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ORIGINAL ARTICLE

Periodontal status in Taiwanese pregnant women

Chuan-Chen Ho [a,b*], Ming-Yung Chou [a,b]

[a] School of Dentistry, College of Oral Medicine, Chung Shan Medical University, Taichung, Taiwan, ROC
[b] Department of Dentistry, Chung Shan Medical University Hospital, Taichung, Taiwan, ROC

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Abstract *Background/purpose:* Few studies have investigated the periodontal status of Taiwanese pregnant women. This study aimed to investigate the periodontal status of pregnant women and to examine its relation to oral hygiene.
Material and methods: This study randomly recruited 477 pregnant women. Among them, 203 women were in their first trimester. Forty-six women completed the study to the end of their third trimester. We also recruited 160 nonpregnant women as the control group. Clinical periodontal parameters were recorded and included probing pocket depth [PPD (mm)], clinical attachment level [CAL (mm)], gingival index simplified [GI-s (%)], and plaque index [PI (%)].
Results: The GI-s of the pregnant group (PG) was higher than that of the control group [CG; (i.e., nonpregnant)], but only the third trimester was statistically significantly different ($P < 0.001$).The full mouth dental PI was higher in the PG than in the CG ($P < 0.001$), particularly in the interproximal areas. The mean PPD was greater in the PG than in the CG ($P < 0.001$) in all tooth areas. The mean CAL was higher in the PG than in the CG ($P < 0.001$), but no difference existed between the different trimesters. The CG had a higher percentage of sites with a shallow PPD, compared to the PG ($P < 0.001$); the PG had a higher percentage of sites with a PPD of 4–6 mm, compared to the CG ($P < 0.001$). Only the PI of the full mouth and lingual tooth surfaces in the third trimester were better than in the first trimester throughout the pregnancy.
Conclusion: Gingival inflammation in pregnant women is positively correlated with the increased deposition of a dental plaque biofilm.

* Corresponding author. School of Dentistry, Chung Shan Medical University, 110, Section 1, Jianguo North Road, Taichung, Taiwan, ROC.
E-mail address: chcho@csmu.edu.tw (C.-C. Ho).

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Introduction

Periodontal diseases such as gingivitis and periodontitis are initiated and perpetuated by microbial infection.[1] The results of such infection could lead to gingival redness and bleeding, and develop to periodontitis with loss of clinical attachment, alveolar bone resorption, tooth mobility, and tooth loss. In Taiwan, a national periodontal survey, which covered the years of 2007 and 2008, was administered to adults aged 18 years or older.[2] Ninety-nine percent of adults had periodontal disease to some extent with 54% of them having periodontal pockets. The link between pregnancy and periodontal inflammation has been known for many years. Pregnancy gingivitis is extremely common and occurs in 30–100% of all pregnant women.[3–6] Current research implies that periodontal disease may alter the systemic health of a patient and adversely affect the well-being of the fetus by elevating the risk of low-birth-weight preterm delivery.[7–11]

A dental plaque biofilm is necessary for initiating periodontal inflammation,[12,13] and other factors (including systemic and local factors) can affect the occurrence and severity of periodontal diseases. Pregnant women are prone to gingival redness, swelling and bleeding, increased probing pocket depth, and tooth mobility in the 2nd–8th month of pregnancy,[14,15] but with no statistically significant attachment loss.[15] It has been postulated that the association of gingivitis with pregnancy is because increased plasma levels of progesterone and estrogen,[16,17] which aggravate pre-existing gingivitis,[13,16–18] and inadequate oral hygiene lead to the persistent accumulation of a dental plaque biofilm.[19,20]

The results of cross-sectional and longitudinal epidemiological studies indicate that the severity of gingival inflammation is positively correlated with the amount of a dental plaque biofilm.[21–23] Experimental gingivitis in a study conducted by Loe et al.[21] in 1965 directly proved that the deposition of a dental plaque biofilm could lead to the development of gingivitis. Furthermore, certain microbes in dental plaque can induce periodontal disease in animals.[24,25] In contrast to nonpregnant women, Kornman and Loesche[17] found that the proportions of anaerobes and aerobes are increased in the second trimester, particularly in *Prevotella intermedius* (*P. intermedius*), until the third trimester, at which point the *P. intermedius* level is decreased. These microbial changes may be associated with the plasma levels of estrogen and progesterone. The purpose of this study was to investigate the oral hygiene status (using the PI) in relation to gingival inflammation in Taiwanese pregnant women.

