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Prevalence of Plaque-Induced Gingivitis in a Sample of the Adult Egyptian Population

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Abstract

AIM: The aim of this cross-sectional study is recording the prevalence and evaluation of the severity of plaque-induced gingival inflammation among a sample of the adult Egyptian population.

PATIENTS AND METHODS: Four hundred and twenty-five subjects in this contemplate were seen from patients visiting the diagnostic clinic at the Faculty of Dentistry, Cairo University. The gingival and plaque indices for each patient diagnosed as having plaque-induced gingivitis were recorded. The pocket depth was also measured.

RESULTS: The incidence of gingivitis was 100% amid adult subjects with an age range between 18-45 years. The average plaque index (PI) was 1.05 ± 0.43, which reflects relatively superior plaque control of the participants. The mean gingival index (GI) was 1.66 ± 0.40, which reflects the presence of moderate gingival inflammation. PD mean values confirmed limitation of inflammation to the gingiva. The sex was correlated to the condition of the inflamed gingiva ($p = 0.014$) and the quantity of biofilm build-up ($p = 0.003$). Females were less affected than males ($p = 0.005$).

CONCLUSION: The outcomes of this contemplate demonstrated that biofilm build-up is stoutly accompanied with elevated incidence of modest to rigorous gingivitis amid adult Egyptian individuals.

Introduction

Gingivitis is a reversible type of periodontal disease in which inflammation is limited to the gingiva without further destruction of the tooth-supporting components [1]. It is regarded as the second main and commonly occurring oral malady following dental caries, disturbing more than 75% of the populace global wise [2] [3] [4]. The incidence of gingival inflammation varies in the conducted studies among the different countries as a consequence of diversity in the studied populations, the age of the included subjects, and the methods of recognising and diagnosing this condition. Epidemiological studies discovered that plaque-induced gingivitis starts early in children, and becomes more common and aggressive with age and widely spreads among all ages [5] [6] [7] [8] [9]. It is caused by the increased accumulation of plaque biofilm near the gingival margins. The clinical features characteristic of this gingival inflammation are the erythematous and spongiotic texture of the gingiva; contour alterations; provoked bleeding; and occurrence of calculus, or plaque with no loss of clinical attachment, or radiographic substantiation of crestal bone resorption [10] [11]. Dental professionals advocated efficient oral hygiene measures to maintain optimal oral health aiming at controlling dental plaque and managing the inflammatory products liberated during the interactions between pathogenic microbiota and host response [12] [13] [4].

Clinically, the sternness and signs of the inflamed gingiva can be assessed by gingival index (GI) of Loe and Silness [15] [16]. In regards to this index, inflamed gingiva can be categorised as mild, moderate, or severe. The occurrence of these symbols of inflammation is regarded as the early phase of the more severe and irreparable form of periodontal destruction as in periodontitis cases. A subject's vulnerability to extend this form of the

disease also is greatly inconsistent and is dependent on the host retort towards perio-pathogens [17] [18] [19], which might be controlled by both acquired and genetic factors that can alter the vulnerability to infectivity [12] [20]. Avoidance of dental biofilm is amassing and untimely treatment of gingival inflammation diminishes the dangers aligned with the advancement of other serious and ruinous appearance of periodontitis [11] [21]. It is reported that gingivitis occurs past 10 - 21 days of biofilm amassing, [22] requiring a regular endeavour to counteract plaque accumulation. A few mulls over found a noteworthy relationship between diminishing the frequency of gingivitis and normal biofilm control procedures [23] [24] [25]. The aim of this study is record the incidence and to evaluate the rigorousness of plaque-induced gingivitis among a sample of the adult Egyptian populace.

