COMPARATIVE EVALUATION OF THE PERIODONTAL STATUS AND SERUM PARAMETERS IN NORMAL BODY MASS INDEX AND OBESE CHILDREN

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Abstract

Objective: Childhood obesity is a growing global health concern, with established associations to both systemic and oral health complications. This study aimed to compare periodontal status and inflammatory biomarkers between obese and non-obese children. Materials and methods: Fifty-two children (26 obese, 26 non-obese) were included, with key parameters such as Plaque Index (PI), Bleeding on Probing (BOP), Probing Depth (PD), and Clinical Attachment Loss (CAL) assessed to evaluate periodontal health. Paraclinical indicators including fibrinogen, C-reactive protein (CRP), and vitamin D (25(OH)D) levels were also measured. Results and Discussion: Results demonstrated that obese children exhibited significantly higher values for PI, BOP, PD, and CAL, indicating poorer periodontal health. Elevated serum fibrinogen and CRP levels, coupled with lower vitamin D levels in obese children, underscored a state of chronic inflammation and potential nutrient deficiency associated with obesity. Conclusion: These findings highlight the need for a multidisciplinary approach to managing obesity and promoting oral health, suggesting that early intervention in dietary and lifestyle factors could mitigate these risks.

Key-words: childhood obesity, oral health, periodontal parameters, systemic inflammation, oral diagnosis, anxiety

Introduction

Childhood obesity is a significant public health issue that has been escalating globally [1]. It occurs when a child's body mass index (BMI) is significantly above the recommended range for their age and height. This condition poses severe risks to both the physical and psychological well-being [2-4]. It can lead to chronic diseases such as diabetes, heart disease, and hypertension [5].

One of the primary causes of childhood obesity is poor dietary habits, often characterized by consuming calorie-dense, nutrient-poor foods. Fast food, sugary beverages, and snacks high in fat and sugar have become everyday staples in children's diets. Coupled with a sedentary lifestyle, where children spend excessive time on screens rather than engaging in physical activities, these habits contribute to rapid weight gain [1].

Genetic factors and family environment also play a role [6, 7]. Children whose parents are obese are more likely to become obese themselves, not only due to shared genetics but also because of family eating patterns and lifestyle choices. Socioeconomic factors can further influence access to healthy foods and recreational facilities, making it more

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difficult for specific populations to maintain a healthy weight.

The psychological effects of obesity in children can be severe. Obese children often experience bullying, social isolation, and low self-esteem, which can lead to depression and anxiety. This emotional toll can exacerbate unhealthy behaviors, creating a cycle of weight gain and emotional distress [8].

Periodontal pathologies in children, while less common and severe than in adults, represent an important area of pediatric dental health. Periodontal disease refers to conditions that affect the supporting structures of the teeth, including the gingival tissue, bone, and periodontal ligaments. In children, these diseases primarily manifest as gingivitis and, less frequently, as periodontitis [9]. Early detection and treatment of periodontal issues are essential to prevent long-term damage and ensure proper oral health development.

Several factors contribute to the development of periodontal diseases in children. Poor oral hygiene is the most significant risk factor, as it leads to the accumulation of plaque and bacteria. Other contributing factors include hormonal changes during puberty, which can increase gum sensitivity, and systemic conditions such as diabetes, which can exacerbate gum inflammation. Additionally, certain medications, nutritional deficiencies, and genetic predispositions can also influence the likelihood of developing periodontal pathologies [10].

The global increase in childhood obesity has drawn considerable attention due to its extensive implications for both systemic and oral health. While the adverse effects of obesity on metabolic and cardiovascular systems are well established [11, 12], its impact on periodontal health in children is an emerging area of concern. Research has demonstrated a significant association between obesity and periodontal disease [13], highlighting the need for a comprehensive approach to prevent and manage these interrelated health issues in children.

Given the high prevalence of obesity and its known association with periodontal disease [14], this study aimed to comprehensively evaluate and compare periodontal health findings, along with serum levels of fibrinogen, C-reactive protein (CRP), and vitamin D (25-hydroxyvitamin D) in children with normal BMI and those classified as obese.

Materials and Methods

This observational study focused on evaluating oral health, particularly critical periodontal indicators between two study groups, given the growing evidence linking obesity to various health complications, including potential oral health issues. The study was conducted following the Declaration of Helsinki, and informed consent was obtained from all participants' parents or guardians before the commencement of the study.

