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Original Research

Association between periodontal disease and Alzheimer's disease: A cross-sectional study

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ABSTRACT

Objectives: To evaluate whether periodontal disease is more prevalent in adults diagnosed with Alzheimer's disease compared to adults without the condition.
Methods: A total of 72 individuals were included in the study: 36 patients diagnosed with Alzheimer's disease (test group) and 36 matched individuals without Alzheimer's disease (control group). The diagnosis of periodontal disease was established through a comprehensive periodontal assessment. Additionally, a microbiological analysis of subgingival plaque samples was conducted to detect the main periodontal pathogens.
Results: Statistically significant differences were observed in periodontitis extent between the two groups: 82.4% of patients in the test group and 56.3% in the control group presented generalized periodontitis. No association was found between the stage and extent of periodontitis and the severity of Alzheimer's disease. Furthermore, no significant associations were observed between the number of years since Alzheimer's diagnosis and periodontal parameters, including the stage (p=0.659), extent (p=0.551), and grade of periodontitis (p=0.769). *Prevotella intermedia* was found in 25% of the test group and 5.6% of the control group (p=0.022). *Campylobacter rectus* appeared in 16.7% of the cases, with a significant difference between groups (p=0.011). *Fusobacterium nucleatum* was more common in the control group (75%) than in the test group (27.8%), with a significant difference (p<0.001). *Eikenella corrodens* was detected only in the control group (22.2%) (p=0.005).
Conclusions: Although the overall prevalence of periodontitis was similar between groups, individuals with Alzheimer's disease tended to present a greater extent of generalized periodontitis. (Rev Port Estomatol Med Dent Cir Maxilofac. 2025;66(4):167-174)

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Introduction

The association between periodontal disease and systemic diseases has been studied over the past few decades. Since the 1980s, there has been a growing interest in the relationship between periodontitis and Alzheimer's disease (AD).[1,2]

Several human studies have established evidence of the association between periodontitis and AD.[1-6] It has been proposed that periodontitis may contribute to the progression of AD through two likely mechanisms.[3-6] In the first mechanism, periodontal pathogens and the host response elevate the levels of pro-inflammatory cytokines and other mediators released into the systemic circulation, thereby increasing the systemic inflammatory burden. Thus, periodontitis may induce a state of systemic/peripheral inflammation.[7] The second mechanism may involve brain invasion by bacteria and viruses residing in the dental plaque biofilm.[1-7]

The main objective of this study was to evaluate whether periodontal disease is more prevalent in adult patients diagnosed with AD than in adult patients without the disease. The secondary objective was to describe the microbiological profile of adult patients with AD and compare it to that of those without AD.

Material and Methods

A matched, cross-sectional study was conducted to assess the potential association between periodontal disease and AD. The study compared the prevalence of periodontal disease among adults diagnosed with AD versus cognitively healthy adults. The relationship between AD severity and periodontitis stage, extent, and grade was also assessed. The study was approved by the Ethics Committee of the Faculty of Dental Medicine, University of Lisbon (CE-FMDUL202133, May 5, 2021), registered at ClinicalTrials.gov (NCT05189132), and conducted following STROBE guidelines.

A sample size calculation conducted before the study indicated that at least 11 participants per group would be required to ensure statistical power and result reliability. A total of 36 adult patients diagnosed with AD were recruited from the Portuguese Alzheimer's Association and included in the test group. The control group consisted of 36 cognitively healthy adults without AD, recruited from the university clinic at the University of Lisbon. Participants were matched for age, sex, smoking status, and diabetes mellitus diagnosis.

Participants in the study were selected based on specific inclusion and exclusion criteria. All individuals attending the Portuguese Alzheimer's Association diagnosed with AD (regardless of age, AD stage, or systemic disease) were included if the following conditions were verified: voluntary consent or consent from a legal representative due to cognitive or functional incapacity; AD diagnosis after cognitive assessment (test group only); at least two teeth located in different quadrants of the oral cavity to ensure a representative periodontal assessment, in accordance with the current classification of periodontal disease. Participants were excluded if they refused oral examination, were edentulous, had undergone periodontal treatment, or had taken antibiotics in the previous six months.

