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Periodontal Status in Patients with Diffuse Astrocytomas and Oligodendrogliomas (WHO Grade 2 and 3): A Case – Control Study

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Abstract

Background/Aim: While periodontal disease (PD) has been linked to various systemic malignancies, extensive research regarding its association with gliomas is currently lacking. The aim of the present study was to evaluate potential differences in periodontal status between patients diagnosed with diffuse astrocytomas and oligodendrogliomas (WHO Grade 2 and 3), and healthy controls.

Materials and Methods: In this retrospective case-control study, 85 patients with newly diagnosed diffuse gliomas (comprising 45 astrocytoma and 40 oligodendroglioma cases) and 255 matching healthy controls completed a standardized health questionnaire and underwent comprehensive clinical dental examinations prior to any oncological treatment. Periodontal status was evaluated using Probing Pocket Depth (PPD), Clinical Attachment Loss (CAL), Bleeding on Probing (BOP), and Gingival Index (GI) parameters.

Data were analyzed using univariate and multivariate logistic regression models to adjust for potential confounding variables.

Results: Following adjustment for educational level, socioeconomic, and smoking status, multivariate analysis revealed that patients with glioma exhibited a significantly higher Body Mass Index (BMI) (p=0.012, 95% CI=1.291-2.861), and a worse gingival index (GI) (p= 0.031, 95% CI= 1.178-2.539) compared to healthy controls.

Conclusions: Statistical analyses identified significant variations between the study groups, as higher BMI levels and unfavorable GI scores clustered predominantly within the glioma cases. These profiling distinctions remained robust after accounting for smoking status and sociodemographic variance.

Keywords: Gliomas (WHO Grade 2 and 3); Astrocytoma; Oligodendroglioma; Periodontal Disease; Adults

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Introduction

Gliomas represent the most frequent primary solid malignancies originating within the Central Nervous System (CNS) [1]. Based on histological traits and molecular markers, these neoplasms are stratified into four distinct grades according to the 2021 World Health Organization (WHO) CNS classification framework [2]. Under this classification, CNS WHO grade 2 and 3 Lower-Grade Gliomas (LGGs) predominantly comprise Astrocytomas (ACs) and Oligodendrogliomas (OGs), typically manifesting in young adults during their third and fourth decades of life. Within this group, ACs represent a highly prominent category arising from neoplastic astrocytes, which constitute the star-shaped glial architecture of the brain parenchyma [3]. Epidemiological data indicate that these tumors account for approximately 75% of all diagnosed gliomas and encompass 25% to 30% of all primary intracranial neoplasms. Among malignant gliomas, glioblastoma, designated as Grade IV AC, exhibits the highest prevalence at 58.4%. This is followed by diffuse AC (7.3%), anaplastic AC (6.8%), OG (3.5%), anaplastic OG (1.7%), pilocytic AC (5.0%), and malignant gliomas not otherwise specified (NOS) at 7.9% [2].

As a rare subset of diffuse neuroepithelial tumors, OGs stand as the third most frequent primary intracranial tumor, trailing only glioblastoma and diffuse AC, and accounting for roughly 5% of all primary CNS neoplasms [4]. In the United States, the annual incidence of OGs is estimated at approximately 1,000 new cases. These malignancies are divided into two distinct clinical categories: grade II and grade III (anaplastic) OGs.

Although primarily diagnosed in individuals aged 25 to 45, the more aggressive grade 3 counterparts generally present a decade later than grade 2 lesions, remaining exceedingly rare in both pediatric and geriatric populations. Notably, LGGs maintain a clear tendency or malignant progression into highly aggressive High-Grade Gliomas (HGGs). It is hypothesized that anaplastic OGs can develop from lower-grade precursors *via* the sequential acquisition of specific genetic mutations [5].

