JNMS Journal of Nippon Medical School https://www.nms.ac.jp/sh/jnms/

Original

Periodontal Disease is a Risk Factor for Placenta-Mediated Pregnancy Complications

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Objective: Although periodontal disease is known to be associated with obstetric complications, evidence is limited regarding the relationship with placenta-mediated pregnancy complications (PMPCs), a major cause of maternal and fetal morbidity and mortality. This study investigated whether periodontal disease is a risk factor for PMPCs.

Methods: We conducted a retrospective case-control study of inpatients with singleton pregnancies who underwent perinatal dental examinations at Nippon Medical School Tama Nagayama Hospital between September 2012 and August 2015. Medical records were reviewed for incident PMPCs, including preterm birth, preeclampsia, small for gestational age birth, placental abruption, and late pregnancy loss. Associations of periodontal disease with PMPCs were assessed using univariate and multivariate logistic regression analyses.

Results: Among 262 inpatients, 130 (49.6%) developed PMPCs. The prevalence of periodontal disease was significantly higher among women with PMPCs than among those without PMPCs (90.0% vs. 75.0%, P = 0.002). Multivariate logistic regression analysis identified periodontal disease as an independent risk factor for PMPCs (adjusted odds ratio [aOR], 2.89; 95% confidence interval [CI], 1.41–5.88). Younger maternal age (<35 years; aOR, 1.75; 95% CI, 1.04–2.92) and a past history of PMPCs (aOR, 3.27; 95% CI, 1.47–7.27) were also significantly associated with PMPCs.

Conclusion: Periodontal disease was independently associated with increased risk of PMPCs. Management of periodontal disease during pregnancy may help prevent PMPCs.

(J Nippon Med Sch 2026; 93 (4): 304–310. https://doi.org/10.1272/jnms.JNMS.2026_93-408)

Keywords: placenta-mediated pregnancy complications, periodontal disease, pregnancy outcome

Introduction

Placenta-mediated pregnancy complications (PMPCs), such as small for gestational age (SGA) birth, preeclampsia, placental abruption, spontaneous preterm birth, and pregnancy loss, are leading causes of maternal and fetal/infant deaths[1,2].

PMPCs are associated with abnormal placentation during early pregnancy, and subsequent impaired placental blood flow results in various disorders. Each PMPC increases both the risk of recurrence of the same complication and the risk of other PMPCs in later pregnancies[3–6], which suggests that abnormal placentation tends to recur in subsequent pregnancies. Additionally, PMPCs predispose patients to cardiovascular disease[7,8] and are considered a major risk factor for cardiovascular disease in the American Heart Association/American Stroke Association guidelines. PMPCs can be inherited[9]. Although complications differ, similarities have been reported. Hence,

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https://doi.org/10.1272/jnms.JNMS.2026_93-408
Received: October 30, 2025; Accepted: April 27, 2026

preventive strategies should target PMPCs as a single condition.

Periodontal disease, including chronic periodontitis, has been implicated in a wide range of systemic conditions, including cardiovascular disease, stroke, diabetes, and pregnancy-related complications, largely through inflammatory pathways[10]. Approximately 40% of pregnant women have periodontal disease[11], and accumulating evidence suggests that maternal periodontal disease is associated with several adverse pregnancy outcomes, including low birth weight[12–14], preterm birth[15], and preeclampsia[16]. A systematic review in 2023 summarized reported associations between periodontal disease during pregnancy and unfavorable perinatal outcomes[17].

Two main biological mechanisms have been proposed to explain these associations. First, periodontal pathogens and components of the oral microbiome may translocate hematogenously to the feto-placental unit. Second, inflammatory mediators released from infected periodontal tissues may exert systemic effects on the feto-placental unit and myometrium, thereby contributing to adverse pregnancy outcomes[18].

