The Effect of Smoking on Periodontal Status of Type- 2 Diabetic Patients in Indonesia: A Pilot Study

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Abstract

The study is designed to compare periodontal status of type-2 diabetic smokers, diabetic non-smokers, and non-diabetic non-smokers. Periodontal status of well-controlled and poorly controlled diabetic smokers and non-smokers was also compared. Cross-sectional study was done on 119 patients with chronic periodontitis, consisting of 20 diabetic smokers (group I), 59 diabetic non-smokers (group II), and 40 non-diabetic non-smokers (group III). Clinical status of all teeth was assessed using plaque index (PI), calculus index (CI), and oral hygiene index (OHI). Periodontal examination was evaluated using papillary bleeding index (PBI), pocket depth (PD), gingival recession (GR), and clinical attachment level (CAL). Group 1 and 2 were further divided into 4 groups: well-controlled diabetic smokers and non-smokers, and poorly controlled diabetic smokers and non-smokers.

Comparison of clinical and periodontal parameters were also done. Results showed significant differences in CI, PBI, PD, GR, and CAL between diabetic groups, both smokers and non-smokers, and non-diabetic non-smokers. No significant differences were found in any parameters between well-controlled diabetic smokers and non-smokers, as well as poorly controlled diabetic smokers and non-smokers.

In conclusion, poor glycemic level can aggravate periodontal breakdown in chronic periodontitis patients. Nevertheless, smoking seems to have little effect on the periodontal destruction in diabetic patients with chronic periodontitis.

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Introduction

Periodontitis is an inflammatory condition which affects supporting structures of the teeth.[1] It is caused by specific microorganisms or groups of specific microorganisms that leads to progressive destruction of the periodontal ligament and alveolar bone.[1] Clinical signs of inflammation such as changes in gingival colour, contour, consistency, as well as bleeding tendency may accompany periodontitis.[1] There will also be periodontal pocket formation, gingival recession, and clinical attachment loss.[1]

Periodontal disease is an oral health problem that has the second highest prevalence worldwide, after caries.[2] The latest Global Burden of Disease Study (1990-2010) stated that severe periodontitis was the 6th most prevalent condition worldwide, affecting around 743 million people or around 11.2% of the population.[3] Prevalence also increased gradually with age, with a sharp increase between the third and fourth decades of life.[3] In Indonesia, periodontitis is a major public health problem with a much higher prevalence of 74.1% according to the National Health Survey (Riskesdas) 2018.[4] Periodontal patients are at risk of mobility and tooth loss, disability, masticatory dysfunction, and poor nutritional status.[5] Periodontitis also affects speech, reduces quality of life, and puts a huge economic burden to the country.[5,6] In South East Asia alone, productivity losses due to untreated periodontitis were estimated at 1,486.59 US$ Million, while that of tooth loss were around 767.89 US$ Million.[6]

Among the many groups of periodontal disease, chronic periodontitis is the most

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prevalent type of periodontitis and is characterised by the slowly progressing nature of the disease.[7] The main causative factor for chronic periodontitis is the quantity of dental biofilm and the composition of the oral microflora.[7] Other risk factors such as local, systemic, immunologic, genetic, and behavioural factors may further predispose this inflammatory condition.[7] Amidst these factors, diabetes mellitus and tobacco smoking are known to be closely-linked to the development and progression of chronic periodontitis.[8]

Diabetes mellitus (DM) refers to a disease with abnormal carbohydrate, fat, and protein metabolism which results in an elevated blood glucose level.[9] Diabetes mellitus is a global health problem and its prevalence has escalated drastically from 108 million in 1980 to 422 million in 2014.[10] Its prevalence has been rising more quickly in middle- and low-income countries including Indonesia, whose prevalence is around 1.5% in 2018, or around 4 million people.[4,10] Many complications are associated to DM such as retinopathy, neuropathy, nephropathy, cardiovascular disease and peripheral vascular disease.[9] A study by Loe confirms that periodontitis is recognised as the 6th complication of diabetes.[11] Scientific evidences pointed out a bidirectional inter-relationship between DM and periodontal disease.[12–14] Ramli, et al. conducted a case-control study between diabetic and non-diabetic patients, and found out that percentage of diabetic patients that were diagnosed with periodontitis (88.1%) was statisticallly higher compared to those of control group (59.5%).[15] Periodontal infection is linked to declining glycemic control in diabetic patients, while DM is also associated with an increased incidence and progression of periodontitis.[12,13] Improving glycemic control also improves periodontal condition in diabetic patients.[14]