Patients and Methods

The study protocol was approved by the Ethical Committee at the National Research Centre (NRC). Included subjects after explaining all the study procedures to them were asked to sign an informed consent stating their approval. Four hundred and twenty - five eligible participants in this contemplate were recruited from the routine dental patients who attended in the oral diagnosis clinic at the Faculty of Dentistry, Cairo University, Egypt. The medical history of each subject was recorded at the time of examination in a special recording form according to the Cornell Medical Index [26]. Exclusion criteria included subjects who were wearing fixed or removable prosthesis, or with orthodontic appliances. Also, subjects under current periodontal treatment, tobacco smokers, female subjects who were pregnant or using oral contraceptives were not included. Subjects with any other systemic conditions that are known to predispose, or exaggerate gingival inflammation, or any subject who was on antibiotics, antifungals, or antiseptic mouthwash for therapeutic reasons over the past 3 months, were not allowed to participate. A minimum of 20 permanent teeth had to be present for including the patient in the study.

The periodontal examination was performed by 2 dentists for all subjects in a dental chair, using a mouth mirror, and a calibrated Williams' periodontal probe [27]. Periodontal charting was made for all participants, and the data was recorded in a special diagnostic format. Periodontal health was defined as the complete absence of gingivitis at any site, and gingivitis was defined as inflammation of the gingiva in at least one site with the absence of clinical attachment loss [11]. Gingival health status was recorded for all study subjects using the gingival index (GI) of Loe and Silness [16]. Dental plaque status was also determined using plaque index (PI) of Silness and Loe [13]. Periodontal pocket depth was also assessed to exclude the presence of any evidence of crestal bone resorption using graduated Williams' probe and measured in mm. In accordance with the GI score, the subject's gingival health was assigned as follows: no inflammation (< 0.1); mild inflammation (0.1 - 1); moderate inflammation (1.1 - 1.9); and severe inflammation (2 - 3) [16]. For the PI score, the subject's plaque status was assigned as follows: excellent (< 0.1); good (0.1 - 0.9); fair (1 - 1.9); and poor (2 - 3) [13]. Oral hygiene care was conducted by the teaching of appropriate tooth brushing techniques and methods for plaque control.

Numerical data were presented as mean ± standard deviation (SD) values. Data were explored for normality using Kolmogorov - Smirnov test of normality. When variables were found to be normally distributed, independent student t-test was used to compare means of variables (clinical parameters) between the two groups (males and females). When variables were found to be non - normally distributed, Mann - Whitney test was used for comparing means of variables between the two groups. Data was collected and saved on a personal computer and analysed using the Statistical Package for Social Sciences (SPSS) software version 22 (IBM Corp, Armonk, NY, USA).

Results

The present study included 425 adult dentate subjects, 241 females (56.71%), and 184 males (43.29%), age range between 18 and 45 years with a mean age of 29.72 ± 6.41 years. The mean age for male subjects was 29.86 ± 6.85 years, which is not statistically significant than females age (29.56 ± 5.96) (p = 0.000). Participants were alienated into 3 groups as regards to their era variety as presented in Table 1.

Table 1: Demographic criteria of the subjects included in this study

Group	Age Range	Gender n (% of total)		Total no. of included subjects n (%)
		Males	Females	
1	18-25	32 (7.53)	130 (30.59)	162 (38.12)
2	26-35	62 (14.59)	67 (15.76)	129 (30.35)
3	36-45	90 (21.18)	44 (10.35)	134 (31.53)
Total		184 (43.30)	241 (56.70)	425 (100)

The average PI for the entire patients was 1.05 ± 0.43, which reflects a modest plaque status of the subjects. Male participants had more plaque buildup in comparison to females (p = 0.003) as presented in table 2 and figure 1. The average PI for males was 1.08 ± 0.44 and 1.01 ± 0.42 for females. Additionally, gingival healthiness and quantity of biofilm amassing were not correlated to the era of the examined participants (p = 0.53). The sex was

interrelated to the inflammation present (p = 0.014) and the quantity of biofilm buildup (p = 0.003). Females were less affected than males (p = 0.005).

Table 2: The plaque grade of the subjects included in this study

Plaque Status	Gender n (% of total)		Total	p-value
	Male	Female		
Excellent	4 (2.17)	18 (7.47)	22 (5.18)	0.063
Good	79 (42.92)	156 (64.73)	235 (55.29)	0.074
Fair	93 (50.54)	60 (24.89)	153 (36)	0.003*
Poor	8 (4.37)	7 (2.91)	15 (3.53)	0.122
Total	184 (43.30)	241 (56.70)	425 (100)	

*significant.

