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Relationship between Preeclampsia and Periodontal Disease

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Abstract

Background and aims. Preeclampsia is one of the causes of mother and newborn mortality; however, the exact etiology has not been identified despite an extensive body of literature. This study was performed to assess whether there is a relationship between the preeclampsia and periodontal disease.

Materials and methods. Sixty pregnant women were allocated to case (with preeclampsia) and control (healthy) groups in this analytical study. Plaque index (PI), gingival index (GI), clinical probing depth (CPD), gingival recession (GR) and clinical attachment level (CAL) were measured in both groups. The evaluations began at delivery till 24 hours postpartum with the patient's informed consent. Data were analyzed using independent t-test for comparing mean values of groups with the Microsoft Excel software.

Results. There were no statistically significant differences in the studied parameters between groups (P>0.05). Gingival recession was seen in only one case.

Conclusion. Within the limits of this study, no relationship was found between preeclampsia and periodontal disease. More research with more sophisticated and precise methods to screen preeclamptic patients and monitor the preeclampsia is suggested.

Key words: **Periodontal disease, preeclampsia, pregnancy.**

Introduction

Preeclampsia manifests as high blood pressure and proteinuria after the twentieth week of pregnancy.[1] It is one of the causes of mother and child death.[1] This condition can progress into eclampsia, an acute and life-threatening complication characterized by the appearance of tonic-clonic seizures, and death of mother and child if no intervention is carried out. In spite of the extensive studies in the recent years, the exact pathophysiology of the disease has not been elucidated. However, the main suspected mechanisms are vascular contraction, hyperresponsiveness and

endothelial cell dysfunction.[2]

One of the proposed mechanisms for the endothelial dysfunction is the presence of a vascular inflammatory state with a production of TNF-α and interleukins in the pregnant mother that can participate in an oxidation process. Based on this hypothesis, free oxygen radicals may cause self-propagating lipid peroxidases and toxic radicals resulting in endothelial cell damage.[3] The damage will produce nitric oxide from endothelial cells that will imbalance the ratio of prostaglandins. On the other side, the microvascular system is provoked and the permeability of the capillaries is increased.[4] Also the production of lipid-rich macrophages will increase, which is characteristic of atherosis.[5] Acute atherosis of placenta in preeclampsia has pathological and clinical manifestations similar to atherosclerosis. Other features include localized endothelial damage and fibrinoid necrosis in the arterial wall along with mononuclear cell and lipid-rich macrophages in the perivascular space. There is evidence indicating that a relationship may exist between chronic infectious diseases such as periodontitis and atherosclerosis.[6] Moreover, there are similarities in the pathophysiology of atherosclerosis and preeclampsia, and even atherosclerotic plaques have been identified in patients with preeclampsia, that all suggest that the periodontal disease might be a risk factor for preeclampsia.[7]

Although some studies have confirmed this relationship, 4-8 there is controversy regarding the findings. The aim of the present study was to investigate the probable relationship between preeclampsia and maternal periodontal disease.

Materials and Methods

The case group consisted of thirty pregnant women with preeclampsia who had been referred to the Obstetrics and Gynecology Section, Al-Zahra Hospital, Tabriz, Iran. Thirty healthy pregnant patients in the same setting were randomly selected from a pool of healthy pregnant women and formed the control group. The inclusion criteria for the pregnant women were (1) first pregnancy, (2) age range of 20-30, (3) no systemic disease affecting the periodontal health, (4) no smoking, and (5) no indication for antibiotic prophylaxis.

The groups were matched for variables including age, socioeconomic status, gestational age, and body mass index (BMI). Preeclampsia was determined by presence of proteinuria, and high maternal blood pressure, that is, > 140 mm Hg for systolic pressure and > 90 mm Hg diastolic pressure in women who had normal blood pressure prior to the twentieth week of pregnancy. Proteinuria was defined as the depletion of more than 0.3 g of protein in a 24-hour gathered urine sample or two random urine specimens obtained at least 4 hours apart demonstrating ≥ 1+ by dipstick testing or measured as > 30 mg/dL protein. Cases of periodontitis were defined as the presence of 1 or more periodontal sites with both a loss of tissue attachment of 3 mm or more and a probing depth of 3 mm or more.

The evaluations were performed from partum till 24 hours postpartum, all after obtaining informed consents. Periodontal examinations were performed with a William's periodontal probe and a headlight in the Obstetrics and Gynecology Section. Plaque index (PI), gingival index (GI), clinical probing depth (CPD), gingival recession (GR) and clinical attachment level (CAL) were measured in 6 locations of Ramfjord teeth. PI and GI were examined according to Löe and Silness.[9,10] GR was measured from CEJ to the gingival margin. In patients with no recession and a gingival margin coronal to CEJ, the CAL was measured as distance between CEJ and bottom of the pocket. In cases with little recession and gingival margin along the CEJ, the CAL was the same as CPD. In cases with recession, the CAL was calculated by sum of GR and CPD. CPD was defined as the distance from the gingival margin to the bottom of the pocket. All numbers were rounded to the nearest millimeters. An average of all locations readings constituted each tooth's record and the average of the records of all Ramfjord teeth resulted in the patient's score for each parameter.