Although associations between periodontal disease and individual obstetric complications such as preterm birth, low birth weight, and preeclampsia have been reported, these outcomes share common pathophysiological features related to abnormal placentation and vascular dysfunction. PMPCs represent a conceptual framework that integrates these disorders as manifestations of a shared underlying mechanism. Evaluating periodontal disease in relation to PMPCs as a unified disease entity may therefore clarify whether periodontal disease is a common risk factor affecting placental vascular function rather than influencing isolated clinical outcomes. However, no previous study has examined the association between periodontal disease and PMPCs as a composite entity. Therefore, we investigated the prevalence of periodontal disease in this inpatient population and evaluated whether it was associated with PMPCs conceptualized as a unified pathophysiological condition.

Methods

This study was approved by the Institutional Review Board of Nippon Medical School (approval number: 388) and conducted in accordance with the ethical review processes of that institution[19]. All inpatient data from September 2012 through August 2015 were obtained from the inpatient database at Nippon Medical School Tama Nagayama Hospital, a university-affiliated tertiary medical center. Each patient's relevant information was collected on a data sheet completed by a full-time technician at the end of hospitalization and saved in a computerized database. Data collected included demographic information, admission and discharge information, and clinical characteristics. Written informed consent was obtained from all participants prior to enrollment.

Using the inpatient database, we conducted a retrospective case-control study of the association between periodontal disease and PMPCs among inpatients with singleton pregnancies who delivered at ≥22 weeks of gestation in our medical center. During the study period, 533 pregnant women admitted to our hospital were initially identified. These admissions included hospitalizations for delivery and for obstetric indications such as threatened preterm labor and maternal or fetal complications. Our facility also receives referrals for higher-risk pregnancies, and the study population therefore included a relatively large proportion of high-risk cases. As shown in **Figure 1**, predefined eligibility criteria were used. These criteria included exclusion of women with multiple pregnancies, unknown gestational age, and those who did not undergo perinatal dental examinations. In addition, women who were hospitalized for suspected PMPCs (e.g., threatened preterm labor or suspected fetal growth restriction) but whose delivery outcomes did not meet the diagnostic criteria for PMPCs were also excluded. Such cases represent borderline clinical conditions rather than clearly uncomplicated pregnancies, and inclusion of such cases in the non-PMPC group could lead to misclassification of outcome status and obscure the contrast between groups. After applying these criteria, 262 women with singleton pregnancies were included in the final analysis. Participants were then classified according to the presence or absence of PMPCs. Information on maternal age, parity, gestational age at delivery, birth weight, maternal complications such as hypertension, diabetes, and thyroid dysfunction, periodontal disease, and medical history was extracted from the database. We also conducted exploratory analyses to examine the association of periodontal disease with each PMPC component. For these analyses, each component was evaluated separately in the full study population (n = 262). For each outcome, women who did not develop the respective PMPC component were used as the comparison group. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated for each component, and P-values were obtained using the chi-square test or Fisher's exact test, as appropriate.

