Gingival Health Status of 2- to 15-year-old Benghazi Children With Type-I Diabetes Mellitus

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ABSTRACT

Purpose: The influence of diabetes on the risk of developing gingivitis has been the subject of much discussion in the literature. Most of the previous literature has focused on adults, with limited studies available on children. The purpose of this study was to evaluate and compare the plaque and gingival health status of diabetic children with healthy controls.

Methods: A comparative study was conducted between 72 children with type-I diabetes mellitus (average age=10.18±3.91-years-old) and 72 healthy controls (average age=10.8±2.78- years-old). Both groups were compared statistically regarding plaque and gingival indices using the t test for equality of means.

Results: The experimental group showed higher mean plaque and gingival scores vs the control group. A statistically significant difference (P<.01) in plaque and gingival scores were observed between the groups in the primary, mixed, and permanent dentition.

Conclusions: Higher dental plaque levels and gingival scores were observed in the diabetic children vs the nondiabetic controls. Additional care for prevention of plaque accumulation and gingivitis should be highly recommended, particularly in diabetic youth.

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One of the primary etiological factors in the development of gingivitis is the bacterial dental plaque.[1,2] The level of individual oral hygiene is directly related to the amount of plaque accumulation on the teeth. It is reasonable to predict that the level of oral hygiene in a population is positively correlated with the prevalence and severity of gingivitis, periodontal disease, and dental caries.[3,4] The influence of diabetes on the risk of developing gingivitis has been the subject of much discussion in the literature. Numerous studies have shown that individuals with diabetes mellitus have an increased rate of gingivitis.[5-7] Higher dental plaque scores in diabetic children vs nondiabetic controls have also been observed by some researchers.[5,8] Some investigators did not find significant differences in clinical periodontal parameters between the insulin-dependent juvenile diabetics and the nondiabetic siblings.[9] Furthermore, diabetic children were shown to have more gingival inflammation than nondiabetic children, in spite of similar plaque scores.[10,11] It has been stated that gingivitis or periodontal pockets in diabetes mellitus occurs due to an altered response of the periodontal tissues to local factors.[12] Considering the contradictory reports of the previous studies and paucity of the literature on this subject in Benghazi, Libya, the present study was conducted to evaluate and compare the plaque and gingival health status of diabetic children with healthy controls.

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METHODS

The experimental group consisted of 72 patients (42 females and 30 males) between 2 and 15-years-old (average age=10.18±3.91-years-old) with type-I diabetes treated in the diabetic outpatient department of Al-Fateh Children's Hospital, Benghazi, Libya. The control group included 72 healthy school children (36 females and 36 males) between 3 and 14-years-old (average age=10.8±2.78-years-old) with no previous history of systemic disease. Controls were matched with the experimental group regarding socioeconomic status and oral hygiene habits. The project was approved by the hospital ethics committee of the faculty of Dentistry, Al-Arab Medical University, Benghazi, Libya. Informed consent to participate in the trial was obtained from all the subjects. Data were collected from the medical records of patients, including systemic complications and proposed treatment. Clinical examinations were carried out by a single examiner using a mouth mirror and an explorer, per World Health Organization criteria.[13] The experimental and control groups were divided equally into primary, mixed, and permanent dentition to assess and compare the plaque and gingival status. The plaque deposits and gingival status were determined using the Silness and Löe plaque index[14] and Löe and Silness gingival index.[15]

The plaque index (**PlI**), which assesses only the plaque thickness at the tooth's gingival area, was recorded for each tooth in 4 possible surface areas: the (1) distofacial; (2) facial; (3) mesiofacial; and (4) lingual. A mouth mirror and explorer were used after air drying of the teeth to assess plaque in the PlI. All the teeth in each subject of the control and experimental groups were assessed. The criteria for the PlI of Silness and Löe appear in Table 1.[14] The PlI score for the area was obtained by totalling the 4 plaque scores per tooth. The sum of the PlI per tooth was divided by 4 to obtain the PlI score for the tooth. The PlI score per person was obtained by adding the PlI scores per tooth and dividing by the number of teeth examined.[14]