Another major public health threats the world has ever encountered is tobacco epidemic. It has killed more than 8 million people a year worldwide, consisting of 7 million deaths resulting from direct tobacco use and 1.2 million deaths from passive-smoking.[16] There is around 1.1 billion smokers globally who mostly live in low- and middle-income countries.[16] Indonesia is ranked 7th among countries with the highest smoking rate, reaching a high value of 39.9%.[17] It is a well-known fact that smoking is associated with increased incidence and progression of periodontitis.[2,18] Smoking changes subgingival bacterial profile in healthy individuals, decreasing beneficial bacteria while also increasing periodontopathogenic bacteria.[19] Nevertheless, there are some conflicting results regarding the effect of smoking on diabetic patients. Obradovic, et al. demonstrated that periodontal disease was more severe in diabetic smokers compared to diabetic non-smokers. The study concluded that smoking negatively affected the course of periodontal disease in diabetic patients and increased the risk of clinical attachment loss.[20] Another study by Gupta et al. also reported that clinical periodontal parameters and the mean levels of salivary MMP-8 were significantly higher in diabetic smokers compared to other groups. This strongly suggests that diabetes and smoking plays an important part in the incidence and severity of periodontal disease.[21] However, another study by Javed et al. yielded different result, whereby periodontal inflammatory conditions of smokers and non-smokers with type-2 Diabetes Mellitus were comparable.[22]

To the authors' knowledge, there are no previous studies that investigate the effect of smoking on periodontal condition of diabetic patients in Indonesia. As such, the purpose of this pilot study is to compare the periodontal status of diabetic smokers, diabetic non-smokers, and non-diabetic non-smokers. In addition, the study also wants to compare the periodontal status of well-controlled and poorly controlled diabetic smokers and non-smokers.

Materials and methods

The study was a cross-sectional study approved by the ethical committee of the School of Medicine and Health Sciences Atma Jaya Catholic University of Indonesia and Atma Jaya Teaching and Research Hospital. Subjects were recruited from outpatients visiting dental clinic at Atma Jaya Teaching and Research Hospital between September 2017 to March 2018. The target population was all patients with chronic periodontitis according to the American Academy of Periodontology International Workshop for Classification of Periodontal Diseases.[23] Subjects were selected using purposive sampling method. All subjects were informed about the study, possible discomforts and risks, as well as possible benefits. Then, they signed written consent forms, were interviewed, and examined.

Subjects were divided into three groups: group 1 for diabetic smokers, group 2 for diabetic non-smokers, and group 3 (control) for non-diabetic non-smokers. Diabetic patients were patients who had been diagnosed with type-2 DM according to the Indonesian Endocrinology Association (*PERKENI*), while smokers were defined according to WHO's Smoking and Tobacco Use Policy.[24,25] Group 1 and 2 consisted of subjects with type 2 DM diagnosed by primary care physicians and internists at Atma Jaya Teaching and Research Hospital. The difference was that subjects of group 1 smoked any tobacco product, either daily or occasionally, while group 2 did not. Group 3 consisted of non-smoking subjects with no systemic disease. Next, group 1 was further categorised into well-controlled and poorly controlled diabetic smokers, and group 2 was also divided into well-controlled and poorly controlled diabetic non-smokers. Well-controlled and poorly controlled diabetes were classified according to glycemic targets by American Diabetes Association, which was 7.0.[26] Exclusion criteria for this study would be subjects who had undergone periodontal treatment for the past 6 months, were taking drugs that affect the healing of periodontal tissue, and had ≤ 6 teeth. Subjects who were pregnant and had any other systemic diseases were also excluded.