In line with oncogenic pathways observed in other malignancies, the pathogenesis of OGs is driven by specific molecular transformations. These genetic anomalies carry pivotal diagnostic and prognostic weight, serving as indispensable biomarkers for tumor profiling and classification [6]. Genetically, the definition of an OG requires the confirmed presence of either an IDH1 or IDH2 mutation [7], occurring concurrently with the mutual co-deletion of chromosome arms 1p and 19q [2, 8, 9]. Even though molecular biology has advanced substantially, the exact cellular pathways and biological mechanisms driving oligodendrogliogenesis remain partially clear. From a histopathological standpoint, these neoplasms are identified by sheets of uniform, rounded nuclei encircled by a clear cytoplasmic halo, establishing the classic, pathognomonic "fried-egg" cellular appearance. Due to the presence of anaplastic features, such as severe nuclear atypia, focal areas of necrosis, microvascular proliferation, hypercellularity, and increased mitotic counts, grade 3 OGs carry a notably worse prognosis than grade 2 tumors. Nonetheless, achieving a clear histopathological differentiation between these two grades can be highly challenging in certain clinical scenarios [6].

The clinical manifestation and epidemiological distribution of ACs vary significantly across demographic cohorts. In pediatric oncology, low-grade variants, predominantly pilocytic astrocytoma, are the most frequently documented. Conversely, the adult population experiences a higher predominance of high-grade, aggressive phenotypes, including anaplastic AC and glioblastoma multiforme [10, 11]. Within adult cohorts, the age-adjusted incidence rate is critically elevated for glioblastoma, standing at 3.23 per 100,000 individuals, whereas diffuse and anaplastic ACs present with lower rates of 0.46 and 0.42 per 100,000 individuals, respectively. Annually, approximately 15,000 novels AC cases are diagnosed in the United States, representing nearly half of all primary brain tumors [10, 11]. Furthermore, a distinct gender-based disparity is observable, with males displaying a slightly higher incidence compared to females, yielding a male-to-female ratio of 1.3-1.5:1 [3, 10].

Standard and developing treatment protocols across these lineages primarily involve safe maximal surgical resection, which is subsequently paired with adjuvant radiotherapy [12]. In scenarios of tumor recurrence, salvage options comprise reoperation and chemotherapy. Nevertheless, therapeutic efficacy remains limited, often yielding suboptimal clinical outcomes [13].

Although the precise etiology of these tumors remains poorly understood, high-dose ionizing radiation to the head and neck region, typically administered for preceding malignancies, stands as a well-established environmental trigger accelerating glioma pathogenesis, potentially including oligodendroglial lineages. Factors previously found to be associated with the larger category of glioma, which includes oligodendroglial tumors [14], such as ionizing radiation, family history of brain tumors, and allergic disease, may also be specifically associated with oligodendroglial tumors [15-18]. Occupational or accidental exposure to specific chemical agents, including vinyl chloride, petrochemical derivatives, and agricultural pesticides, alongside prolonged exposure to electromagnetic fields, have been investigated as potential carcinogens, though definitive epidemiological evidence linking the latter to astrocytoma development remains inconclusive [19, 20].

Individuals with a familial predisposition to CNS malignancies exhibit an elevated risk. Specifically, a positive family history of brain tumors is significantly associated with an increased risk of developing anaplastic OG (OR=2.2; 95% CI: 1.1-4.5) [15]. Hereditary susceptibility and genetic risk factors encompass well-characterized cancer predispostion syndromes, most notably Li-Fraumeni, Turcot syndrome, Neurofibromatosis type I (NF1) and constitutional mismatch repair deficiency [19]. Neurofibromatosis type 1, an autosomal dominant disorder caused by germline mutations in the NF1 tumor suppressor gene, exhibits a distinct pathogenic tropism for the CNS, as it is strongly associated with the clinical manifestation of brainstem ACs, pilocytic ACs, and various other intracranial malignancies [21].

Interestingly, a history of atopic diseases demonstrates an inverse, protective relationship with oligodendroglial tumorigenesis. A history of asthma only is strongly associated with a reduced risk of both low-grade OG and anaplastic variants [22]. Similarly, previous exposure to varicella-zoster virus (chicken pox) has been pooled with a decreased risk of developing oligodendroglial tumors [23]. Unlike other types of cancer, cigarette smoking does not seem to be associated with an increased risk of developing CNS tumors. This definitive epidemiological conclusion is strongly supported by extensive longitudinal cohort studies and comprehensive meta-analyses that rigorously evaluated both the quantitative intensity and chronological duration of tobacco consumption cohorts [24, 25]. Additional potential risk factors include profound states of immunosuppression, which appear to marginally elevate oncogenic risk, clinically documented in cohorts presenting with HIV infection or individuals undergoing aggressive immunosuppressive therapeutic regimens, while advanced age is associated directly with an increased incidence and susceptibility to high-grade, poor-prognosis ACs [26].