A total of 52 children were selected for the study and categorized into two groups: non-obese (n=26) and obese children (BMI≥25) (n=26). The inclusion criteria ensured that participants had no systemic health conditions, had non-restorable teeth, fully erupted permanent teeth, and were undergoing stable orthodontic treatment, if applicable. To ensure the accuracy of the results, children who had recently used antibiotics, anti-inflammatory medications, or other

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systemic medications that could influence periodontal health were excluded. Standard anthropometric measurements, including BMI, height, and weight, were recorded for all participants.

A complex clinical examination was conducted to assess the oral health thoroughly. Periodontal indices were evaluated using a UNC-15 periodontal probe to measure several key parameters: Plaque Index (PI), probing depth (PD), clinical attachment loss (CAL), and bleeding on probing (BOP). These indices provided insights into the level of plaque accumulation, gingival health, and overall periodontal status in both groups.

To minimize bias and ensure the reliability of the clinical data, all participants were examined by multiple trained examiners. The principal examiner provided consistent oversight and instruction throughout the examination process, ensuring standardization in applying periodontal measurements. This approach was essential to reduce inter-examiner variability and ensure that the results reflected genuine differences in periodontal status between the obese and non-obese children rather than differences in the examiners' techniques.

Serum fibrinogen was measured through a Clauss clotting assay, where the rate of clot formation in plasma is calculated after adding a high concentration of thrombin. This method provides a quantitative fibrinogen level. CRP levels were measured using the enzyme-linked immunosorbent assay (ELISA), which detected and quantified CRP with high sensitivity. The 25-hydroxyvitamin D (25(OH)D) was assessed through liquid chromatography-tandem mass spectrometry (LC-MS/MS).

Once the data was collected, the gathered data were subjected to statistical analysis using SPSS software (version 21.0, IBM, Chicago, USA). Statistical tests, including the chi-square test, were applied to compare differences between the two groups. The results were presented as means, standard deviations, confidence intervals (with 95% CI), frequencies, and percentages. Significance levels were set at $p<0.05$ to determine whether the differences in periodontal health between obese and non-obese children were statistically significant.

Results

The study was conducted on 52 subjects divided into two groups: normal BMI child subjects (n=26) and obese child subjects (n=26). The mean age of the patients was 11.15±2.03 years old for the non-obese group and 10.97±1.87 years old for the obese patients (Table 1). The standard deviations indicate a slight variation in the age range within both groups. Still, the overall distribution of ages is comparable between obese and non-obese children.

The gender distribution is nearly balanced in both groups, with a slightly higher number of males in both the non-obese and obese groups (Table 1).

Both groups show a similar distribution of children from urban and rural environments. The majority of participants in both groups come from urban areas, with about 70% in each group (Table 1).

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Table 1. Demographic data for the study groups

Parameter		Non-Obese Group (n=26)	Obese Group (n=26)
Age (Mean ± Standard Deviation)		11.15±2.03	10.97±1.87
Gender [n, (%)]	Male	14 (53.84%)	15 (57.69%)
	Female	12 (46.16%)	11 (42.31%)
Environment [n, (%)]	Urban	18 (69.23%)	19 (73.07%)
	Rural	8 (30.77%)	7 (26.93%)

Table 2. Clinical findings in the study groups

Parameter	Non-Obese Group (n=26)	Obese Group (n=26)	P-value
BMI	18.12±2.51 (17.16, 19.08)	29.33±2.87 (28.23, 30.43)	<0.001
PI	0.53±0.17 (0.46, 0.60)	0.82±0.23 (0.73, 0.91)	0.04
BOP	2.95 ±0.79 (2.65, 3.25)	4.49 ±0.61 (4.26, 4.72)	<0.001
PD (mm)	1.24±0.44 (1.07, 1.41)	2.58±0.72 (2.30, 2.86)	0.008
CAL (mm)	0.13±0.02 (0.12, 0.14)	1.83±0.38 (1.68, 1.98)	0.002

Values are expressed as Mean ± Standard Deviation, (Confidence Interval set at 95%); BMI: Body Mass Index; PI: Plaque Index; BOP: Bleeding on Probing Index; PD: Probing Depth; CAL: Clinical Attachment Loss

The mean BMI for the obese group was considerably higher than that of the non-obese group, as expected by their classification (29.33±2.87 vs. 18.12±2.51) (Table 2). This difference underscores the group division and sets a foundation for examining how obesity may correlate with other periodontal health indicators.