The outcomes of this contemplate revealed that 100% of all subjects presented with a variety of inflammation signs (GI scores were more than 0.1). The average GI score for participants was 1.66 ± 0.40, which shows a modest gingivitis. Females had fewer indications of gingival inflammation in contrast to males (*p* = 0.001) as revealed in Table 3 and Figure 2.

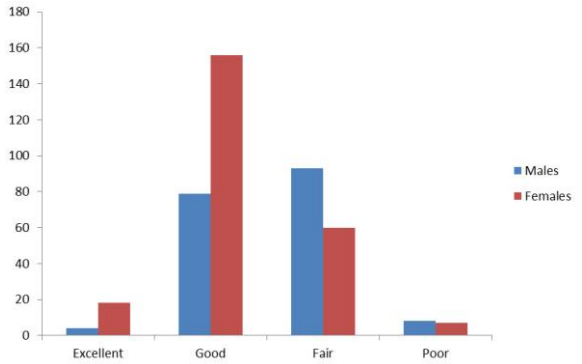

Figure 1: Histogram showing plaque scores among males and females

The mean GI for males was 1.72 ± 0.45, and 1.3 ± 0.35 for females. 110 patients (25.88%) had a GI score equivalent to 2, which demonstrates stern gingivitis and slight flow of blood on probing. Bleeding gingiva on probing was considerably lower in females 42.2% as compared with males 54.4% (*p* = 0.016).

Table 3: The gingival health status of subjects in this study

Gingival Health Status	Gender n (% of total)		Total	p-value
	Male	Female		
No inflammation	0 (0.0)	0 (0.0)	0 (0.0)	-------
Mild GI	4 (2.18)	23 (9.54)	27 (6.36)	0.003*
Moderate GI	102 (55.43)	186 (77.18)	288 (67.76)	0.014*
Severe GI	78 (42.39)	32 (13.28)	110 (25.88)	0.588
Total	184 (43.30)	241 (56.70)	425 (100)	-------

*significant.

Discussion

Assessing the prevalence of gingival inflammation caused by plaque biofilm accumulation in adulthood worldwide is complicated as a consequence of the variety of study populations, hereditary background, and contributing ecological factors. Additionally, the existence of various clinical methodologies for diagnosing and defining gingival inflammation and lack of evident objective cut-off points between health and disease add to the complexity.

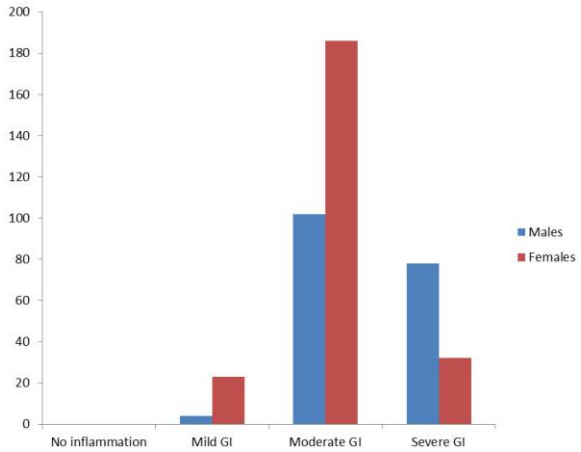

Figure 2: Gingival health status among males and females

Numerous previously conducted epidemiological contemplates revealed that the incidence of gingivitis in adults is changeable around 50-100% for dentulous subjects [5]. In the present contemplate; the incidence of gingivitis was 100% amid the examined participants with an age range between 18 and 45 years. Unlike various studies, there were no preliminary or washout periods or any oral hygiene education before the oral assessment. Subjects included did not change their diet or regular practice to diminish the effects of these parameters on dental plaque. Subjects did utilise dental treatment services only in case of pain or other emergencies [26] [27] [28] [29].