Material and methods

This study randomly recruited 477 pregnant women [i.e., pregnant group (PG)] from the Obstetrics Department of Chung Shan Medical University Hospital (Taichung, Taiwan) between January 2010 and July 2011. Among these individuals, 145 women were in the third trimester of pregnancy, 129 women were in the second trimester of pregnancy, and 203 women were in the first trimester of pregnancy. These 203 women were followed up during pregnancy. We lost 157 patients because they did not complete their follow up on account of moving to other cities and changing doctors, or because they did not want to continue follow up. Forty-six women completed the study to the end of their third trimester. The inclusion criteria for the PG were: they had to have no systemic disease, have a minimum of 20 remaining teeth (not including the 3rd molars), and had no antibiotic medications or periodontal therapy (including ultrasonic scaling) 6 months before the study. The control group (CG, $n = 160$) had the same inclusion criteria, except they were not pregnant (Tables 1 and 2).

The periodontal parameters included the probing pocket depth (PPD) (measured in mm) from the gingival margin to bottom of the pocket,[26,27] the CAL (in mm) from the cementoenamel junction (CEJ) to the bottom of the pocket,[28,29] the gingival index simplified (GI-s; measured by %) to indicate gingivitis,[30] and the PI (measured by %).[31,32] Only one periodontal specialist performed all examinations.

Statistical analysis

This study compared the periodontal condition of the pregnant women in the three trimesters versus the nonpregnant controls. We used one-way analysis of variance to evaluate the significance of the difference between the means of the three trimesters. Scheffe's multiple comparison testing was used to determine the significance of the difference between the three pregnancy trimesters. The Student *t* test was used to evaluate the periodontal condition of the longitudinal follow up of the pregnant women and to evaluate between the first trimester and third trimester. A value of $P < 0.05$ was considered statistically significant.

Results

The clinical periodontal parameters between the PG and the CG

The GI-s, PI, PPD, and clinical attachment level (CAL) were significantly different between the PG and the CG ($P < 0.001$); however, these parameters were not significantly different between the trimesters (Tables 3 and 4). The full mouth GI-s was only significantly different at the third trimester ($P < 0.001$). In addition, the GI-s was not

Table 1 Distribution of the study participants.

	Patients	
	No.	%
Control	160	25
1st Trimester[a]	203	32
2nd Trimester	129	20
3rd Trimester	145	23
Total	637	100

[a] Forty-six of the study participants in the first trimester were followed up at the third trimester.

Table 2 Age distribution of the study participants.

Age (y)	1st Trimester		2nd Trimester		3rd Trimester		Control	
	No.	%	No.	%	No.	%	No.	%
<25	29	14.4	17	13.2	17	11.7	139	86.8
25–29	101	50.0	64	49.6	62	42.8	17	10.6
30–34	55	27.2	34	26.3	49	33.8	4	2.6
>34	17	8.4	14	10.9	17	11.7	0	0.0
Total	202	100.0	129	100.0	145	100.0	160	100.0

Table 3 Comparisons of the different groups.

	Difference between the means					
Parameters	CG vs. I	CG vs. II	CG vs. III	I vs. II	I vs. III	II vs. III
GI-s (%)	2.66	2.45	6.02*	−0.21	3.36	3.57
PI (%)	11.58*	9.50*	12.27*	−2.08	0.69	2.77
PPD (mm)	0.18*	0.22*	0.19*	0.04	0.01	−0.03
CAL (mm)	0.13*	0.17*	0.16*	0.04	0.03	−0.01

* Indicates a value of $P < 0.001$, for Scheffe's test.
CAL = clinical attachment level; CG = control group; GI-s = gingival index simplified; PI = plaque index; I = 1st trimester; II = 2nd trimester; III = 3rd trimester; PPD = probing pocket depth.