The CIs for the PI values show a significant difference between the obese and non-obese groups. The obese group has higher plaque accumulation (mean = 0.82) than the non-obese group (mean = 0.53) (Table 2).

The CIs for BOP reveal a substantial difference between the two groups. The obese group shows a significantly higher tendency for gingival bleeding during probing (mean = 4.49%) compared to the non-obese group (mean = 2.95%) (Table 2).

The obese group has significantly deeper pockets (mean = 2.58 mm) than the non-obese group (mean = 1.24 mm). Moreover, the obese group showed a dramatic difference in CAL compared to the non-obese group. The mean CAL in the obese group (1.83 mm) is substantially higher than in the non-obese group (0.13 mm), indicating more severe periodontal damage and loss of supporting structures for the teeth (Table 2).

Table 3. Paraclinical findings in the study groups

Parameter	Non-Obese Group (n=26)	Obese Group (n=26)	P-value
Fibrinogen (mg/dL)	263 ± 25.23 (253.12, 272.88)	385 ± 29.78 (373.48, 396.52)	<0.001
CRP (mg/L)	0.3 ± 0.06	3.4 ± 0.17	<0.001

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	(0.28, 0.32)	(3.33, 3.47)	
25(OH)D (ng/mL)	36.47 ± 2.75 (35.41, 37.53)	19.72 ± 1.64 (19.10, 20.34)	<0.001

Values are expressed as Mean ± Standard Deviation, (Confidence Interval set at 95%); CRP: C-reactive protein; 25(OH)D: 25-hydroxyvitamin D

The obese group has significantly higher fibrinogen levels than the non-obese group (385 ± 29.78 mg/dL vs. 263 ± 25.23 mg/dL). The CIs do not overlap, indicating that the difference is statistically significant (Table 3).

CRP levels are substantially higher in the obese group (3.4 ± 0.17mg/L), with no overlap in CIs between groups.

Vitamin D levels are considerably lower in the obese group (19.72 ± 1.64ng/mL), with distinct CIs between groups, confirming a statistically significant difference.

Discussion

Obesity is characterized by an excessive accumulation of adipose tissue, which is metabolically active and contributes to a chronic low-grade inflammatory state. Adipocytes, or fat cells, secrete pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), exacerbating systemic inflammation [15]. This inflammatory response extends to the periodontium, the tissues that support the teeth, increasing the susceptibility to periodontal disease. The inflammatory mediators released in obesity can impair immune function and exacerbate tissue destruction, which is central to the pathophysiology of periodontal disease.

Periodontal disease in children typically begins with gingivitis, an inflammation of the gums characterized by redness, swelling, and bleeding [9]. If untreated, gingivitis can progress to periodontitis, a more advanced condition that involves the destruction of the supporting bone and connective tissues. While severe periodontitis is less common in children than in adults, obesity has been identified as a contributing factor that can accelerate the onset and severity of periodontal conditions in younger populations.

BMI serves as the primary indicator for categorizing children into obese and non-obese groups in this study. The wide gap in BMI values between the two groups confirms the validity of their classification. While BMI itself is not a direct measure of periodontal health, its strong correlation with various systemic and inflammatory conditions is well-documented. Obesity, as measured by BMI, is often linked to a state of chronic low-grade inflammation [16]. This systemic inflammation affects multiple organ systems, including the periodontium.

The significantly higher BMI in the obese group sets the stage for the following findings, as it suggests that children with higher BMI may have more significant systemic inflammation, which could contribute to the deterioration of periodontal health. Therefore, understanding how BMI correlates with the other periodontal parameters becomes essential in identifying how obesity directly or indirectly influences oral health.

The Plaque Index (PI) reflects the accumulation of bacterial plaque on the teeth, which is a key contributor to periodontal disease. The CIs for PI show that obese children have significantly

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higher plaque levels than their non-obese counterparts, suggesting a direct link between obesity and poor oral hygiene or the increased likelihood of plaque buildup. These results were consistent with Sfasciotti et al. [17] in 2016 and Scorzetti et al. [18].