The gingival index (**GI**) of each tooth was recorded of each tooth in 4 possible areas: the (1) distofacial papilla; (2) facial margin; (3) mesiofacial papilla; and (4) entire lingual margin. A blunt probe was used to assess the bleeding potential of the gingival tissues. Each of the 4 gingival units was assessed according to the criteria appearing in Table 2.[15] Totalling the scores around each tooth provided the GI score for the area. The scores around each tooth were totalled and divided by 4, to obtain the GI score for the tooth. Totalling all of the scores per tooth and dividing by the number of teeth examined provided the GI score per person. Gingival index scores associated with varying degree of gingivitis appear in Table 3.[15]

The plaque and gingival indices were scored for the control and experimental groups and compared statistically using the *t* test for equality of means. A *P*-value of <.01 was considered statistically significant. Children who needed dental treatment in both groups were referred to the Faculty of Dentistry, Pedodontics Clinic, Al-Arab Medical University, Benghazi, Libya.

RESULTS

The mean ages and standard deviations of the control and experimental groups were 10.8±2.78 and 10.18± 3.91-years-old, respectively. Table 4 shows the mean plaque and gingival scores for the control and experimental groups observed in the primary, mixed, and permanent dentition. Mild gingivitis, as shown in Figure 1, was the average finding in the control group, which was considered a baseline for comparison with the experimental group. Moderate gingivitis was the salient feature in the experimental group, as shown in Figure 2.

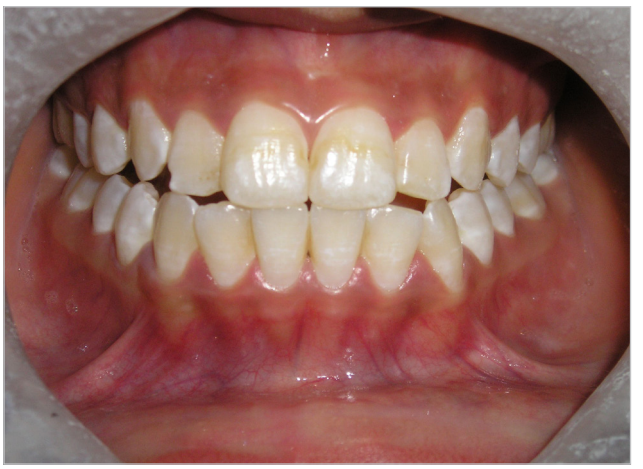

Figure 1. Showing the average finding of mild gingivitis in the control group.

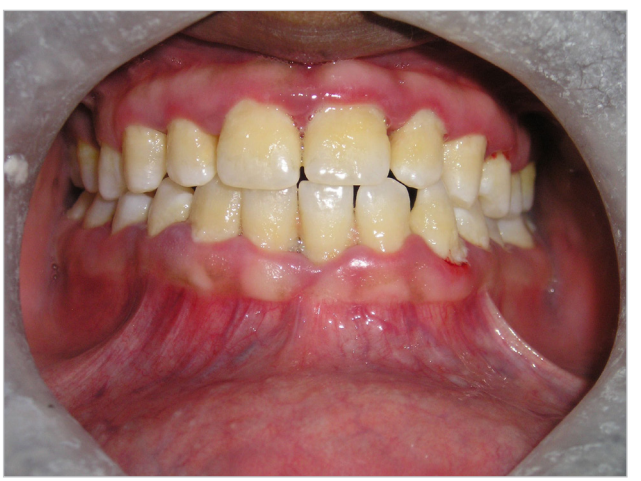

Figure 2. Showing the salient feature of moderate gingivitis in the diabetic children.

Table 1. Criteria for the Plaque index (Silness and Löe)[14]

Plaque index score	Condition
0	No plaque in the gingival area
1	A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be recognized only by running a probe across the tooth surface
2	Moderate accumulation of soft deposits within the gingival pocket and on the gingival margin and/or adjacent tooth surface, which can be seen by the naked eye
3	Abundance of soft matter within the gingival pocket and/ or the gingival margin and adjacent tooth surface

Table 2. Criteria for the Gingival index (Löe and Silness)[15]

Gingival index scores	Condition
0	Normal gingiva
1	Mild inflammation, slight change in color, and slight edema; no bleeding on palpation
2	Moderate inflammation, redness, edema, and glazing; bleeding on palpation
3	Severe inflammation, marked redness and edema, and ulcerations; tendency to spontaneously bleed

Table 3. Gingival index scores associated with varying degree of gingivitis[15]

Gingival index scores	Condition
0.1-1.0	Mild gingivitis
1.1-2.0	Moderate gingivitis
2.1-3.0	Severe gingivitis

The experimental group showed higher mean plaque and gingival scores for the primary, mixed, and permanent dentition compared with the control group as shown in Table 4. Table 5 depicts a highly significant difference ($P<.01$) in plaque and gingival scores that was observed between the groups in the primary, mixed, and permanent dentition.