A study on Chinese subjects ageing between 18 and 90 years performed by Zhang et al. [27] revealed the presence of gingival inflammations in 97.9% of subjects examined. Also, Li et al. [29] documented that gingivitis was detected in 95.7% of American adult subjects with age ranging 18 - 90 years. A study conducted among the Saudi adult population by Idrees et al. 2014 [29] revealed the occurrence of gingival inflammations in 100%. This in line with the present results obtained in this contemplate which confirmed the presence of gingival inflammations in 100% of participants.

In two earlier investigations, the decisive factors for defining the presence of gingivitis was GI = 0.5, or above. Gingivitis should be detected at three sites or more to be regarded as inflammation. On the contrary, our criteria for pointing out gingivitis is its presence in at least a single location, or GI > 0.1 [29], and this revealed the elevated incidence of this condition in the present contemplate as comparable to other research results with this narrow age range (18 - 45 years) [27] [28].

In the present contemplate, the predominance of participants with gingival bleeding was 25.88%, and it was superior in men as compared to women. An elevated incidence of gingival bleeding in men was also recorded 2009 in Australia [30] among male participants who confirmed that the existence of accumulated plaque deposits is intimately related to the occurrence of gingivitis [13] [30] [31]. This is by the present study. The obtained results in this study are in line with earlier studies that clarified the significant correlation of sex with gingival illness and biofilm buildup [5] [27] [29]. This might be because men are less anticipated to visit the dental practitioner regularly with their poor state of mind towards wellbeing in comparison to females [32]. This contemplates demonstrated that era was not correlated with the presence of gingivitis and the measure of biofilm amassing. This might be due to the restricted age scope of included subjects in the vicinity of 18 and 45 years as in contrast to other conducted studies [5] [28] [29]. Also, firm prohibition criterions have been assumed to play a critical role to achieve these outcomes. As indicated by the World Health Organization, the era aggregate between 35 - 44 years was regarded as the principle set since the majority populaces at this age range showed signs of oral illness and diverse forms of periodontal diseases [33]. Furthermore, the occurrence of periodontitis rises with the era. Adults more than 50 years have the greatest jeopardy for being involved [33] [34]. Zhang et al. [28] confirmed that the age set older than 59 years had considerably superior GI in comparison to youth. Our outcomes are constant with a former contemplate that showed no interrelationships between era and gingival inflammation [29] [30]. The results are similar to a report published by the Saudi National Office of Statistics and a study conducted among the Saudi adult population [29].

The mean results for gingivitis had resemblance also to those revealed in former studies of 0.99 from Saudi Arabia, 1.23 from Swiss people, 1.2 from the Gambia, 1.05 from the USA, and 1.1 from China representing populations from different regions [35].

In a study conducted on 1650 adults from three South American cities, the results revealed 95.6% with a GI ≥ 0.5. This data corroborates the information presented for adults from Jordan and United States of America which showed 75.8% and 93.9% respectively [36].

In conclusion, this study despite its limitations demonstrated that plaque amassing is firmly connected with an elevated predominance of modest to severe gingivitis among the sample of the Egyptian subjects seen. Additional investigations are mandatory to perceive the causes that may add to this elevated incidence of plaque-induced gingivitis. Public preventive outlines ought to be evaluated and re-actualised on a broad and efficient level.

References

1. Yiming LI, Sean LEE, Philippe Hujoel, Mingfang SU, Wu Zhang, Jay Kim, Yun Po Zhang, William Devizio. Prevalence and severity of gingivitis in American adults. American Journal of Dentistry. 2010; 23:1-13.

2. Califano JV. Research, Science and Therapy Committee American Academy of Periodontology. Position paper: periodontal diseases in children and adolescents. J Periodontol. 2003; 74:1696-1704. https://doi.org/10.1902/jop.2003.74.11.1696 PMid:14682670

3. Angst, PD, Piccinin, FB, Oppermann, RV, Marcantonio, RA, Gomes, SC. The response of molars and non-molars to a strict supragingival control in periodontal patients. Braz Oral Res. 2013; 27(1):55-60. https://doi.org/10.1590/S1806-83242013000100010 PMid:23306627