One possible explanation for this is dietary habits. Children with obesity are more likely to consume high-sugar and high-carbohydrate diets [19], which create an ideal environment for plaque formation. Sugary foods promote the growth of pathogenic bacteria that lead to dental plaque, which, if not properly managed, can evolve into gingivitis and periodontitis. Additionally, obesity may be associated with reduced motivation for maintaining proper oral hygiene, which can exacerbate plaque buildup.

The high PI values in obese children indicate a greater risk of developing gingival inflammation and subsequent periodontal disease, as plaque is the initiating factor in periodontal pathologies. This finding emphasizes the need for targeted interventions in obese children to promote better oral hygiene practices and reduce plaque accumulation.

The significantly higher BOP in the obese group suggests that these children have more pronounced gum inflammation than their non-obese counterparts. Gum inflammation, as measured by BOP, is an early sign of gingivitis, which, if untreated, can progress to more severe forms of periodontal disease. These results are confined to Scorzetti et al. [18] and Zuza et al. [20], which can be due to a rise in levels of pro-inflammatory cytokines in the GCF of obese subjects reported by Khocht et al. [21].

The elevated BOP in obese children may be partly due to the inflammatory state associated with obesity. Adipose tissue produces pro-inflammatory cytokines, such as TNF-α and interleukin-6 (IL-6), which can exacerbate inflammation throughout the body, including in the periodontal tissues [22]. This systemic inflammation weakens the body's ability to manage local infections, such as those caused by oral bacterial plaque, thereby increasing the likelihood of periodontal disease.

Furthermore, the difference in BOP between the two groups suggests that obesity not only affects the systemic health of children but also directly influences their oral health by exacerbating the inflammatory response to plaque. This finding reinforces the importance of monitoring periodontal health in obese children and implementing preventative measures to reduce gingival inflammation.

Probing depth (PD) is a critical clinical indicator of periodontal disease progression. It measures the depth of the spaces or pockets between the teeth and gums, with deeper pockets being a sign of more severe disease. The confidence intervals for PD show a marked difference between the obese and non-obese groups, with obese children exhibiting significantly deeper periodontal pockets. These results were in agreement with Scorzetti [18], where more pockets were prevalent in obese subjects, and with Zuza et al. [20], where periodontal status was better in subjects with a normal BMI.

This finding is concerning because deeper probing depths are associated with advanced periodontal disease, where the support structures of the teeth (gingival tissue and bone) begin to deteriorate. The

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higher PD values in the obese group suggest that these children are at a greater risk of more advanced periodontal conditions, which could eventually lead to tooth mobility or loss if not addressed. The deeper probing depths in obese children could be attributed to the combined effects of chronic inflammation, poor oral hygiene, and dietary factors. The interplay between these factors accelerates the destruction of periodontal tissue, leading to deeper pockets. Therefore, this finding highlights the need for early intervention and more aggressive periodontal care in obese children to prevent the progression of periodontal disease.

Clinical Attachment Loss (CAL) is one of the most critical indicators of periodontal health, measuring the extent to which the periodontal ligament and bone have been destroyed. The CAL values show a stark contrast between the two groups, with obese children experiencing significantly more attachment loss than their non-obese peers. The mean CAL in the obese group (1.83 mm) is a clear sign of periodontal tissue destruction, indicating that obese children are more prone to severe periodontal disease. This destruction is likely exacerbated by the systemic inflammatory state associated with obesity, as well as by poor oral hygiene and high levels of plaque. The more significant attachment loss in obese children means that they are at a higher risk for tooth mobility, potential tooth loss, and long-term periodontal complications.

This difference in CAL underscores the need for proactive periodontal care in obese children. Preventive measures, such as improved oral hygiene practices, regular dental visits, and dietary counseling, are crucial to halting or slowing down the progression of periodontal tissue loss in this population.

The combination of higher PI, BOP, PD, and CAL in obese children points to a clear relationship between obesity and poorer periodontal health. Obesity seems to create a biological and behavioral environment that promotes periodontal disease. From a biological standpoint, the chronic inflammatory state induced by excess adipose tissue negatively affects the immune response in the gums, making them more susceptible to infection and inflammation. From a behavioral perspective, obese children may be less likely to maintain proper oral hygiene. They may consume diets high in sugar and refined carbohydrates, increasing their periodontal risk.