The classification of periodontal and peri-implant diseases and conditions proposed by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) was used to classify periodontal disease.[8]

All clinical measurements were performed with a periodontal probe (PCP12, Hu-Friedy®) by a calibrated, nonblinded periodontist (V.R.R.). Plaque accumulation was assessed using the dichotomous plaque index (PI) to evaluate the presence or absence of plaque on four tooth sites and the gingival index (GI) to record the presence or absence of bleeding at the same sites.[9,10] Probing pocket depth (PPD), bleeding on probing (BOP), and gingival recession were measured and recorded at six sites per tooth. Tooth mobility and the presence of furcation lesions were also evaluated.[11,12] All participants underwent a radiographic evaluation. Intra-oral apical radiographs were used to identify the tooth with the greatest bone loss. Intra-examiner reliability was tested by repeating PPD and gingival recession measurements every two clinical exams, totaling 30 observations. Intraclass correlation coefficient values were 0.811 for PPD and 0.855 for gingival recession, indicating a good agreement.[13]

The microbiological analysis was performed using samples of subgingival dental plaque. The samples were collected from the site with the greatest probing depth in each quadrant. A sterile paper point from the microbiological kit (micro-IDent® kit) was inserted into the base of the periodontal pocket and left in place for 10 seconds. After sample collection, the paper point was placed in an individually labeled transfer tube. The samples were sent to Joaquim Chaves Laboratory, where polymerase chain reaction tests (PCRs) were performed to identify the following bacteria: *Aggregatibacter actinomycetemcomitans*, *Tannerella forsythia*, *Treponema denticola*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Fusobacterium nucleatum*, *Eikenella corrodens*, and *Campylobacter rectus*. The microbiological analysis was performed using DNA-Strip technology, involving three main steps:

- DNA was extracted from subgingival plaque using the automated MagnaPure system, which employs magnetic beads to isolate purified nucleic acids.
- Amplification was performed via two multiplex PCRs with biotinylated primers on a Veriti thermal cycler, using the micro-IDent plus11 kit.
- Reverse hybridization on membrane strips enabled probe binding to single-stranded amplicons, with detection by enzymatic color reaction; the resulting band intensity reflected bacterial DNA levels.

The cognitive assessment was performed by psychologists from the Portuguese Alzheimer's Association. Upon admission, all participants completed the Mini-Mental State Examination and the Lisbon Battery for Dementia Assessment and were classified using the Global Deterioration Scale.[14] After diagnosis, patients were divided into mild, moderate, and advanced stages of AD.[14-16]

Data were entered into Microsoft Excel (Microsoft Excel 365, Redmond, USA) and analyzed using SPSS version 29 (IBM, Armonk, NY, USA). Demographic variables, periodontitis presence and classification, clinical and microbiological pa-

rameters in the total sample, as well as AD's stage and duration in the test group, were assessed. Descriptive statistics included means, standard deviations, ranges for continuous variables, and absolute and relative frequencies for categorical variables.

Group comparisons were performed using Student's t-tests for independent samples or Mann-Whitney U tests for continuous variables, depending on whether the assumption of normality (assessed via the Kolmogorov-Smirnov test) was met. Chi-square or Fisher's exact tests were used for categorical variables – the latter when ≥20% of expected counts were <5. Continuous variables were illustrated with simple or grouped boxplots and categorical variables with bar charts. A significance level of 0.05 was applied throughout.

Results

The study population comprised 30.6% men and 69.4% women. The mean age was comparable between the two groups (75.89 years in the control group, 75.50 years in the test group). Regarding risk factors, 18 participants were diagnosed with controlled type II diabetes, with glycated hemoglobin (HbA1c) levels ranging from 6.5% to 7.3%, evenly distributed across both groups. None of the participants were smokers (Tables 1 and 2).

Table 1. Descriptive statistics for age in the overall sample, by sex, and by group.

	Age (years old)		
	Mean (SD)	[min; max]	p
Overall sample	75.69 (7.91)	[54; 93]	
Sex			
Feminine	76.22 (8.10)	[54; 93]	
Masculine	74.50 (7.49)	[61; 85]	
Group			0.836
Control (n=36)	75.89 (7.59)		
Test (n=36)	75.50 (8.32)		

SD – Standard deviation; min – minimum; max – maximum.