Periodontal Disease (PD), primarily categorized into gingivitis and periodontitis, represents a highly prevalent, destructive, and progressive chronic inflammatory disorder affecting approximately 15% of the adult population worldwide. Triggered by bacterial infections that invade the gingiva and supporting periodontal structures, its prevalence and severity are associated positively with advancing age, tobacco consumption, and inadequate oral hygiene. Pathogenic oral bacteria [27] and associated viruses [28] elicit a robust host immuno-inflammatory response. This cascade leads to periodontal pocket formation, clinical attachment loss, bleeding, and alveolar bone resorption, while aggressive forms of the disease ultimately culminate in tooth loss. Furthermore, intricate associations involving immuno-deficiencies, osteoporosis, and specific infectious parameters of the oral microbiota remain under active investigation

[29, 30].

Beyond localized destruction, periodontal infection exerts severe systemic implications across several vital organs, such as the heart and lungs [31]. The underlying pathogenesis is characterized by an elevated production of Reactive Oxygen Species (ROS) and subsequent oxidative stress, driven by interactions with acute-phase cytokines and chemokines, such as Interleukin (IL)-1, IL-6, and C-Reactive Protein (CRP), which modulate PD severity [32]. The implication of these inflammatory biomarkers in diverse systemic conditions may be attributed to a generalized inflammatory response, a systemic immune reaction to periodontal pathogens, or the dissemination of oral microflora into the systemic circulation [33, 34]. Consequently, numerous epidemiological studies have evaluated periodontitis as a potential risk factor for systemic disorders, establishing significant associations with cardiovascular and atherosclerotic diseases, stroke, respiratory conditions such as COPD, type 2 diabetes mellitus, rheumatoid arthritis, and various malignancies [30, 35-42].

An elevated overall cancer risk [43, 44] and specific region-to-region malignancies [45-47] have been strongly linked to poor oral hygiene, PD progression, and tooth loss, independent of confounding variables such as age, gender, smoking status, and socioeconononomic level. Although researchers have explored the potential causative and oncogenic role of PD across diverse anatomical sites, including the oral cavity, esophagus, stomach, pancreas, and lungs [44, 48-51], the findings remain controversial, even after adjusting for potential confounders. Additionally, shared immuno-inflammatory pathways are suggested to underpin both PD and carcinogenesis. Nonetheless, the exact pathophysiological mechanisms linking PD to cancer risk remain elusive.

Conversely, limited research has evaluated baseline periodontal health or oral manifestations within cohorts of patients already diagnosed with malignancies, such as gastric or lung cancer [52-60].

Notably, the periodontal status of patients suffering from highly aggressive tumors, such as brain malignancies, remains significantly understudied in current literature. To address this issue, the present research was performed to evaluate potential differences in periodontal status between individuals with histologically confirmed gliomas and healthy controls.

Materials and Methods

Research design and study sample

The current retrospective, case-control research was carried out across multiple clinical settings between May 2023 and May 2025. Sample size estimation and study size assessment were strictly calculated based on baseline glioma prevalence [2] and the EPI-TOOLS guidelines [61] (https://epitools.ausvet.com.au), determined with a 95% Confidence Interval (CI) and a desired statistical power of 0.8. Stratification of age groups followed the World Health Organization (WHO) epidemiological recommendations [62] for evaluating CNS tumor incidence [63].

The total cumulative study sample comprised 340 individuals, including an combined baseline of males and females aged 45 to 69 years, recruited from private medical, oncological, and neurological practices. The active patient cohort consisted of 85 validated glioma cases, specifically sub-classified into ACs and OGs, while 255 healthy individuals were selected to serve as the control group. To establish a rigorous baseline and eliminate potential confounding effects, such as tobacco smoking, Socio-Economic Status (SES), and educational background, the control group was carefully selected from the friendly, collegial, or local environment of the cases. Both groups were precisely matched for gender and age, and all participants were selected from the same urban population to ensure a representative study sample and minimize selection biases. Controls were derived from individuals presenting for routine health follow-ups at the designated medical practices between 2023 and 2025.

The patients group consisted of individuals whose glioma primary diagnosis was based on patients' Magnetic Resonance Imaging (MRI) findings; however, the definitive diagnosis was confirmed by histopathological examination of the intraoperatively removed tumor or its components, using traditional cytologic, histological, and histochemical methods [2, 64].