Table 4 Periodontal status between the control group and the pregnant group.

Parameters	CG	PG (trimester)		
		1st	2nd	3rd
		Mean (SD)		
GI-s (%)*	60 (16)	63 (16)	63 (16)	66 (17)
PI (%)***	73 (16)	84 (9)	82 (11)	85 (10)
PPD (mm)***	2.20 (0.22)	2.38 (0.28)	2.43 (0.28)	2.40 (0.31)
CAL (mm)***	1.86 (0.15)	1.99 (0.23)	2.03 (0.21)	2.02 (0.25)

* $P < 0.05$ and *** $P < 0.001$, for the ANOVA test.
ANOVA = analysis of variance; CAL = clinical attachment loss; CG = control group; GI-s = gingival index simplified; PI = plaque index; PG = pregnant group; PPD = probing pocket depth; SD = standard deviation.

significantly different between the different tooth sites (i.e., buccal, lingual, and interproximal; Tables 5 and 6).

The PI was significantly different between the PG and the CG ($P < 0.001$). However, there was no difference in this parameter between the different trimesters. The PI was only significantly different at the interproximal sites between the PG and the CG ($P < 0.001$). The PI of different sites was not different between the different trimesters (Tables 7 and 8).

The mean PPD of the PG and the CG was statistically significantly different ($P < 0.001$), but not significantly different between the different trimesters (Table 9). The means of the PPD were significantly different at the

Table 5 The gingival index simplified between the control group and the pregnant group.

Tooth area (site)	CG	Trimester		
		1st	2nd	3rd
		Mean (SD)		
Total*	60 (16)	63 (16)	63 (16)	66 (17)
Buccal	9 (9)	8 (5)	8 (6)	8 (4)
Lingual	12 (11)	11 (3)	11 (3)	11 (5)
Interproximal	44 (12)	45 (11)	45 (12)	47 (11)

* $P < 0.05$, for the ANOVA test.
ANOVA = analysis of variance; CG = control group; SD = standard deviation.

Table 6 Gingival index simplified at different tooth areas.

	Difference between the means					
Tooth area	CG vs. I	CG vs. II	CG vs. III	I vs. II	I vs. III	II vs. III
Total	2.66	2.45	6.02*	−0.21	3.36	3.57
Buccal	−0.41	−0.83	−0.18	−0.43	0.22	0.65
Lingual	−0.59	−0.55	−0.35	0.05	0.24	0.19
Interproximal	0.98	0.76	3.19	−0.22	2.22	2.43

* $P < 0.001$, for Scheffe's test.
CG = control group; I = 1st trimester; II = 2nd trimester; III = 3rd trimester.

different tooth areas (i.e., sites), but was not significantly different between the different trimesters (Table 10). In terms of the distributions of different PPDs, we found the following: (1) a significantly smaller percentage of PPD of 1–3 mm among the PG than among the CG; (2) a significantly larger percentage of PPD of 4–6 mm among the PG than among the CG; and (3) CG and PG both very rarely had areas with a PPD ≥ 7 mm (Table 11).

The mean CALs were significantly different between the CG and the PG ($P < 0.001$). No significant difference existed between the different trimesters (Table 3).

Longitudinal follow up of the periodontal parameters from the first to the third trimesters

The comparisons of the GI-s, PI, PPD, and CAL revealed that only the mean PI values were significantly different between the first trimester and the third trimester ($P > 0.05$; Tables 12 and 13).

Discussion

The link between pregnancy and periodontal inflammation has been known for many years. Pregnancy gingivitis is extremely common, and occurs in 30–100% of all pregnant women.[3–6] Current research implies that periodontal disease alters the systemic health of the patient and adversely affects the well-being of the fetus by elevating the risk of low-birth-weight preterm delivery.[7–11] Pregnancy gingivitis usually occurs from the 2nd month to the 8th month of

Table 7 Percentage of tooth surfaces with plaque [based on the plaque index (%)] between the control group and the pregnant group.