DISCUSSION

Gingivitis is a term used to describe nondestructive periodontal disease.[16] The most common form of gingivitis is in response to bacterial biofilms (also called plaque) adherent to tooth surfaces, termed plaque-induced gingivitis, which is the most common form of periodontal disease. Clinically, gingival inflammation is seen as redness, swelling, and bleeding upon probing.[17] The products of biofilm bacteria, such as lipopolysaccharide molecules, are known to initiate a chain of reactions in the tissue leading to a host response as well as the destructive process.[18] The predominant cells of the periodontium, gingival fibroblasts, are capable of producing prostaglandins, interleukins (IL-1β, IL-6, IL-8), tumor necrosis factor-alpha (TNF-α), and interferon- gamma (IFN-γ).[19] It is hypothesized that these mediators modulate inflammation. In the absence of treatment, gingivitis may progress to periodontitis, which is a destructive form of periodontal disease.[17]

Gingivitis is a highly prevalent condition that can begin in childhood. Studies have reported that 35% to 85% of 3- to 6-year-old children have gingivitis.[20-23] In children, as in adults, the most common cause of gingivitis is plaque, which is favored by poor oral hygiene.[24]

Higher dental plaque scores in diabetic children vs nondiabetic controls was observed in the present study, and this finding agrees with previous studies.[5,8] In the present investigation, mild gingivitis was an average finding in the controls. By and large, moderate gingivitis was commonly observed in the experimental group. Higher mean gingival scores in young diabetics compared with healthy controls were observed, which is incongruent with previous findings.[5-7]

Diabetic children endure many health problems in their lifetime. Oral complications of diabetes mellitus include xerostomia, cheilosis, reduced salivary flow, and, particularly, the increased risk of periodontal breakdown.[25]

Currently, the most widely accepted theory for the pathogenesis of periodontal destruction in diabetics has been offered by Glickman, the father of modern periodontology, who considered diabetes mellitus as not the direct cause, but a predisposing condition for periodontal disease. The reason attributed for increased risk for periodontal destruction could be chronic hyperglycemia, which adversely affects the synthesis, maturation, and maintenance of collagen and extracellular matrix, thereby causing delayed wound healing. Increased collagenase activity and decreased collagen synthesis is found in diabetics with chronic hyperglycemia. In the hyperglycaemic state, numerous proteins and matrix molecules undergo a nonenzymatic glycosylation, resulting in advanced glycation end products (**AGEs**). The formation of AGEs is excessive in hyperglycaemic conditions. AGE formation cross-links collagen, making it less soluble and likely to be normally repaired or replaced, thereby making the tissue susceptible to breakdown.[26] The result of these changes in collagen metabolism is an alteration in normal homeostatic collagen turnover. This change in homeostasis may alter wound healing responses to chronic microbial wounding of the periodontium.

AGEs activate a receptor known as "receptor for AGEs" (**RAGE**), which is found in the periodontium.

The AGE-RAGE interaction on monocytes increases cellular oxidant stress and activates the transcription factor nuclear factor kappa B (**NF-kB**), which alters the phenotype of the monocyte/macrophage and results in the increased production of proinflammatory cytokines such as IL-1β and TNF-α.[27,28] These proinflammatory cytokines contribute to the pathogenesis of periodontal diseases and probably also play a major role in diabetics. Neutrophil adherence, chemotaxis, and phagocytosis are often impaired, which may inhibit bacterial killing and, therefore, increase periodontal destruction.[29,30]

The increased levels of circulating hormones during puberty cause a greater degree of inflammation and gingival bleeding, and there is a related shift in subgingival flora.[31,32] These alterations may cause exacerbation of gingivitis in diabetic children during puberty.