A detailed history of each subject pertaining to age, sex, smoking status, systemic, and oral disease was recorded. Measurement of HbA1C was only done to diabetic subjects. Subjects visited dental clinic of Atma Jaya Teaching and Research Hospital to undergo clinical examinations by one of the two examiners who had undergone interexaminer and intraexaminer calibration process. Dental mirrors and explorers (Crown, Japan) were used to measure plaque index (PI) and calculus index (CI) according to Loe & Silness.[27,28] Oral hygiene index was obtained by adding PI and CI. Periodontal examination was evaluated by measuring papillary bleeding index (PBI) according to Saxer and Muehlemann [29], periodontal pocket depth (PD), gingival recession (GR), and clinical attachment level (CAL). Clinical measurements of PD, GR, and CAL were carried out in all teeth, except for third molars. Measurements of PD, GR, and CAL were done at 6 sites per tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual/palatal, mid-lingual/palatal, and disto-lingual/palatal). The value for PD, GR, and CAL recorded in the data was taken from the site with the greatest CAL and its associated PD and GR.[30] All periodontal examinations were recorded using periodontal probe (Hu-Friedy, USA). The severity of periodontitis was classified according to the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.[30]

Statistical analysis was conducted using SPSS 22.0. Numerical data was summarized as mean ± standard deviation (SD) while categorical data was shown as value and percentage. Comparison between PI, CI, OHI, PBI, PD, GR, and CAL among the three initial groups were assessed using one One-Way Anova and Post-hoc analysis Bonferroni for normally distributed data or Kruskal-Wallis and Mann-Whitney for abnormally distributed data. A further comparison between well-controlled diabetic smokers and non-smokers, and poorly controlled diabetic smokers and non-smokers were done using Independent T-Test or Mann-Whitney alternative. A value of $P < 0.05$ was considered statistically significant.

Results

This study included a total of 119 subjects which consisted of 20 diabetic smokers, 59 diabetic non-smokers, and 40 non-diabetic non-smokers (table 1).

	Diabetic Smokers (n=20)	Diabetic Non-Smokers (n=59)	Non-Diabetic Non-Smokers (n=40)
Age (year)	53.80 ± 7.39	55.51 ± 6.33	52.23 ± 8.66
Gender			
Male	16 (80%)	17 (29%)	25(63%)
Female	4 (20%)	42 (71%)	15 (37%)
HbA1C (%)	7.63 ± 1.54	7.93 ± 2.16	-
Dental visit history of the last 6 months	1 (5%)	11 (19%)	5 (13%)
Toothbrush frequency			
1x/day	1(5%)	11 (19%)	4 (10%)
2x/day	17 (85%)	39 (66%)	25 (63%)
3x/day	2 (10%)	8 (13%)	11 (27%)
4x/day	0	1 (2%)	0
Use of mouthwash	3 (15%)	4 (7%)	2 (5%)
Use of dental floss	2 (10%)	4 (7%)	3 (8%)
Periodontitis Severity			
Stage II	3 (15%)	4 (7%)	8 (20%)
Stage III	17 (85%)	55 (97%)	32 (80%)

Table 1. Demographic Data of Diabetic Smokers, Diabetic Non-Smokers, and Non-diabetic Non-Smokers.

Age of the subjects ranged between 32 and 70 years old, with mean age of 53.80 ± 7.39 for diabetic smokers, 55.51 ± 6.33 for diabetic non-smokers, and 52.23 ± 8.66 for non-diabetic non-smokers. There were 58 males and 61 females participated in the study. More male

subjects were found in diabetic smokers (80%) and non-diabetic non-smokers (63%), while only 29% male subjects were found in diabetic non-smokers. HbA1C value for diabetic groups were comparable, with a value of 7.63 ± 1.54 for diabetic smokers and 7.93 ± 2.16 for diabetic non-smokers. The percentage of subjects who had dental visit history for the last 6 months varied from 5% in diabetic smokers to 13% in non-diabetic non-smokers. Most subjects brushed their teeth 2x/day, and this was noted in 85% diabetic smokers, 66% diabetic non-smokers, and 63% non-diabetic non-smokers. Nevertheless, only 5-15% of subjects in each group included mouthwash and 7-10% included flossing in their oral hygiene procedure. In all groups, more than 80% of the subjects suffered from stage III periodontitis, which was characterised by interdental clinical attachment loss of ≥ 5 mm at site of greatest loss.