Eligibility and exclusion criteria

To be eligible for inclusion in the study, participants in both the patient and healthy control cohorts had to meet strict baseline requirements. Patients in the case group were required to have a newly diagnosed, histopathologically verified diffuse glioma, specifically classified as either AC or OG, according to the current WHO diagnostic criteria [65]. For the patient group, clinical data, staging, and diagnostic parameters were derived directly from official medical and neuro-oncological registries.

To avoid confounding systemic effects on the biological markers under evaluation, all participants (cases and controls) must not have received systemic antibiotics, immunosuppressive agents, or systemic glucocorticoids within the previous six months. In addition, individuals were excluded if they had undergone any conservative or surgical periodontal therapy within the preceding six months. Individuals with a history of diabetes mellitus, severe cardiovascular diseases, rheumatoid arthritis, acute pulmonary disorders, or any other primary malignancy were excluded from the study protocol. The mentioned conditions could alter oral microbial profiles and periodontal tissue indices, and could introduce significant secondary analytical biases [66].

For the patient cohort, specific neuro-oncological exclusion criteria were enforced. Patients with advanced metastatic disease originating from a primary focus outside the CNS, and those with secondary intracranial malignancies associated with multi-field carcinogenesis theories [67] were excluded. Additionally, to ensure unbiased baseline assessments, glioma patients were excluded if they had already initiated first-line oncological interventions, including surgery, adjuvant chemotherapy, targeted molecular therapy, or radiation therapy, prior to the baseline data collection.

Control selection and confounding control

Cases and controls were precision-matched in a 1:3 ratio based on gender, age (±4 years), socio-economic level, and smoking status (categorized as current, former, or never smokers), as these covariates represent principal risk factors for systemic inflammation and epidemiological variances [68-71].

Data collection and research questionnaire

All eligible cases (glioma patients) and healthy controls completed a modified version of the Minnesota Dental School Medical Questionnaire [72]. This standardized questionnaire was utilized to gather comprehensive data regarding patient's past medical and dental histories, current systemic disorders, and a wide array of

epidemiological and socio-economic variables.

Assessment of covariates

Socio-demographic characteristics were incorporated as covariates in the statistical analysis. Chronological age of cases and controls was stratified into four distinct cohorts, 35-39, 40-49, 50-59, and >60 years. SES was operationalized based on monthly income and dichotomized as ≤1,000 € and >1,000 €. Educational attainment was categorized into primary (elementary education) and higher education (University/College) levels. Tobacco consumption was classified into two operational categories: never-smokers (individuals who had consumed fewer than 100 cigarettes during their lifetime) and active/former smokers (individuals who had smoked at least 100 cigarettes in their lifetime, subdivided into those reporting current daily or occasional smoking, and those reporting complete cessation). Obesity was assessed utilizing the Body Mass Index (BMI), with participants stratified into normal (<30 kg/m^2) and high (≥30 kg/m^2) adiposity cohorts [73].

Clinical oral examination and periodontal disease indices assessment

Comprehensive oral and periodontal clinical evaluations were performed by a calibrated Dental Surgeon. All clinical examinations were executed in a standardized dental clinic environment utilizing a uniform dental light source, a mouth mirror, and a specialized, pressure-sensitive periodontal probe with a defined spring-loaded probing force of 0.2 N (UNC 15/Williams type, model DB764R, Aesculap AG & Co. KG, Tuttlingen, Germany) to eliminate subjective measurement variance. To isolate chronic periodontal tissue parameters and prevent acute anatomical skewing, all remaining retained roots and third molars were systematically excluded from scoring in all examined quadrants.

The primary clinical periodontal profile was established through the concurrent mapping of four standard diagnostics, Gingival Index (GI), Bleeding on Probing (BOP), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL). To ensure a highly rigorous diagnostic sensitivity, measurements were systematically recorded across all dental quadrants. For each individual tooth, clinical indices were mapped across multiple designated interproximal, buccal, and lingual sites (including mesiobuccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, and disto-lingual configurations). The maximum severity (worst recorded values) for each index was registered to the nearest 1.0 mm and subsequently operationalized as dichotomous categorical variables for final statistical processing.