Table 2. Comparisons regarding demographic variables and oral hygiene habits between study groups using the [a] chi-square test or [b] Fisher's exact test

		Group		
		Control n=36	Test n=36	
		n (%)	n (%)	p
Sex	Feminine	25 (69.4)	25 (69.4)	1.000 [a]
	Masculine	11 (30.6)	11 (30.6)	
Education	3rd class	1 (2.8)	1 (2.8)	0.292 [b]
	4th class	19 (52.8)	16 (44.4)	
	6th class	2 (5.6)	0 (0)	
	9th class	1 (2.8)	4 (11.1)	
	12th class	10 (27.8)	7 (19.4)	
	Higher education	0 (0)	1 (2.8)	
	No education	3 (8.3)	7 (19.4)	
Smoking habits	No	36 (100)	36 (100)	–
	Yes	0 (0)	0 (0)	
Diabetes	No	27 (75)	27 (75)	1.000 [a]
	Yes	9 (25)	9 (25)	
Frequency of brushing per day	0 a 1	17 (47.2)	25 (69.4)	0.145 [a]
	2 a 3	12 (33.3)	6 (16.7)	
	>3	7 (19.4)	5 (13.9)	
Interproximal brushing	No	32 (88.9)	33 (91.7)	1.000 [b]
	Yes	4 (11.1)	3 (8.3)	
Chemical control of the bacterial plaque	No	33 (91.7)	32 (88.9)	1.000 [b]
	Yes	3 (8.3)	4 (11.1)	

n – absolute frequency; % – relative frequency

The periodontal parameters are presented in Table 3. Periodontitis was diagnosed in 94.4% of patients in the test group and 88.9% in the control group. The difference between the groups was not statistically significant (Table 4). No significant differences were observed between the test and control groups regarding periodontitis stages and grades. Both groups had similar proportions of patients across stages I to IV and grades A to C. However, the extent of periodontitis differed significantly, with a higher prevalence of generalized periodontitis in the AD group compared to controls (p = 0.021) (Table 4).

The average time since AD diagnosis was 6.08 years, with most patients in the moderate stage (Tables 5 and 6). No relationship was found between the stage or extent of periodontitis and the severity of AD stages (Tables 7 and 8). No significant associations were observed between the number of years since AD diagnosis and periodontal parameters, including periodontitis stage, extent, and grade (Tables 9, 10, 11, and 12).

Regarding the microbiological data, *A. actinomycetemcomitans* was found in 13.9% of patients in both groups. The percentage of *T. forsythia* was similar in both groups: 52.8% of the control group and 47.2% of the test group. *P. gingivalis* was isolated in 23.6% of cases: 19.4% of the control group versus 27.8% of the test group. The species *T. denticola* was found at higher levels in the test group compared to the control group: 44.4% and 30.6%, respectively, with no statistically significant difference. The presence of *P. intermedia* was 25% in the test group and 5.6% in the control group, with a statistically significant difference between the groups (p=0.022). *C. rectus* was isolated in only 16.7% of cases and showed a statistically significant difference between the groups (p=0.011): 2.5% of cases in the control group and 27.8% in the test group. *F. nucleatum* was

Table 3. Comparison of clinical parameters between study groups (presence/absence of Alzheimer's disease) using the Mann-Whitney U test.

	Groups		
	Control Mean (SD)	Test Mean (SD)	p
PI	52.01 (27.58)	57.69 (25.92)	0.444
GI	27.75 (22.08)	27.29 (22.18)	0.942
PPD	2.85 (0.9)	2.85 (1.06)	0.897
BOP	26.72 (22.47)	20.52 (16.05)	0.478
GR	2.4 (0.7)	2.5 (0.88)	0.758
Mobility	1.38 (0.41)	1.39 (0.47)	0.894
Furcation	1.2 (0.42)	1.67 (0.83)	0.278
Attachment loss	8.28 (3.24)	8.08 (3.26)	0.503
N° of teeth	17.81 (6.46)	15.14 (6.06)	0.052

SD – standard deviation; PI – plaque index; GI – gingival index; PPD – probing pocket depth; BOP – bleeding on probing; GR – gingival recession

Table 4. Comparisons regarding the presence and classification of periodontitis between study groups using the [a] chi-square test or [b] Fisher's exact test.