Clinical Parameters	Diabetic Smokers (n=20)	Diabetic Non-Smokers (n=59)	Non-Diabetic Non-Smokers (n=40)	*P* Value
Plaque Index (PI)	1.37 ± 0.69	1.44 ± 0.69	1.42 ± 0.57	0.91
Calculus Index (CI)	1.90 ± 0.92	1.68 ± 0.84	1.10 ± 0.72	*p* < 0.001*
Oral Hygiene Index (OHI)	3.27 ± 1.51	3.13 ± 1.30	2.99 ± 1.18	0.72
Papillary Bleeding Index (PBI)	1.10 ± 0.92	1.07 ± 1.04	1.57 ± 0.79	0.003*
Pocket Depth (PD) /mm	3.75 ± 2.63	4.03 ± 1.90	5.10 ± 0.84	*p* < 0.001*
Gingival Recession (GR) /mm	5.15 ± 3.54	3.69 ± 2.79	0.48 ± 0.85	*p* < 0.001*
Clinical Attachment Level (CAL) / mm	8.90 ± 4.14	7.73 ± 3.01	5.57 ± 1.24	*p* < 0.001*

Table 2. Comparison of Clinical Parameters among Diabetic Smokers, Diabetic Non-Smokers, and Non-Diabetic Non-Smokers

Statistical analysis using One Way Anova for PI and OHI, and Kruskal-Wallis for CI, PBI, PD, GR, and CAL.* Statistically significant if P<0.05.

Clinical Parameters	*P* Value		
	Diabetic Smokers vs Diabetic Non-Smokers	Diabetic Smokers vs Non-Diabetic Non-Smokers	Diabetic Non-Smokers vs Non-Diabetic Non-Smokers
Plaque Index (PI)	1.00	1.00	1.00
Calculus Index (CI)	0.32	0.001*	0.001*
Oral Hygiene Index (OHI)	1.00	1.00	1.00
Papillary Bleeding Index (PBI)	0.72	0.048*	0.001*
Pocket Depth (PD) /mm	0.25	0.003*	*p*<0.001*
Gingival Recession (GR) /mm	0.11	*p*<0.001*	*p*<0.001*
Clinical Attachment Level (CAL) /mm	0.38	0.002*	*p*<0.001*

Table 3. Statistical Comparison of Clinical Parameters between Study Groups.

Statistical analysis using post-hoc analysis Bonferroni for PI and OHI, and Mann-Whitney for CI, PBI, PD, GR, and CAL.* Statistically significant if *P*<0.05.

Clinical parameters and their associated statistical comparisons were shown in table 2 and 3. The mean plaque index of diabetic smokers was 1.37 ± 0.69, which was lowest compared to diabetic non-smokers (1.44 ± 0.69) and non-diabetic non-smokers (1.42 ± 0.57). However, there was no difference in the mean PI between all groups. The mean calculus index of diabetic smokers (1.90 ± 0.92) was higher compared to diabetic non-smokers (1.68 ± 0.84) and non-diabetic non-smokers (1.10 ± 0.72). There were significant differences in mean CI of both diabetic groups compared to control (*P*<0.001), but no difference was found between diabetic smokers and non-smokers. The mean oral hygiene index of all groups was comparable, which was 3.27 ± 1.51 for diabetic smokers, 3.13 ± 1.30 for diabetic non-smokers, and 2.99 ± 1.18 for non-diabetic non-smokers. No significant differences were found among them.

There was no significant difference between the mean papillary bleeding index of diabetic smokers and diabetic non-smokers, which was 1.10 ± 0.92 and 1.07 ± 1.04 respectively. However, both values were significantly lower compared to non-diabetic non-smokers at 1.57± 0.79 (*P*=0.003). The highest mean pocket depth was found in non-diabetic non-smokers (5.10 ± 0.84 mm), followed by diabetic non-smokers (4.03 ± 1.90 mm) and diabetic smokers (3.75 ± 2.63 mm). Significant differences were found between diabetic smokers and control (*P*=0.003), as well as between diabetic non-smokers and control (*P*<0.001)