Nevertheless, poor oral hygiene maintenance due to poor manual tooth-brushing dexterity in children results in increased plaque accumulation, which can further exaggerate gingivitis in diabetic children. It has been reported that tooth-brushing by children younger than 10-years-old is inefficient.[33] This can be attributed to a lack of motivation and poor manual dexterity which is usual at this age.[34] Effective oral hygiene instructions should be imparted to the diabetic children. Instructions should be given according to the child's degree of readiness for tooth-brushing and should include systematic training and reinforcement. Children should be educated about oral self-care, and intensive individual training regarding oral hygiene maintenance should be imparted according to their level of psychological development.[35]

The parents should brush and floss their children's teeth until they develop sufficient manual dexterity to do so effectively. Establishing a consistent and effective daily oral care prevents gingivitis.

Pediatric health care providers should be alert for signs of gingival and periodontal disease in children and adolescents. Plaque and calculus deposits, which are the most important pathogenic factors responsible for gingivitis in the oral cavity, should be removed through careful self-care and regular professional care to reduce the risk of gingivitis and periodontitis in diabetic children. Correction of plaque-retentive factors should be considered, such as over-contoured crowns, open and/or overhanging margins, ill-fitting appliances, caries, and tooth malposition.

Reinforcement of oral hygiene instructions, regular professional oral prophylaxis, and the use of antimicrobial/antiplaque mouthwash and dental floss are highly recommended in diabetic pediatric patients. The pediatrician's concern is to maintain good metabolic control and to make young diabetic patients aware of a diet that suits their appropriate nutritional needs. It leaves no doubts, however, that in children with type 1 diabetes, proper management of the primary disease should be accompanied by prevention, early detection, and treatment of gingival and periodontal diseases. The assessment of gingival status reported here, as well as data reported by other authors, supports the assumption that children suffering from type 1 diabetes mellitus can be at risk of gingival and periodontal diseases. Metabolic imbalances in the tissues can lower the resistance of diabetics to infection and so influence the initiation, development, and progression of gingival and periodontal disease.

Monitoring the HbA1c (glycosylated hemogobulin) levels may improve treatment in diabetics. Further rigorous, systematic study is warranted to determine the magnitude and duration of improvement in glycemic control following treatment of gingivitis and periodontal infections in diabetic children so as to possibly reduce the burden of complications of diabetes mellitus.

Table 4. Comparison of mean plaque and gingival indices in control (C) and experimental (E) groups

Samples		N	Mean±(SD)	Std. error Mean
Primary dentition plaque index	C	24	0.50±0.21	0.04
	E	24	0.69±0.11	0.02
Primary dentition gingival index	C	24	0.42±0.50	0.10
	E	24	1.25±0.61	0.12
Mixed dentition plaque index	C	24	1.09±0.31	0.06
	E	24	1.34±0.23	0.05
Mixed dentition gingival index	C	24	0.54±0.59	0.12
	E	24	1.63±0.71	0.15
Permanent dentition plaque index	C	24	1.11±0.27	0.05
	E	24	1.35±0.20	0.04
Permanent dentition gingival index	C	24	0.58±0.58	0.12
	E	24	1.79±0.59	0.12

Table 5. Comparison of control and experimental groups by Student's t test

Samples	*t* test	df	Significance (2- tailed)	Mean difference±(SD)	95% confidence interval of the difference	
					Lower	Upper
Primary dentition plaque index	-4.023	46	0.00 (*P*<.01)*	-0.20±0.05	-0.29	-0.10
Primary dentition gingival index	-5.171	46	0.00 (*P*<.01) *	-0.83±0.16	-1.16	-0.51
Mixed dentition plaque index	-3.178	46	0.00 (*P*<.01) *	-0.25±0.08	-0.41	-0.09
Mixed dentition gingival index	-5.752	46	0.00 (*P*<.01) *	-1.08±0.19	-1.46	-0.70
Permanent dentition plaque index	-3.478	46	0.00 (*P*<.01) *	-0.24±0.07	-0.38	-0.10
Permanent dentition gingival index	-7.144	46	0.00 (*P*<.01) *	-1.21±0.17	-1.55	-0.87

** Statistically significant.*

CONCLUSIONS

In the present study,

1. Higher dental plaque levels and gingival scores were observed in type-I diabetic children vs non-diabetic controls; thus, children with type-I diabetes mellitus are possibly at risk for gingivitis.
2. Moderate gingivitis was the salient feature in diabetic children.

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