4. Rebelo MA, Lopes MC, Vieira JM, Parente, RC. Dental caries and gingivitis among 15 to 19 year-old students in Manaus, AM, Brazil. Braz Oral Res. 2009; 23(3):248-254. https://doi.org/10.1590/S1806-83242009000300005 PMid:19893958

5. Ababneh KT, Abu Hwaij ZM, Khader YS. Prevalence and risk indicators of gingivitis and periodontitis in amulti-centre study in North Jordan: a cross sectional study. BMC Oral Health. 2012; 12:1. https://doi.org/10.1186/1472-6831-12-1 PMid:22214223 PMCid:PMC3271955

6. Chrysanthakopoulos NA. Prevalence of gingivitis and associated factors in 13–16-year-old adolescents in Greece. European Journal of General Dentistry. 2016; 5:58-64. https://doi.org/10.4103/2278-9626.179536

7. Gopinath V. Oral hygiene practices and habits among dental professionals in Chennai. Indian J Dent Res. 2010; 21:195-200. https://doi.org/10.4103/0970-9290.66636 PMid:20657087

8. Jain N, Mitra D, Ashok KP, Dundappa J, Soni S, Ahmed S. Oral hygiene-awareness and practice among patients attending OPD at Vyas Dental College and Hospital, Jodhpur. J Indian Soc Periodontol. 2012; 16(4):524-528. https://doi.org/10.4103/0972-124X.106894 PMid:23493177 PMCid:PMC3590720

9. Jain M, Mathur A, Kumar,S, Duraiswamy P, Kulkarni S. Oral hygiene and periodontal status among Terapanthi Svetambar Jain monks in India. Braz Oral Res. 2009; 23(4):370-376. https://doi.org/10.1590/S1806-83242009000400004 PMid:20027442

10. Jordan RA, Lucaciu A, Fotouhi K, Markovic L, Gaengler P, Zimmer S. Pilot pathfinder survey of oral hygiene and periodontal conditions in the rural population of The Gambia (West Africa). Int J Dent Hyg. 2011; 9(1):53-59. https://doi.org/10.1111/j.1601-5037.2009.00435.x PMid:21226851

11. Oswal KC. Oral hygiene practice amongst patients visiting Terna Dental College. J Oral Health Comm Dent. 2013; 7:33–36.

12. Marsh PD. Contemporary perspective on plaque control. Br Dent J. 2012; 212(12):601-606. https://doi.org/10.1038/sj.bdj.2012.524 PMid:22722123

13. Mizutani S, Ekuni D, Furuta M, Tomofuji T, Irie K, Azuma T, Kojima A, Nagase J, Iwasaki Y, Morita M. Effects of self-efficacy on oral health behaviours and gingival health in university students aged 18- or 19-years-old. J Clin Periodontol. 2012; 39 (9):844-849. https://doi.org/10.1111/j.1600-051X.2012.01919.x PMid:22780323

14. Poyato-Ferrera M, Segura-Egea JJ, Bullón-Fernández P. Comparison of modified Bass technique with normal tooth brushing practices for efficacy in supragingival plaque removal. Int J Dent Hyg. 2003; 1(2):110-114. https://doi.org/10.1034/j.1601-5037.2003.00018.x PMid:16451532

15. Loe H. The Gingival Index, the Plaque Index and the Retention Index Systems. J Periodontol. 1967; 38(6):610-616. https://doi.org/10.1902/jop.1967.38.6.610 PMid:5237684

16. Loe H, Silness J. Periodontal disease in pregnancy. I.

prevalence and severity. Acta Odontol Scand. 1963; 21:533-551. https://doi.org/10.3109/00016356309011240 PMid:14121956

17. Lang NP, Schätzle MA, Löe H. Gingivitis as a risk factor in periodontal disease. J Clin Periodontol. 2009; 36(10):3-8. https://doi.org/10.1111/j.1600-051X.2009.01415.x PMid:19432625

18. Pari A, Ilango P, Subbareddy V, Katamreddy V, Parthasarthy H. Gingival diseases in childhood – A review. J Clin Diagn Res. 2014; 8:ZE01-4. https://doi.org/10.7860/JCDR/2014/9004.4957