Tooth area	CG	PG (trimester)		
		1st	2nd	3rd
		Mean (SD)		
Total*	73 (16)	84 (9)	82 (11)	85 (10)
Buccal	7 (9)	8 (6)	9 (7)	9 (4)
Lingual	13 (14)	14 (5)	13 (3)	13 (3)
Interproximal*	57 (13)	62 (6)	61 (7)	64 (6)

* $P < 0.001$, for the ANOVA test.
ANOVA = analysis of variance; CG = control group; I = 1st trimester; II = 2nd trimester; III = 3rd trimester; PG = pregnant group; SD = standard deviation.

Table 8 The plaque index at different tooth areas.

Tooth area	Difference between the means					
	CG vs. I	CG vs. II	CG vs. III	I vs. II	I vs. III	II vs. III
Total	11.58*	9.50*	12.27*	−2.08	0.69	2.77
Buccal	1.11	1.32	1.57	0.21	0.45	0.24
Lingual	1.50	0.50	0.30	−0.99	−1.20	−0.21
Interproximal	5.25*	4.28*	6.39*	−1.00	1.14	2.11

* $P < 0.001$, for Scheffe's test.
ANOVA = analysis of variance; CG = control group; I = 1st trimester; II = 2nd trimester; III = 3rd trimester.

Table 9 Mean pocket depth between the control group and the pregnant group.

Tooth area	CG	PG (trimester)		
		1st	2nd	3rd
		Mean (SD)		
Total*	2.20 (0.22)	2.38 (0.28)	2.43 (0.28)	2.40 (0.31)
Buccal*	1.92 (0.22)	2.06 (0.28)	2.12 (0.24)	2.08 (0.28)
Lingual*	2.04 (0.23)	2.16 (0.28)	2.16 (0.29)	2.16 (0.29)
Interproximal*	2.31 (0.27)	2.51 (0.34)	2.53 (0.35)	2.53 (0.34)

* $P < 0.001$, for the ANOVA test.
ANOVA = analysis of variance; CG = control group; I = 1st trimester; II = 2nd trimester; III = 3rd trimester; PG = pregnant group; SD = standard deviation.

Table 10 The mean probing pocket depth of the control group and the pregnant group.

Tooth area	Difference between the means					
	CG vs. I	CG vs. II	CG vs. III	I vs. II	I vs. III	II vs. III
Total	0.18*	0.22*	0.19*	0.04	0.01	−0.03
Buccal	0.14*	0.19*	0.15*	0.06	0.02	−0.04
Lingual	0.12*	0.12*	0.12*	0.00	0.00	0.01
Interproximal	0.20*	0.23*	0.22*	0.03	0.02	0.00

* $P < 0.001$, for Scheffe's test.
ANOVA = analysis of variance; CG = control group; I = 1st trimester; II = 2nd trimester; III = 3rd trimester; PG = pregnant group.

Table 11 The distribution of the probing pocket depth in the control group and the pregnant group.

PPD (mm)	CG	PG (trimester)		
		1st	2nd	3rd
		Mean %, (SD)		
1 − 3 mm*	97 (3)	95 (5)	95 (6)	94 (7)
4 − 6 mm*	2 (3)	5 (5)	5 (6)	5 (7)
≥ 7 mm	1 (1)	0 (0)	0 (0)	1 (8)

* $P < 0.001$, for the analysis of variance test.
CG, control group; PG, pregnant group; PPD, probing pocket depth; SD, standard deviation

Table 12 Periodontal changes between the first and third trimesters.