On the contrary, the mean gingival recession of diabetic smokers (5.15 ± 3.54 mm) was higher compared to diabetic non-smokers (3.69 ± 2.79 mm) and non-diabetic non-smokers (0.48 ± 0.85 mm). There were significant differences in mean GR between diabetic groups and non-diabetic group (*P*<0.001). No significant difference was found between diabetic smokers and diabetic non-smokers. The mean clinical attachment level was highest for diabetic smokers (8.90 ± 4.14 mm), followed by diabetic non-smokers (7.73 ± 3.01 mm) and non-diabetic non-smokers (5.57 ± 1.24 mm). Again, significant were found between diabetic smokers and control (*P*=0.002), and diabetic non-smokers and control (*P*<0.001). No difference was found between diabetic smokers and diabetic non-smokers.

The researchers reclassified diabetic smokers into 2 groups, namely the well-controlled and poorly controlled diabetic smokers. The same thing was done to diabetic non-smokers group, which was divided into well-controlled and poorly controlled diabetic non-

smokers. Then, the researchers compared the clinical parameters between well-controlled diabetic smokers and non-smokers (table 4), as well as between poorly controlled diabetic smokers and non-smokers (table 5).

Clinical Parameters	Well-Controlled Diabetic Smokers (n=8)	Well-Controlled Diabetic Non-Smokers (n=26)	*P* Value
HbA1C	6.04 ± 0.34	6.18 ± 0.80	0.21
Plaque Index (PI)	1.29 ± 0.60	1.25 ± 0.63	0.87
Calculus Index (CI)	1.80 ± 0.85	1.48 ±0.84	0.36
Oral Hygiene Index (OHI)	3.09 ± 1.36	2.73 ± 1.36	0.52
Papillary Bleeding Index (PBI)	0.83 ± 0.65	0.76 ± 0.90	0.57
Pocket Depth (PD) /mm	3.38 ± 1.51	4.15 ± 1.74	0.26
Gingival Recession (GR) /mm	4.63 ± 4.00	3.50 ± 2.49	0.34
Clinical Attachment Level (CAL) /mm	8.00 ± 3.93	7.65 ± 2.95	0.95

Table 4. Comparison of Clinical Parameters between Well-Controlled Diabetic Smokers and Non-Smokers.

Statistical analysis using Independent T-Test for PI, CI, OHI, and GR, and Mann-Whitney for HbA1C, PBI, PD, and CAL.* Statistically significant if *P*<0.05.

Clinical Parameters	Poorly Controlled Diabetic Smokers (n=12)	Poorly Controlled Diabetic Non-Smokers (n=33)	*P* Value
HbA1C	8.68 ± 0.98	9.30 ± 1.88	0.53
Plaque Index (PI)	1.43 ± 0.77	1.59 ± 0.71	0.40
Calculus Index (CI)	1.97 ± 1.00	1.84 ± 0.81	0.68
Oral Hygiene Index (OHI)	3.39 ± 1.65	3.44 ± 1.18	0.91
Papillary Bleeding Index (PBI)	1.30 ± 1.04	1.32 ± 1.08	0.91
Pocket Depth (PD) /mm	4.00 ± 3.22	3.94 ± 2.05	0.52
Gingival Recession (GR) /mm	5.50 ± 3.34	3.85 ± 3.03	0.10
Clinical Attachment Level (CAL) /mm	9.50 ± 4.34	7.79 ± 3.10	0.33

Table 5. Comparison of Clinical Parameters between Poorly Controlled Diabetic Smokers and Non-Smokers.

Statistical analysis using Independent T-Test for CI and OHI, and Mann-Whitney for HbA1C, PI, PBI, PD, GR and CAL.* Statistically significant if P<0.05.

Table 4 showed that HbA1C of well-controlled diabetic smokers (6.04 ± 0.34 %) and non-smokers (6.18 ± 0.80 %) were comparable and not statistically significant. It was also found that plaque index, calculus index, and oral hygiene index of well-controlled diabetic smokers were higher compared to the non-smokers group (1.29 ± 0.60 vs 1.25 ± 0.63 for PI, 1.80 ± 0.85 vs 1.48 ±0.84 for CI, and 3.09 ± 1.36 vs 2.73 ± 1.36 for OHI). Likewise, papillary bleeding index, gingival recession, and clinical attachment level of well-controlled diabetic smokers were higher compared to diabetic non-smokers (0.83 ± 0.65 vs 0.76 ± 0.90 for PBI, 4.63 ± 4.00 mm vs 3.50 ± 2.49 mm for GR, and 8.00 ± 3.93 mm vs7.65 ± 2.95 mm for CAL). Pocket depth of well-controlled diabetic non-smokers (4.15 ± 1.74 mm) was the only variable who had higher value compared to diabetic smokers group (3.38 ± 1.51 mm). No significant difference was found in any of the clinical parameters between the two groups.