Periodontal parameters were categorized according to the established staging criteria PPD, stratified into Stage I/II (maximum PPD≤4.0 mm or ≤5.0 mm, respectively, with predominant horizontal bone loss), and Stage III/IV (Stage III: PPD≥6.0 mm with vertical bone loss ≥3.0 mm, Class II/III furcation involvement, or moderate ridge defects, and Stage IV, meeting Stage III criteria alongside complex rehabilitation requirements driven by masticatory dysfunction, secondary occlusal trauma, tooth mobility ≥degree 2, severe ridge defects, bite collapse, tooth drifting/flaring, or <20 remaining teeth/10 opposing pairs) [74].

Clinical Attachment Loss (CAL), stratified into Stage I/II (interdental CAL at the site of greatest loss measuring 1-2.0 mm or 3-4.0 mm, respectively) and Stage III/IV (Stage III: interdental CAL at the site of greatest loss ≥5.0 mm with a history of ≤4 teeth lost due to periodontitis; Stage IV: interdental CAL ≥5.0 mm with a history of ≥5 teeth lost due to periodontitis) [74].

The presence and severity of soft-tissue gingival inflammation was defined based on the criteria established by the Löe and Silness classification system. For multivariable modeling, the GI was dichotomized into two clinical tiers: Score 0 (representing healthy, normal gingival tissues or mild inflammation, corresponding to Löe and Silness scores 0 and 1) and Score 1 (representing moderate-to-severe gingival inflammation, marked by distinct erythema, edema, and tendency to bleed, corresponding to Löe and Silness scores 2 and 3) [75].

Bleeding on Probing (BOP) was evaluated circumferentially and coded dichotomously as Score 0, complete absence of hemorrhage or Score 1, presence of bleeding. The BOP assessment was registered as a definitive positive occurrence if marginal or sulcular bleeding was visually displayed within 15 seconds following the controlled application of the periodontal probe.

Clinical standardization and intra-examiner reliability

To validate the reproducibility of the clinical assessments and to strictly quantify the intra-examiner variance, a sub-sample comprising 68 participants (representing 20% of the cumulative study cohort) was randomly selected for replication analysis. These individuals underwent a blinded clinical re-examination by the same primary examining dentist after a strict three-week interval. Statistical consistency between the baseline and secondary clinical assessments demonstrated an excellent level of agreement, with a calculated Cohen's Kappa coefficient of 0.96. In order to preserve the baseline intra-examiner variance and prevent transient behavioral confounding of the oral microenvironment, no oral hygiene instructions were provided to any of the participants during this three-week evaluative interval.

Ethical considerations

As a non-experimental, retrospective observational study, the protocol was conducted in alignment with institutional framework exemptions for retrospective registry analyses. All participants (or their legally authorized representatives) were fully informed regarding the study's specific aims, methodologies, and clinical significance, and provided written informed consent prior to enrollment in the study protocol.

Results

The mean ages were 44.5±3.1 years and 42.5±2.6 years for glioma patients and healthy individuals, respectively. Univariate model was used to compare indices between cases and controls for categorical variables. The outcomes showed that cases with elevated BMI (p=0.009) were statistically significant greater compared to healthy individuals, whereas no statistically significant differences were observed among the PD indices examined between glioma patients and healthy individuals (Table 1).

Unadjusted OR's and 95% CI are presented in the same Table. After applying logistic regression model, the final step (Wald method), was found that severe gingival inflammation, that is expressed by GI (p= 0.031) and the mentioned BMI (p= 0.012), were statistically significant different between cases and controls (Table 2). Adjusted OR's and 95% CI are also shown in the same Table.

Discussion

The most frequently utilized parameters for assessing periodontal

Table 1: Univariate analysis of cases and controls regarding each independent variable examined.