		Group		
		Control n =36 n (%)	Test n=36 n (%)	p
Periodontitis	No	4 (11.1)	2 (5.6)	0.674[b]
	Yes	32 (88.9)	34 (94.4)	
Stage of periodontitis	I	1 (3.1)	0 (0)	0.606[b]
	II	6 (18.8)	7 (20.6)	
	III	18 (56.3)	16 (47.1)	
	IV	7 (21.9)	11 (32.4)	
Extent of periodontitis	Generalized	18 (56.3)	28 (82.4)	0.021[a]
	Localized	14 (43.8)	6 (17.6)	
Grade of periodontitis	A	16 (50)	18 (52.9)	0.466[b]
	B	14 (43.8)	11 (32.4)	
	C	2 (6.3)	5 (14.7)	

n – absolute frequency; % – relative frequency

Table 5. Descriptive statistics for time since Alzheimer's disease (AD) diagnosis (years).

	(Test group, n=36)	
	Mean (SD)	[min,max]
Years since AD diagnosis	6.08 (1.66)	[3;11]

SD – standard deviation; min – minimum; max – maximum.

Table 6. Descriptive statistics for the stage of Alzheimer's disease.

		(Test group, n=36)
		n (%)
Stage of Alzheimer's disease	1	6 (16.7)
	2	22 (61.1)
	3	8 (22.2)

n – absolute frequency; % – relative frequency

Table 7. Cross-tabulations between periodontitis stage and Alzheimer's disease stage.

		Stage of Alzheimer's disease			Total	r_S (p)
		1	2	3		
Stage of periodontitis	II	1	4	2	7	-0.074 (0.677)
	III	4	8	4	16	
	IV	1	9	1	11	
Total		6	21	7	34	

rS – Spearman's correlation coefficient

Table 8. Cross-tabulations between periodontitis extent and Alzheimer's disease stage.

		Stage of Alzheimer's disease			Total	r_s (p)
		1	2	3		
Extent of periodontitis	Generalized	5	17	6	28	-0.023 (0.899)
	Localized	1	4	1	6	
Total		6	21	7	34	

r_s – Spearman's correlation coefficient

Table 9. Comparison of time since Alzheimer's disease (AD) diagnosis (years) between patients with and without periodontitis using the Mann-Whitney U test.

	Periodontitis		p
	No Mean (SD)	Yes Mean (SD)	
Years since AD diagnosis	5.50 (0.71)	6.12 (1.70)	0.537

SD – standard deviation

observed in 51.4% of included cases: in 75% of the control group and 27.8% of the test group, with statistically significant differences (p=<0.001). The presence of the species *E. corrodens* was statistically significantly different between groups (p=0.005): it was more prevalent in the control group (22.2%) and was not detected in any case in the test group (Table 13).

Discussion

The results of this study revealed a high prevalence of periodontitis in the study population, with a tendency for greater severity and extent in the AD group. These findings are consistent with previous literature, which suggests an association between periodontal disease and neurodegenerative disorders, possibly due to challenges in maintaining oral hygiene, altered systemic inflammatory profiles, and immune compromise in these patients.[4]

A higher frequency of stage IV periodontitis was observed in the AD group (32.4%) compared to the control group (21.9%). This may suggest that more advanced periodontitis in individuals with AD could be associated with an enhanced systemic inflammatory state. Moreover, AD has been linked to systemic inflammatory alterations that may impair local immune responses, thereby contributing to periodontal tissue breakdown.[3-5]

Table 10. Relationship between the years since Alzheimer's disease (AD) diagnosis and the stage of periodontitis.

Correlation				
			Years since AD diagnosis	Stage of periodontitis
Spearman's rho	Years since AD diagnosis	Correlation coefficient	1.000	-0.078
		Sig. (2-tailed)	.	0.659
		N	36	34
	Stage of periodontitis	Correlation coefficient	-0.078	1.000
		Sig. (2-tailed)	0.659	.
		N	34	66

Table 11. Relationship between the years since Alzheimer's disease (AD) diagnosis and the grade of periodontitis.

Correlation				
			Years since AD diagnosis	Grade of periodontitis
Spearman's rho	Years since AD diagnosis	Correlation coefficient	1.000	0.052
		Sig. (2-tailed)	.	0.769
		N	36	34
	Grade of periodontitis	Correlation coefficient	0.052	1.000
		Sig. (2-tailed)	0.769	.
		N	34	66

Table 12. Time since Alzheimer's disease (AD) diagnosis (years) and the extent of periodontitis using the Mann-Whitney U test.