The data were analyzed using Microsoft Excel. Initially, *f*-test was utilized to examine the variance homogeneity. Subsequently, *t*-test was performed to compare the mean scores of case and control groups. A 5% significance level was used.

Results

The patients in the case and control group had an average age of 24.5 ± 2.6 and 21.5 ± 2.1 years, respectively.

The results of the periodontal assessments are shown in Table 1. The mean scores of PI, GI and CPD did not show any statistically significant differences between case and control groups. Although the mean CAL score in the case group was higher compared to the control group, the difference did not reach statistical significance (P = 0.057).

Table 1. The association between case and control groups with periodontal parameters

Parameter	Mean ± SE		P value
	Case	Control	
PI	1.09 ± 0.28	1.08 ± 0.29	0.918
GI	1.46 ± 0.33	1.40 ± 0.26	0.436
CPD	2.49 ± 0.44	2.36 ± 0.34	0.217
CAL	1.60 ± 0.54	1.37 ± 0.33	0.057

PI: Plaque index; GI: gingival index; CPD: clinical probing depth (mm); CAL: clinical attachment level (mm).

GR was observed only in one case and, therefore, no statistical analysis was performed for this parameter.

Discussion

The results of this case-control study did not demonstrate a relationship between preeclampsia and periodontal disease. In the present study, there were no statistically significant differences in GI and PI between groups ($P > 0.05$). Our finding regarding PI concurs with other studies.[2,8,11-13] Although the GI values obtained in this study are consistent with those of other studies, [2, 13] Canakci et al,[12] in contrast, found that the preeclamptic patients had higher percentage of areas with bleeding on probing (BOP) compared to the healthy peers. When interpreting BOP and GI findings, the mild increase in systemic inflammation during normal pregnancy should be considered, which is more prominent in the preeclamptic patients.[14] There are theories that blame the periodontal disease for both initiating and aggravating the preeclampsia;[15,16] however, the increase in BOP and GI parameters in the preeclapmtic patients might be due to the increased inflammation, and the periodontal inflammation per se may not be the etiological factor for preeclampsia. In fact, preeclampsia has been suspected for its possible co-induction of periodontal disease.[17]

There was no significant difference in CPD between groups ($P = 0.22$). Although our CPD results agree with Khader et al,[13] they contradict other studies.[2,12,18-20] One study demonstrated a significant difference in CPD between case and control groups.[19] In the latter study, mother's age, ethnicity, smoking during pregnancy, and age at pregnancy were evaluated and results were analyzed independent from these variables. Oettinger-Barak et al[2] also showed a significant difference in CPD between case and control groups.

In another study, the difference in CPD values was again significant.[20] These results were independent from the age, educational level, smoking during pregnancy, alcohol consumption, and high blood pressure. Cota et al[20] did, however, advocate caution in linking between preeclampsia and the periodontal disease as both have multi-factorial etiology and are influenced by many factors.

In the present study, CAL values were not statistically different between the studied groups. In a previous study, however, a significant difference in CAL was observed in preeclampsia.[2] The same study showed a correlation between serum cytokine level and preeclampsia pathogenesis. Other studies have also shown significant differences in CAL between case and control groups,[11,12,18,20] while another study failed to show such statistical significance in sites with CAL $\geq$ 3 mm, which is consistent with the present study.[13]

In this study, the parameters that were used to match the groups in previous studies have been employed. However, there is no study (including the present research) to consider all inclusion or exclusion criteria cited in the literature. For example, serum lipid profile was not adjusted in group matching in our study as was done by Canakci et al.[12] Other suspected factor is sample size. Boggess et al[19] and Canakci et al[12] used a sample size of 39 and 41 preeclamptic patients, respectively, and found a statistically significant difference in CAL between groups. On the other hand, Khader et al[13] included 115 preeclamptic patients in their study and did not find a statistically significant difference in CAL between the studied groups. The CAL finding along with the heterogeneity that has been seen in periodontopathogens identification might be due to the patient susceptibility to diseases and genetic issues similar to what happens in the periodontal conditions. In other words, either periodontal or uteroplacental reaction is prone to biological diversity among different individuals. CAL may be an endpoint parameter showing the history of periodontal destruction and not necessarily the present destructive profile. Finding marked differences in the periodontal inflammatory parameters as it was shown in some studies may not end in concluding that inflammation is enough for causing preeclampsia,[12] since it has been shown that preeclampsia might be the result of even subclinical inflammation.[21]

Further studies on the subject with bigger sample size and surveys on the microbial flora and cytokine analysis are suggested.

In conclusion, within the limits of this study, there were no relationship between preeclampsia and periodontal disease.

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