19. Broadbent JM, Thomson WM, Boyens JV, Poulton R. Dental plaque and oral health during the first 32 years of life. J Am Dent Assoc. 2011; 142(4):415-26. https://doi.org/10.14219/jada.archive.2011.0197 PMid:21454848

20. Antunes JL, Peres MA, Frias AC, Crosato EM, Biazevic MG. Gingival health of adolescents and the utilization of dental services, state of São Paulo, Brazil. Rev Saude Publica. 2008; 42:191-199. https://doi.org/10.1590/S0034-89102008000200002 PMid:18372971

21. Baehni PC, Takeuchi Y. Anti-plaque agents in the prevention of biofilm-associated oral diseases. Oral Dis. 2003; 9(1):23-29. https://doi.org/10.1034/j.1601-0825.9.s1.5.x PMid:12974527

24. Jain Y. A comparison of the efficacy of powered and manual toothbrushes in controlling plaque and gingivitis: a clinical study. Clin Cosmet Investig Dent. 2013; 5:3-9. https://doi.org/10.2147/CCIDE.S40656 PMid:23674927 PMCid:PMC3652371

25. Rode Sde M, Gimenez X, Montoya VC, Gomez M, Blanc SL, Medina M, et al. Daily biofilm control and oral health: consensus on the epidemiological challenge--Latin American Advisory Panel. Braz Oral Res. 2012; 26(1):S133-S143. https://doi.org/10.1590/S1806-83242012000700020

27. Zhang J, Xuan D, Fan W, Zhang X, Dibart S, De Vizio W, et al. Severity and prevalence of plaque-induced gingivitis in the Chinese population. Compend Contin Educ Dent. 2010; 31(8):624-629. PMid:20960992

28. Li Y, Lee S, Hujoel P, Su M, Zhang W, Kim J, et al. Prevalence and severity of gingivitis in American adults. Am J Dent. 2010; 23(1):9-13. PMid:20437720

29. Majdy M. Idrees, Saleh N. Azzeghaiby, Mohammad M. Hammad, Omar B. Kujan. Prevalence and severity of plaque-induced gingivitis in a Saudi adult population. Saudi Med J. 2014; 35(11):1373-1377.

30. Agnes O. Umoh, Clement C. Azodo. Prevalence of gingivitis and periodontitis in an adult male population in Nigeria. Nigerian Journal of Basic and Clinical Sciences 2012; 9(2):65-69. https://doi.org/10.4103/0331-8540.108465

31. Australian Research Centre for Population Oral Health, The University of Adelaide, South Australia. Periodontal diseases in the Australian adult population. Aust Dent J. 2009; 54(4):390-393. https://doi.org/10.1111/j.1834-7819.2009.01167.x PMid:20415940

32. Burt B. Research, Science and Therapy Committee of the American Academy of Periodontology. Position paper: epidemiology of periodontal diseases. J Periodontol. 2005; 76(8):1406-1419. https://doi.org/10.1902/jop.2005.76.8.1406 PMid:16101377

33. Greenwell H. Committee on Research, Science and Therapy. American Academy of Periodontology. Position paper: Guidelines for periodontal therapy. J Periodontol. 2001; 72(11):1624-1628. https://doi.org/10.1902/jop.2001.72.11.1624 PMid:11759876

34. Farsi JM. Dental visit patterns and periodontal treatment needs among Saudi students. East Mediterr Health J. 2010; 16(7):801-806. PMid:20799540

35. Sreenivasan PK, Prasad KVV, Javali SB. Oral health practices and prevalence of dental plaque and gingivitis among Indian adults. Clinical and Experimental Dental Research. 2016; 2(1):6-17. https://doi.org/10.1002/cre2.15

36. Carvajal P, Gómez M, Gomes S, Costa R, Toledo A, Solanes F, Romanelli H, Oppermann R, Rösing C, Gamonal J. Prevalence, severity, and risk indicators of gingival inflammation in a multi-center study on South American adults: a cross sectional study. Journal of Applied Oral Science. 2016; 24(5):524-34. https://doi.org/10.1590/1678-775720160178 PMid:27812624 PMCid:PMC5083031