognitive effects of specific AF treatments in AD populations.

The strengths of this study include the selection of both AF (+) and AF (-) groups from a population with confirmed AD diagnoses, ensuring diagnostic homogeneity and enabling robust group comparisons. The combined analysis of cognitive and radiological data, along with the use of multivariate regression models, allowed for a more controlled evaluation of AF's possible impact on cognitive function.

Nevertheless, the study is limited by its modest sample size, short follow-up duration, and lack of comprehensive data on AF subtype or anticoagulant use. These constraints limit the generalizability of our findings and underscore the need for larger, longer-term prospective studies.

Limitations

This study has several limitations. First, the relatively small sample size and the short six-month follow-up limit the generalizability and longitudinal interpretation of the findings. Second, key clinical parameters such as AF subtype (paroxysmal, persistent, or permanent), anticoagulant use, and AF duration were not comprehensively analyzed due to limited data availability. These factors may have influenced the results and should be considered in future studies.

In addition, the use of a single cognitive screening tool (MMSE) limits the ability to detect more subtle or domain-specific cognitive changes. Future research should employ comprehensive neuropsychological assessments that evaluate multiple cognitive domains, including attention, memory, and executive function.

Long-term, multicenter prospective studies with larger cohorts are needed to better clarify the independent effects of AF on cognitive decline in patients with Alzheimer's disease. Incorporating functional imaging techniques-such as fMRI and PET-may also enhance understanding of the pathophysiological links between AF and neurodegeneration. Finally, future studies should explore the differential cognitive effects of specific oral anticoagulants (e.g., DOACs vs. vitamin K antagonists) to inform more personalized treatment approaches.

CONCLUSION

This study investigated the potential impact of AF on cognitive function and structural brain changes in patients with Alzheimer's disease. The findings indicated that cognitive decline was more pronounced in Alzheimer's patients with AF, particularly in relation to cortical atrophy, as evidenced by significantly higher Koedam scores. A statistically significant reduction in MMSE scores was observed over a six-month period in the AF (+) group, whereas no such decline occurred in the AF (-) group. Additionally, regression analyses revealed a borderline significant negative effect of AF on cognitive performance after adjusting for baseline MMSE scores. These results suggest that AF may be associated with more pronounced cortical atrophy and cognitive decline in Alzheimer's disease, although confirmation through larger and longer-term studies is needed. Given the exploratory nature of the study and the limited sample, these findings should be considered hypothesis-generating rather than conclusive. Monitoring cognitive function in patients with both AD and AF may be important, but treatment decisions should be based on a comprehensive clinical evaluation.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Sancaktepe Şehit Prof. Dr. İlhan Varank Training and Research Hospital Ethics Committee (Date: 09.04.2025, Decision No: 2025/107).

Informed Consent
Written informed consent was obtained from all participants or their legal guardians.

Referee Evaluation Process
Externally peer-reviewed.

Conflict of Interest Statement
The authors have no conflicts of interest to declare.

Financial Disclosure
The authors declared that this study has received no financial support.

Author Contributions
All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

Data, Material and/or Code Availability
The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

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