Table 5 showed the comparison of clinical parameters between poorly controlled diabetic smokers and non-smokers. HbA1C of the non-smokers group (9.30 ± 1.88 %) was higher than the smokers group (8.68 ± 0.98 %), although the difference was not statistically significant. Plaque index, oral hygiene index, and papillary bleeding index were also higher in the poorly-controlled diabetic non-smokers compared to the smokers group (1.59 ± 0.71 vs 1.43 ± 0.77 for PI, 3.44 ± 1.18 vs 3.39 ± 1.65 for OHI, and 1.32 ± 1.08 vs 1.30 ± 1.04 for PBI). On the other hand, calculus index, pocket depth, gingival recession, and clinical attachment level was higher in the smokers group (1.97 ± 1.00 vs 1.84 ± 0.81 for CI, 4.00 ± 3.22 mm vs 3.94 ± 2.05 mm for PD, 5.50 ± 3.34 mm vs 3.85 ± 3.03 mm for GR, and 9.50 ± 4.34 mm vs 7.79 ± 3.10 mm for CAL). Similar to the result in table 4, there was no significant differences in any clinical parameters between poorly controlled diabetic smokers and non-smokers.

Discussion

This study reports the comparison of periodontal status of diabetic smokers, diabetic non-smokers, and systemically healthy patients. Table 1 shows that diabetic smokers had the greatest percentage of subjects with proper oral hygiene maintenance, which consists of toothbrush frequency 2x/day (85%), the use of dental floss (15%), and the use of mouthwash (10%). Nevertheless, table 2 and 3 show that there were no significant differences in plaque index and oral hygiene index among the three groups. This contradicts some literatures which suggest that plaque index and oral hygiene index of smokers or diabetic subjects are poorer compared to control group. [31,32] A possible explanation for this is the fact that better oral hygiene maintenance in diabetic smokers counteract the influence of poor glycemic control or smoking on plaque build-up and oral hygiene index. This is supported by Csikar, et al. who

observed that smokers perceived themselves as having poorer oral health status compared to non-smokers and tried to compensate that by performing better oral hygiene practice.[33] This is also supported by Abdullah, et al. who stated that diabetic patients perceived their condition to be more serious than that of periodontitis.[34] We assume that diabetic patients with periodontitis would probably adhere more strictly to DM treatment, including oral hygiene maintenance, than that of periodontitis alone.

This study also reports that there were significant differences in calculus index among the study groups, particularly between the diabetic groups, both smokers and non-smokers, and non-diabetic non-smokers (P<0.001). Similar result was obtained in a study by Obradović, et al. who concluded that supragingival and subgingival calculus index in type II DM subjects (smokers and non-smokers) were higher than healthy, non-smoking subjects.[20] They also stated that type I DM subjects had even higher calculus index than the rest of the groups.[20] This result is also supported by Rani and Anandan who observed that increased serum Alkaline Phosphatase and calcium level were found in patients with type II DM compared to control.[35] Increased in ALP enzyme activity causes the release of phosphate ions, leading to more calculus formation and exacerbate the condition of periodontitis in type II DM.[35]