		Years since AD diagnosis			
		Mean	Standard deviation	Minimum	Maximum
Extent of Periodontitis	Generalized	6.0	1.4	3.0	8.0
	Localized	6.7	2.9	3.0	11.0

Table 13. Association between bacterial presence and study groups (presence/absence of Alzheimer's disease) using the [a] chi-square test or [b] Fisher's exact test.

		Group		p
		Control n (%)	Test n (%)	
A. actinomycetemcomitans	No	31 (86.1)	31 (86.1)	1.000 a
	Yes	5 (13.9)	5 (13.9)	
T. forsythia	No	17 (47.2)	19 (52.8)	0.637 a
	Yes	19 (52.8)	17 (47.2)	
P. gingivalis	No	29 (80.6)	26 (72.2)	0.405 a
	Yes	7 (19.4)	10 (27.8)	
T. denticola	No	25 (69.4)	20 (55.6)	0.224 a
	Yes	11 (30.6)	16 (44.4)	
P. intermedia	No	34 (94.4)	27 (75)	0.022 a
	Yes	2 (5.6)	9 (25)	
C. rectus	No	34 (94.4)	26 (72.2)	0.011 a
	Yes	2 (5.6)	10 (27.8)	
F. nucleatum	No	9 (25)	26 (72.2)	<0.001 a
	Yes	27 (75)	10 (27.8)	
E. corrodens	No	28 (77.8)	36 (100)	0.005 b
	Yes	8 (22.2)	0 (0)	

n – absolute frequency; % – relative frequency.

The extent of periodontitis was one of the few parameters that showed statistically significant differences between groups, with a higher proportion of AD patients presenting with generalized periodontitis. This finding suggests that, in addition to increased severity, periodontal disease in these patients may affect more teeth, increasing the functional impact and the need for more complex therapeutic interventions. Periodontitis in AD patients tends to be more severe and widespread due to chronic systemic inflammation, which can exacerbate immune responses and impair tissue repair.[17]

Another relevant aspect is the progression of periodontitis. Although no statistically significant difference was found in the grade of the disease between the groups, the presence of individuals with more advanced grades in the AD group suggests that periodontitis in these patients may progress more aggressively or respond inadequately to conventional treatment. The results of this research agree with other studies published in the literature.[1-7,18-24] Additionally, some researchers highlight the importance of the duration of periodontal disease. They found no significant link between periodontal disease and AD initially, but after 10 years, long-term exposure increased the risk of developing AD by 1.7 times.[25,26]

The current literature regarding the link between periodontal pathogens and AD is controversial. Patients with AD tend to have poorer oral hygiene and a higher risk of periodontal diseases. However, pathogenic bacteria found in patients with AD have been either associated with an increased risk of incidence or mortality of the disease or inversely associated with these risks.[18,22,23]

Although the brain is considered an immune-privileged organ, there is increasing evidence that systemic inflammation contributes to neurodegeneration through microglial activation and the release of pro-inflammatory molecules, thereby promoting the progression of AD.[5] Accordingly, studies indicate that bacteria can induce local inflammatory damage, which, in chronic cases, may act as a trigger for neuroinflammation, significantly contributing to neurodegeneration and AD. For this reason, periodontal pathogens have been investigated and associated with the development and progression of AD. However, there is currently no clinical evidence to describe a direct link between periodontal disease and AD, although several studies propose indirect connections.[5,27,28]

Due to the absence of longitudinal data, the results can only provide correlational evidence suggesting that bacterial periodontal infections may be an additional factor in the pathogenesis of cognitive impairment and AD. Any correlation, even if statistically significant, cannot be interpreted as a cause-and-effect relationship in either direction; it merely indicates an association between events.

Although this study presents relevant findings, its modest sample size and cross-sectional design prevent establishing temporality or causal relationships between periodontal disease and AD, which is an important limitation. Due to AD's long preclinical phase, it is unclear whether periodontal destruction preceded its onset. Clinical data from the test group were collected outside a dental clinic, possibly affecting examination accuracy. Additionally, the chronic nature and the risk factors of both diseases require longitudinal studies to determine if periodontitis influences AD progression or if neurodegeneration worsens periodontal disease.