Fibrinogen, a protein essential for blood clotting, is also a known marker of inflammation. Elevated fibrinogen levels are often associated with various inflammatory and cardiovascular conditions in adults [23], but research increasingly highlights its relevance in children, particularly in the context of obesity. In comparing obese and non-obese children, significant differences in fibrinogen levels underscore the impact of obesity on the body's inflammatory response and potential health risks.

As observed in our study, obese children consistently show higher levels of fibrinogen compared to their non-obese counterparts. This difference is primarily attributed to the chronic low-grade inflammatory state that obesity promotes. Excess adipose tissue produces pro-inflammatory cytokines, signaling proteins that stimulate an immune response [22]. This process increases fibrinogen production as part of the body's response to

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perceived inflammation. While inflammation is part of the body's natural defense, chronic inflammation caused by obesity places a persistent burden on the circulatory system, contributing to the risk of developing cardiovascular diseases even in childhood.

The implications of elevated fibrinogen levels in obese children are substantial. Persistent high fibrinogen levels increase blood viscosity, making it harder for blood to flow smoothly and efficiently through blood vessels. Over time, this can contribute to arterial stiffness and heighten the risk of developing hypertension and atherosclerosis in later life. In contrast, non-obese children tend to have lower fibrinogen levels, reflecting their overall healthier inflammatory status and reduced cardiovascular risk.

Addressing elevated fibrinogen in obese children is critical, as it is both a marker and mediator of long-term health risks. Interventions aimed at weight reduction, increased physical activity, and balanced nutrition may help reduce inflammation and lower fibrinogen levels, thus decreasing the risk of cardiovascular complications. Understanding and monitoring fibrinogen levels in childhood provide early insights into obesity's impact on health, underscoring the importance of preventive measures and lifestyle changes to promote healthier outcomes.

C-reactive protein (CRP) is a well-established marker of inflammation and is often used to assess the risk of cardiovascular and metabolic diseases. In children, CRP levels are significantly influenced by body weight, with research showing marked differences between obese and non-obese children. Elevated CRP levels in obese children reflect a state of chronic low-grade inflammation that has implications for their long-term health [24].

Our study demonstrated that CRP levels are substantially higher in obese children compared to their non-obese peers. This elevation is mainly due to the pro-inflammatory environment created by excess adipose tissue, which releases cytokines that stimulate CRP production in the liver. This chronic inflammatory state is concerning, as it not only indicates the body's persistent immune response but also contributes to the early onset of metabolic complications, including insulin resistance and endothelial dysfunction [25].

In contrast, non-obese children generally exhibit low CRP levels, reflecting a minimal inflammatory response under normal conditions. This difference in CRP between the two groups highlights a significant health divide: while non-obese children are less likely to experience the adverse effects of inflammation, obese children are at an increased risk of developing conditions related to prolonged inflammation, such as type 2 diabetes and hypertension.

The elevated CRP levels in obese children underscore the importance of early intervention. Weight management through balanced nutrition, regular physical activity, and lifestyle changes can help reduce CRP levels, mitigating inflammation and lowering the risk of chronic health issues. Monitoring CRP in obese children offers valuable insight into their inflammatory status. It serves as a reminder of the profound impact that obesity has on childhood health, with consequences that may extend into adulthood.

Vitamin D is critical in bone health, immune function, and metabolic

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processes [26]. In comparing obese and non-obese children, significant differences in vitamin D levels highlight the impact of body weight on this essential nutrient. Our results show that obese children have lower levels of vitamin D than their non-obese peers, a discrepancy with important health implications.

One primary reason for lower vitamin D levels in obese children is the fat-soluble nature of the vitamin. In cases of excess adipose tissue, vitamin D becomes sequestered within fat stores, reducing its bioavailability in the bloodstream [27]. This sequestration means that, even if intake levels are similar, obese children often have less circulating vitamin D, which is necessary for the body's immediate needs, including bone mineralization and immune support. Lower levels of vitamin D in obese children also increase their risk of bone density issues. They may weaken immune defenses, making them more susceptible to infections and other health complications.

In contrast, non-obese children typically exhibit higher vitamin D levels, reflecting better absorption and bioavailability. This sufficiency in vitamin D is linked to healthier bone development, reduced inflammation, and improved immune function, underscoring the benefits of maintaining adequate vitamin D levels.