In terms of papillary bleeding index, this study finds that PBI of diabetic groups are significantly lower compared to non-diabetic non-smokers group. This contradicts result by Sayeeganesh et al. who state that when blood glucose level increases, gingival bleeding increases as well.[36] In diabetic patients, there are some vascular changes found in the gingiva. An accumulation of advanced glycated end products (AGEs) in the basement membrane of endothelial cells results in thicker membrane and increased production of Vascular Endothelial Growth Factor (VEGF). VEGF leads to increased angiogenesis and gingival bleeding in diabetic patients.[37] Nevertheless, in our study, we deduce that basement membrane thickening occurs in a greater degree compared to angiogenesis by VEGF, resulting in less tendency to bleed for diabetic groups. In addition, the lowest PBI level occurs in diabetic smokers. This is in agreement with earlier study by Sreedevi et al. who investigated the effect of smoking on periodontal parameters of smokers and non-smokers. [38] The study observes that both groups showed similar plaque levels, but smokers had lowered gingival and bleeding index, and increased calculus index.[38] Smoking is known to cause vasoconstriction of gingival vessels, which in turn represses vascular properties of inflammation such as bleeding, hyperaemia, and exudation.[38]

Comparison of pocket depth, gingival recession, and clinical attachment loss among the three groups shows interesting results. Significant differences are only found when comparing diabetic groups (both smokers and non-smokers) and control. This is similar to the study by Orbak, et al. and Haseeb, et al.[39,40]. The greatest gingival recession and clinical attachment loss are found in diabetic smokers, followed by diabetic non-smokers, and lastly by control group. It can be inferred that periodontal inflammation gets worse with smoking and poorer glycemic control. Putri, et al. investigated the difference in pocket depth and gingival recession between smokers and non-smokers with chronic periodontitis in Indonesian population. Their study showed similar result to our study, in which significant differences of both variables were found between smokers and non-smokers.[2] However, an interesting difference exist between the two studies. Their study showed that pocket depth of smokers was greater than non-smokers, while our study showed the opposite. The difference in result could be caused by the difference in research methodology, in which their data was obtained from medical record with different

operators taking the data.

Several mechanisms have been linked to increased susceptibility of diabetic patients with periodontal breakdown, namely changes in microflora, defective polymorphonuclear leukocyte (PMN), and altered collagen metabolism. Individuals with diabetes have higher glucose content in the gingival fluid and blood compared to those without diabetes with similar plaque and gingival index scores.[41] These changes induce qualitative changes in bacteria that contribute to the severity of periodontal disease, especially in poorly-controlled diabetes.[41] Nevertheless, to date there is insufficient evidence to point out any specific altered microflora and the exact roles of those microorganisms to the progression of periodontitis.[41] Deficiencies of PMN have also been linked to increased susceptibility to infection in patients with diabetes.[41] Impaired chemotaxis, defective phagocytosis, or impaired adherence result in diminished defence against periodontal pathogens, leading to bacterial proliferation.[41]

In diabetic patients, hyperglycaemic state leads to nonenzymatic glycosylation of numerous protein and matrix molecules, resulting in an accumulation of advanced glycation end products (AGEs).[41] This AGEs formation also occurs in normal glucose level. However, in hyperglycaemic environment, too much AGEs is formed.[41] Cross-linking of AGE and collagen makes the collagen less soluble and less likely to be repaired or replaced.[41] Tissue integrity is weakened due to damaged collagen that remains in the tissue for longer period. In short, collagen in the tissue of poorly-controlled diabetic patients is older and more prone to destruction by periodontal infection.[41]

An overwhelming body of evidence supports smoking as a major risk factor for increased prevalence and severity of periodontal destruction. Destruction of periodontal tissue may be caused by changes in microbiology, host response to bacterial challenge, or a combination of both.[42] Jiang et al. did a study about the impact of smoking on subgingival microflora. They found out that smoking promotes pathogenesis of subgingival biofilm by improving biofilm formation and virulence as well as creating favorable environment for periodontal pathogens.[42] Smoking also compromises various aspects of the innate and adaptive host immune response.[43] Chemotaxis and phagocytosis abilities of neutrophils in the periodontium are weakened, resulting in impaired clearance and enhanced colonization of periodontal bacteria.[44] In addition, smoking reduces the level of IgG antibody and increases production of reactive oxygen species in smokers, which subsequently leads to the decline of innate immune response to periodontal pathogens.[45,46]