Another limitation is that variables such as medication use, oral hygiene practices, and the level of autonomy in daily care among patients with AD were assessed only through questionnaires, without direct control, potentially impacting the results. Thus, these variables' potential impact should be considered

when interpreting the findings. Patients with AD often experience cognitive decline that can compromise their ability to maintain adequate oral hygiene, leading to higher susceptibility to periodontal disease. Additionally, dependence on caregivers for daily oral care introduces variability in hygiene practices, which may influence periodontal outcomes. These factors could act as confounders in the observed associations between AD and periodontitis, highlighting the need for careful assessment of patient autonomy and caregiver involvement. Future studies should examine these variables and investigate systemic and neuroinflammatory biomarkers to clarify potential interaction mechanisms between AD and periodontitis.

The detection of specific periodontal pathogens in subgingival plaque is widely used to indicate active periodontal disease. However, despite the microbiological relevance, this methodology has limitations. Samples were taken from one site per quadrant, targeting the deepest pocket, which may not reflect the full microbial diversity of the oral cavity. Sampling with absorbent paper points has inherent technical constraints, and the 10-second collection period may be insufficient in areas with dense biofilm or altered crevicular fluid. Additionally, this technique mainly captures planktonic bacteria, potentially underestimating species strongly attached to tooth surfaces or embedded in biofilm. Other issues, such as cross-contamination, transport, and storage conditions, may also affect outcomes.

Conclusions

This study highlights a higher prevalence and greater extent of periodontitis and distinct subgingival microbial profiles in individuals with AD when compared to controls. Although no significant association was found between the severity or duration of AD and periodontal parameters, the findings suggest a potential link between cognitive impairment and periodontal health. Therefore, further research is needed to establish a clear associative relationship between periodontitis and AD.

Conflict of interest

The authors have no conflicts of interest to declare.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed their work center protocols on access to patient data and for its publication.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

CRediT authorship contribution statement

Vanessa Rocha Rodrigues: Conceptualization, Data curation, Formal analysis, Investigation, Project administration, Resources, Validation, Visualization, Writing – original. **Mónica Amorim:** Formal analysis, Validation, Writing – review & editing. **Gil Alcoforado:** Conceptualization, Methodology, Supervision, Validation; Writing – review & editing. **Susana Noronha:** Conceptualization, Methodology, Supervision, Validation, Writing – review & editing.

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Associação entre doença periodontal e doença de Alzheimer: um estudo transversal

RESUMO

Objetivos: Avaliar se a doença periodontal é mais prevalente em adultos diagnosticados com doença de Alzheimer em comparação com adultos sem a patologia.
Métodos: Foram incluídos 72 indivíduos no estudo: 36 pacientes diagnosticados com doença de Alzheimer (grupo teste) e 36 indivíduos emparelhados sem Alzheimer (grupo controlo). O diagnóstico de doença periodontal foi estabelecido através de uma avaliação periodontal abrangente. Adicionalmente, foi realizada uma análise microbiológica de amostras de placa subgengival para detetar os principais patógenos periodontais.
Resultados: Foram observadas diferenças estatisticamente significativas na extensão da periodontite entre os dois grupos: 82,4% dos doentes do grupo teste e 56,3% do grupo controlo apresentaram periodontite generalizada. Não foi encontrada relação entre o estádio ou a extensão da periodontite e a gravidade dos estádios da doença de Alzheimer. Também não foram observadas associações significativas entre o número de anos desde o diagnóstico de Alzheimer e os parâmetros periodontais, incluindo o estádio (p=0,659), a extensão (p=0,551) e o grau da periodontite (p=0,769). A Prevotella intermedia foi encontrada em 25% dos doentes do grupo teste e em 5,6% do grupo controlo (p=0,022). O Campylobacter rectus apareceu em 16,7% dos casos, com diferença significativa entre os grupos (p=0,011). A Fusobacterium nucleatum foi mais comum no grupo controlo (75%) do que no grupo teste (27,8%), com diferença significativa (p<0,001). A Eikenella corrodens foi detetada apenas no grupo controlo (22,2%) (p=0,005).
Conclusões: Embora a prevalência de periodontite tenha sido semelhante em ambos os grupos, os doentes com doença de Alzheimer apresentaram mais casos de periodontite generalizada. (Rev Port Estomatol Med Dent Cir Maxilofac. 2025;66(x):xxx-xxx)

Palavras-chave:
Doença de Alzheimer
Doença periodontal
Periodontite