Parameter	Trimester		*t* value	P
	1st	3rd		
	Mean (SD)	Mean (SD)		
GI-s (%)	62 (18)	67 (14)	1.596	0.114
PI (%)	85 (7)	79 (14)	−2.317	0.023*
PPD (mm)	2.38 (0.28)	2.34 (0.28)	0.661	0.510
CAL (mm)	1.95 (0.25)	2.03 (0.24)	1.395	0.166

* $P < 0.05$, for the Student *t* test.
CAL = clinical attachment loss; CG = control group; GI-s = gingival index simplified; PI = plaque index; PG = pregnant group; PPD = probing pocket depth; SD = standard deviation.

pregnancy and with easy bleeding, redness, edema, and increased PPD.[1] The anterior region of the mouth is affected more often, and the interproximal sites tend to be most involved. Increased tissue edema may lead to increased pocket depth. The present study found increased gingivitis (GI-s, ≥ 63%) during pregnancy. Further analysis indicated that the GI-s was significantly different between the CG and the second trimester. The mean of the total GI-s and the GI-s at different tooth areas (i.e., sites) were higher in the PG than in the CG, but was not different between the different trimesters. These findings were in agreement with previous reports[20,21] and with the report of Niederman[33] who stated that gingival inflammation is significantly increased throughout pregnancy (e.g., the GI is significantly higher in the second or third trimester of pregnancy than in the first trimester, and the mean GI value is lower in nonpregnant women than in women in their second or third trimester of pregnancy).

The study by Loe et al[21] in 1965 confirmed that dental plaque could induce gingivitis. In the present study, the

Table 13 Percentage of tooth surfaces with plaque (based on the plaque index) between 1st and 3rd trimester.

Tooth area	Trimester		*t*	P
	1st	3rd		
	Mean (SD)	Mean (SD)		
Total	85 (7)	79 (14)	−2.317	0.023*
Buccal	10 (9)	8 (10)	−0.972	0.334
Lingual	15 (8)	12 (3)	−2.259	0.026*
Interproximal	62 (7)	62 (6)	0.017	0.987

* $P < 0.05$, for the Student *t* test.
SD = standard deviation.

mean total PI was high in the different trimesters, particularly in the interproximal areas, which indicated that pregnancy gingivitis was positively correlated with the dental PI. This finding is consistent with the study of Loe et al.[21] Therefore, promoting plaque control could reduce dental plaque and gingivitis during pregnancy.

The current study found that pregnancy could result in increases in the PPD (for PG vs. CG, $P < 0.001$) and in the different trimesters. The mean CALs were different between the PG and the CG, but not different between the trimesters, which suggests that pregnancy does not lead to further attachment loss. The increases in PPD during pregnancy with the clinical manifestations of gingival redness and swelling were because of pseudopocket formation.

We noticed that 86.8% of our CG were younger than 25 years. The prevalence of periodontal disease increases with age, although it is unlikely that becoming older in itself greatly increases susceptibility to periodontal disease. It is more likely that the cumulative effects of disease over a lifetime (e.g., deposits of plaque and calculus[34,35]) revealed that the PG harbored more plaque than the CG (Tables 7 and 8). Burt[36] concluded that some loss of periodontal attachment and alveolar bone is to be expected in older persons, but age alone in a healthy adult does not lead to a critical loss of periodontal support.

Establishing a healthy oral environment and maintaining optimal oral hygiene levels are the primary objectives in pregnant women. The hormone response can not be changed; therefore, the first trimester is the period of organogenesis (i.e., the fetus is highly susceptible to environmental influence) and the last half of this trimester has a risk of premature delivery and discomfort for a woman. Thus, the second trimester is the safest period for providing routine dental care. In addition, drug therapy in pregnant patients is controversial because drugs can affect the fetus by diffusion across the placenta, especially during the first trimester. Therefore, the emphasis of dental care is on controlling active disease and eliminating potential problems that could arise in late pregnancy.

In conclusion, the results of our present study support that gingivitis in pregnant women could be associated with hormonal changes. However, inadequate oral hygiene (as indicated by a high PI) could be a contributing factor. The harboring of potential periodontal pathogens in the pockets of pregnant women could be a predisposition to the development of further periodontal breakdown in susceptible individuals. We recommend professional dental prophylactic scaling during the second trimester of pregnancy to reduce the risk of advanced periodontal destruction.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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