Strategies such as vitamin D supplementation, increased exposure to sunlight, and weight management can help improve levels. Raising vitamin D levels can reduce the risks associated with deficiency, including bone disorders and immune dysfunction, and promote better health outcomes. This difference in vitamin D status highlights the broader impact of obesity on nutrient availability and long-term well-being in children.

Several mechanisms have been proposed to explain the relationship between obesity and compromised periodontal health in children. First, the chronic inflammatory state induced by obesity may directly influence the inflammatory processes within the gingival tissues, promoting the development and progression of periodontal disease. Elevated levels of circulating cytokines and other inflammatory markers in obese individuals can exacerbate periodontal tissue damage and interfere with normal tissue repair mechanisms.

Second, obesity is often associated with poor dietary habits, such as high consumption of sugar-laden and processed foods, which can contribute to both weight gain and poor oral health. These diets not only lead to an increased risk of dental caries but also promote the formation of dental plaque, a key etiological factor in the development of periodontal disease. Plaque accumulation provides a reservoir for pathogenic bacteria, further aggravating gum inflammation and tissue breakdown.

Additionally, obesity is known to impair immune function, reducing the body's ability to effectively combat infections, including those affecting the periodontium [28]. This immune dysfunction may explain why children with obesity are more prone to developing periodontal diseases and exhibit a slower recovery from gingival inflammation compared to their non-obese counterparts.

The lifestyle factors commonly associated with childhood obesity further exacerbate the risk of periodontal disease. Sedentary behavior, lack of regular physical activity, and poor nutritional choices are

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typical among obese children and contribute to both systemic and oral health challenges [229. Inadequate oral hygiene practices, often linked to socioeconomic and behavioral factors, further increase the risk of periodontal problems in this population.

Moreover, children with obesity may experience psychological factors such as low self-esteem or depression [30], which can negatively affect their motivation to engage in proper oral hygiene practices. The combination of poor dietary choices, insufficient physical activity, and inadequate oral hygiene forms a detrimental cycle that exacerbates both obesity and periodontal disease.

While this study provides important insights into the relationship between obesity and periodontal health in children, several limitations should be considered. Firstly, the sample size of 52 children, though adequate for preliminary analysis, may not fully represent the broader pediatric population. A larger and more diverse sample would strengthen the generalizability of the findings across different demographic and socioeconomic groups.

Additionally, potential confounding factors, such as dietary habits, physical activity, socioeconomic background, and parental influence on lifestyle, are not addressed in this study. These variables could independently impact both obesity and periodontal health, affecting the validity of observed associations. Acknowledging these factors would improve the study's reliability.

Furthermore, the study does not consider psychological and behavioral factors, such as depression or low self-esteem, which are common in obese children and can negatively affect oral hygiene and health habits. Including these variables in future research would offer a more holistic view of how obesity influences periodontal health.

Addressing the dual challenge of childhood obesity and periodontal disease requires an integrated, multidisciplinary approach. Health professionals, including pediatricians, dentists, and dietitians, must collaborate to promote healthy lifestyles that encompass both systemic and oral health. Public health initiatives aimed at reducing childhood obesity should emphasize the importance of balanced nutrition, regular physical activity, and the reduction of sugar intake. These measures not only help prevent obesity but also reduce the risk of dental and periodontal problems.

Furthermore, preventive dental care is critical for mitigating the progression of periodontal disease in children with obesity. Regular dental check-ups, early intervention, and education on proper oral hygiene practices are essential components of a comprehensive approach to oral health. Dentists should be vigilant in monitoring the periodontal health of children who present with obesity and consider the systemic implications of their condition when devising treatment plans.

Conclusions

The significantly higher values for all periodontal and serum parameters in obese children reflect a concerning trend: obesity is strongly associated with poor periodontal health, and inflammation could play a significant role in connecting these two maladies. Given the impact of both systemic inflammation and behavioral

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factors such as diet and oral hygiene on the progression of periodontal disease, these findings emphasize the need for comprehensive health strategies that address both obesity and oral health. Preventive interventions and education for children and their caregivers should focus on improving diet, oral hygiene, and regular dental care to mitigate the long-term effects of obesity on periodontal health.

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