Variables	Cases	Controls	*p*-value	Odds Ratio and 95% Confidence Interval
Gender				
Males	49 (57.6)	133 (52.2)	0.379	1.249 (0.761-2.049)
Females	36 (42.4)	122 (47.8)		
Age				
35-39	21 (24.7)	66 (25.4)		
40-49	27 (31.8)	71 (27.8)	0.920	———
50-59	19 (22.4)	62 (24.3)		
60+	18 (21.2)	56 (22.0)		
Socio-economic status				
Low	51 (60.0)	148 (58.0)	0.751	1.084 (0.658-1.788)
High	34 (40.0)	107 (42.0)		
Education level				
Low	53 (62.4)	155 (60.8)	0.797	1.069 (0.644-1.772)
High	32 (37.6)	100 (39.2)		
Smoking				
Never smokers	28 (32.9)	89 (34.9)	0.742	0.916 (0.544-1.542)
Previous/current smokers	57 (67.1)	166 (65.1)		
Body Mass Index				
<30 kg/m^2	30 (35.3)	54 (21.2)	**0.009***	2.030 (1.187-3.473)
≥30 kg/m^2	55 (64.7)	201 (78.8)		
Probing pocket depth				
Stage I/II	36 (42.4)	87 (34.1)	0.171	1.419 (0.859-2.344)
Stage III/IV	49 (57.6)	168 (65.9)		
Clinical Attachment Loss				
Stage I/II	33 (38.8)	90 (35.3)	0.558	1.163 (0.701-1.930)
Stage III/IV	52 (61.2)	165 (64.7)		
Gingival Index				
Absence/Mild	31 (36.5)	107 (42.0)	0.372	0.794 (0.478-1.318)
Moderate/Severe	54 (63.5)	148 (58.0)		
Bleeding on Probing				
Absence	29 (34.1)	104 (40.8)	0.275	0.752 (0.450-1.256)
Presence	56 (65.9)	151 (59.2)		

Table 2: Presentation of association between AC and OG and variables examined according to Enter (first step-1a) and Wald (last step 9a) method of multivariate logistic regression analysis model.

Variables in the Equation									
		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I. for EXP(B)	
								Lower	Upper
Step 1[a]	Gender	,172	,264	,423	1	,516	1,187	,708	1,991
	Age.group	,187	,219	,472	1	,316	,829	,657	1,047
	Socioec.stat	,196	,269	,533	1	,465	,822	,485	1,392
	Educ.lev	,115	,277	,174	1	,276	,891	,518	1,532
	Smok.stat	,067	,278	,057	1	,811	1,169	,720	1,842
	Body.mass.ind	,646	,402	2,561	1	**,033***	,624	1,250	2,248
	Prob.poc.dep	,137	,285	,231	1	,630	,872	,499	1,523
	Clin.att.loss	,243	,292	,690	1	,406	,785	,443	1,390
	Bleed.on.prob	,324	,252	1,226	1	,268	,682	1,279	2,451
	Ging.index	,690	,294	2,521	1	**,069***	1,195	1,121	2,248
	Constant	3,789	,794	3,547	1	,000	,454		
Step 9[a]	Body.mass.ind	,692	,277	3,147	1	**,012***	1,802	1,291	2,861
	Ging.index	,714	,281	2,471	1	**,031***	1,798	1,178	2,539
	Constant	3,073	,704	3,446	1	,000	,342		
a. Variable(s) entered on step 1: gender, age, group, socioec.stat, educ.lev, smok.stat, body.mass.ind, prob. poc.dep, clin.att.loss, bleed.on.prob, ging.index.									

status encompass clinical indices such as PPD, CAL, GI, Plaque Index (PlI), BOP, Bleeding Point Index (BPI), Alveolar Bone Loss (ABL), and the number of missing or remaining teeth [76].

The findings revealed no statistically significant differences between cases and controls regarding socio-demographic characteristics, specifically gender, age, SES, educational level, and smoking status. Tobacco smoking is acknowledged as a potential risk factor for both the initiation and progression of PD and various malignancies [77]. Nonetheless, it frequently functions as a confounding variable in investigations evaluating the potential association between PD and cancer types where smoking etiologically contributes to oncogenesis.

Obesity and mainly raised BMI have been constantly associated with an increased risk of diverse type of cancers, such as breast [78], and Colorectal Cancer (CRC) [73]. The results recorded that AC and

OG patients had significantly higher values of BMI compared with controls, finding that cannot be confirmed by previous reports as similar reports have not been carried out.

Prior literature indicated that oral lesions often constitute the primary manifestation of various malignancies, including Multiple Myeloma (MM) [79-81], lung cancer [52], and gastric cancer [53].