The result in table 3 shows that there were no differences in the periodontal status (pocket depth, gingival recession, and clinical attachment loss) between diabetic smokers and non-smokers. This is in sync with the result of Javed, et al.[22] The authors then try to investigate whether smoking has any influence on periodontal status of diabetic subjects whose glycemic status are comparable. This is done by regrouping diabetic subjects into four categories, well-controlled diabetic smokers and non-smokers, as well as poorly-controllled diabetic smokers and non-smokers. The results also show that there were no significant differences in periodontal status between well-controlled diabetic smokers and non-smokers (table 4), and poorly controlled diabetic smokers and non-smokers (table 5). Nevertheless, GR and CAL were still higher in smokers group compared to non-smokers group, but not statistically significant. Thus, the authors still reach to the same conclusion that smoking does not have a significant impact on the periodontal health of well-controlled and poorly controlled diabetic patients, especially in patients with more severe stage of periodontitis. In this study, >80% of the

subjects in each group were at stage III in periodontitis severity.

Most studies report that periodontal condition is more severe in diabetic smokers compared to diabetic non-smokers, which is somewhat similar to this study, although not statistically significant.[20,21] The authors hypotesise that the reason for this difference could be due to the fact that glycemic control, which is measured by HbA1C, is better in diabetic smokers (7.63 ± 1.54) compared to diabetic non-smokers (7.93 ± 2.16). It is likely that periodontal destruction caused by AGE formation in smokers and non-smokers with DM were comparable, hence the smilar periodontal inflammatory parameters among the two groups.

It is well-known that several factors influence the etiopathology of chronic periodontitis.[7] Nevertheless, the composition of the oral microflora, which is measured by plaque accumulation, is considered as the main factor leading to periodontal destruction.[7] Other factors such as systemic condition, genetics, and environmental factors do not cause periodontitis, but may predispose, accelerate, or increase the progression of the disease. [47] This theory supports the results in our studies which reported that there were no significant differences in periodontal status between well-controlled diabetic smokers and non-smokers, and poorly controlled diabetic smokers and non-smokers. In all those groups, it is likely that microbiological factors, measured by plaque index, calculus index, and oral hygiene index, are comparable, leading to comparable periodontal status of the subjects. As such, the level of glycemic control or smoking factors will not cause significant alteration on the periodontal breakdown of the subjects.

As periodontal disease has multi-factorial etiologies, the treatment itself must address each of the factors.[7] In periodontal patients with predisposing factors like diabetes and smoking, both medical and dental professionals must work to together to create a systematic medical-dental coordinated care (M-DCC), as described in a pilot project by Yaacob, et al.[48] In their study, the physicians provide standard diabetic care, counsel risk factors such as smoking, exercise, and oral hygiene, motivate patients to complete periodontal therapy, and share HbA1c results with periodontists.[48] Likewise, periodontists provide periodontal treatment, counsel for risk factors, motivate patients to undergo DM monitoring and treatment, and share periodontal status of the patients to the physicians.[48] Yaacob et al. stated that Type-2 DM patients with chronic periodontitis who underwent non-surgical periodontal therapy under M-DCC showed significant improvement in periodontal parameters after 6 months.[48]

A limitation of the present study is that there are no data regarding the duration of diabetes. Longer duration of diabetes has been linked to greater prevalence and severity of periodontal disease.[49] Furthermore, the present study does not categorise cigarette smoking into conventional cigarette and electronic cigarette. Different types of cigarettes may have different effects on periodontal tissue. Lastly, data regarding local factors that can aggravate periodontal diseases in the area of periodontal measurement (malocclusion, overhanging restoration, trauma from occlusion, or bruxism) are not included.[7] Those confounding factors should be taken into account when conducting further studies. As the sample size of this study is also limited, furhter study involving larger sample size with biomolecular parameters should be done to further clarify the relationship between type-2 DM, smoking, and periodontal disease.

Conclusions

There were significant differences in calculus index, papillary bleeding index, periodontal pocket depth, gingival recession, and clinical attachment level between diabetic groups, both smokers and non-smokers, and

non-diabetic non-smokers. Meanwhile, there were no significant differences in any parameters between well-controlled diabetic smokers and non-smokers, as well as between poorly controlled diabetic smokers and non-smokers. Poor glycemic level can aggravate periodontal breakdown in chronic periodontitis patients. Nevertheless, smoking seems to have little effect on the periodontal destruction in diabetic patients with chronic periodontitis.

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Declaration of Interest

The authors report no conflict of interest.

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