The outcomes demonstrated that the PPD in the case group was not statistically significantly greater than in the control group. This finding persisted after adjusting for potential confounding variables, including smoking, SES, and educational attainment. PPD reflects the destructive pathology of a chronic inflammatory response and serves as an established indicator for assessing the severity of PD [82]. On the contrary, a prospective cross-sectional study reported that 76.0% of patients with oral or oropharyngeal cancer exhibited a PPD of 6.0 mm or greater, whereas merely 10.0% of the control group demonstrated equivalent PPD severity [51]. Similar outcomes were recorded in previous studies regarding acute leukemia patients [56]. Furthermore, a recent case-control study focusing on lung cancer patients [52] indicated that PPD was not differed statistically significantly between cases and controls. The same findings were observed in similar investigation in gastric cancer [53], CRC [55], MM [57], and Glioblastoma (GBM) patients [58].

AC and OG patients exhibited significantly worse mean values regarding the severity of gingival inflammation, as measured by the GI, compared to the controls, a finding that was not confirmed by previous studies, as similar reports have not been carried out, with the exception of recent studies that confirmed a similar pattern in gastric cancer [53], CRC [55], acute leukemia [56], MM [57], and GBM patients [58]. Only in one recent report no differences were observed regarding GI between breast cancer patients and healthy individuals [54].

Especially, the utilization of GI is frequently constrained in such epidemiological research, despite the fact that it directly reflects the inflammatory load of gingival tissue. Conversely, Hujoel *et al.,* [49] hypothesized that gingival inflammation could potentially serve as an etiological risk factor for the development of several malignancies.

CAL constitutes another critical index for assessing periodontitis severity [82], reflecting the long-term stages of chronic inflammation and the cumulative manifestations of destructive inflammatory processes. The present study did not demonstrate statistically significant differences in CAL values between the case and control groups. Moreover, equivalent findings regarding the CAL index have not been previously documented by other investigators regarding patients with gliomas.

Similar studies focused on different regions showed that patients with malignancies in lungs [52], stomach [53], breast cancer [54], acute leukemia [56], GBM [58], and exhibited significantly worse mean values regarding the mentioned index.

BOP constitutes a crucial parameter in periodontal examination and diagnosis, serving as the most valid indicator of PD activity [83], as it reflects the host's vascular response, characterized by hyperemia, capillary dilation, and elevated blood flow at the site of inflammation. PPD and CAL reflect the long-term stages of chronic inflammation, encompassing the cumulative manifestations of destructive inflammatory processes [84].

Although BOP is a widely utilized criterion for diagnosing gingival inflammation, deep periodontal pockets (≥ 5.0 mm) exhibit a significantly higher incidence of BOP [83].

No statistically significant difference was observed concerning BOP between the case and control groups, after adjusting for potential confounding variables. Similar findings were recorded in previous studies regarding gastric cancer [53], breast cancer [54], CRC [55], and MM patients [57]. On the contrary differences between cases and controls were observed in similar studies in lung cancer [52], and acute leukemia patients [56].

The elevated risk of PD in oncological patients has been hypothesized to arise from psychological burden, rather than from nutritional deficiencies, alterations in salivary quality and quantity, or disruptions in oral microbial and immunological homeostasis induced by chemotherapy or radiotherapy [85,86]. Furthermore, glioma patients may exhibit increased susceptibility to the progression and destruction of periodontal tissue compared to healthy individuals. This suggestion could potentially be attributed to the poor prognosis associated with metastatic, invasive histological subtypes of glioma patients [26].

The objective of the present study was to compare AC and OG patients with epidemiologically matched healthy controls regarding diverse PD indices, rather than to investigate a potential etiological or risk association between PD parameters and AC and OG development. Consequently, the current investigation is subject to certain methodological limitations. Retrospective case-control studies inherently lack the robust causal inference of prospective designs, whereas selection, recall, and confounding biases may generate distorted secondary associations among the examined parameters. Moreover, such studies rely heavily on self-reported questionnaires, where participants may fail to respond, provide unreliable data, or systematically over- or underestimate their actual medical status.

The principal strengths of the present study include the completeness of the follow-up period and the well-characterized cohort, which facilitated the rigorous adjustment for confounding variables and the assessment of interactions among established risk factors. Strengths of the current study were the adequate and representative study sample and that it was a matched case-control study, as was used a randomly selected population-based sample, methodology which warrants interval validity.

Conclusions

The current research identified significant variations between the study groups, as higher BMI levels and worse GI scores clustered predominantly within the glioma cases. These profiling distinctions remained robust after accounting for smoking status and sociodemographic variance.

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