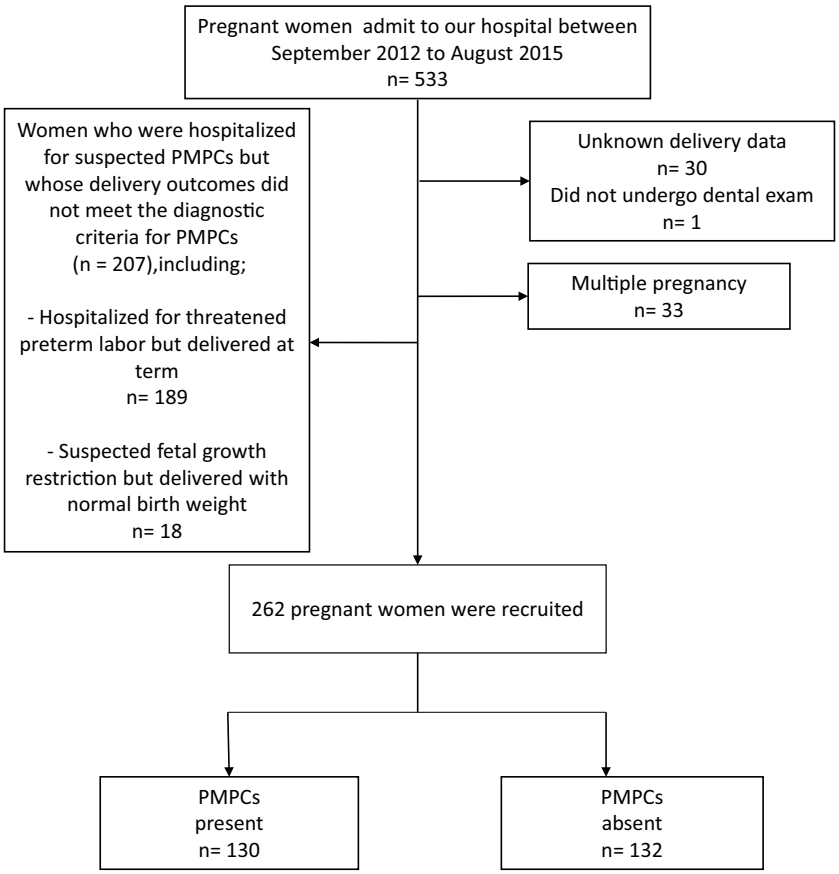

Figure 1 Flow diagram of the study population
Women with multiple pregnancies, unknown gestational age, those who did not undergo perinatal dental examinations, and those who did not meet the predefined eligibility criteria (including threatened preterm labor with term delivery and suspected fetal growth restriction with normal birth weight) were excluded.

PMPCs included spontaneous preterm birth, preeclampsia, SGA, placental abruption, and pregnancy loss. SGA was defined as birth weight and height both less than the 10th percentile when compared to standard values for birth size for the respective gestational age. The diagnosis of preeclampsia was based on the criteria of the American College Obstetricians and Gynecologists: a sustained systolic blood pressure of ≥140 mm Hg or a sustained diastolic blood pressure of ≥90 mm Hg on two separate readings and proteinuria of 2+ or more on dipstick or 24-h urine protein collection of ≥300 mg or a protein-to-creatinine ratio >0.3 after 20 weeks of gestation[20]. Placental abruption was diagnosed based on clinical features and identification of retroplacental clots on ultrasound.

Periodontal examinations were conducted during hospitalization before delivery by two dentists (AS and EU). The timing of the dental examination depended on gestational age at admission and was not standardized across participants. Clinical parameters, such as probing depth and presence/absence of bleeding on probing (BOP), were recorded. Probing depth was defined as the depth from the gingival margin to the location where the periodontal probe entered when the bottom of the periodontal pocket was pushed with a force equivalent to approximately 20–30 grams using a periodontal probe. During examination, any bleeding from the bottom of the periodontal pocket was considered as BOP. As periodontitis progresses, probing depth increases and BOP is more likely to occur[21]. Probing depths were recorded at three sites each on the labial and palatal or lingual sides with six representative teeth (16th, 12th, 24th, 36th, 32nd, and 44th teeth)[22]. Periodontal disease was defined as a probing depth of >3 mm and the presence of BOP at any of the six sites of each tooth.

Because this study was designed as a retrospective case-control study, ORs and 95% CIs were estimated using logistic regression analyses. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Continuous variables were presented

Table 1 Baseline characteristics of participants, by placenta-mediated pregnancy complications (PMPCs) status

	PMPC presents (n=130)	PMPCs absent (n=132)	P value
Maternal age	33.1±5.0	33.4±5.5	0.618
Age <35 years	82 (63.1%)	66 (50.0%)	0.033
Primiparous	73 (56.2%)	73 (55.3%)	0.890
Periodontal disease	117 (90.0%)	99 (75.0%)	0.002
History of PMPCs	26 (20.0%)	10 (7.6%)	0.004
Hypertension	2 (1.5%)	4 (3.0%)	0.684
Diabetes	1 (0.8%)	1 (0.8%)	1.000
Thyroid disease	2 (1.5%)	4 (3.0%)	0.684

Data are presented as mean ± SD or number (%), as appropriate.

Table 2 Exploratory analysis of associations of periodontal disease with individual components of placenta-mediated pregnancy complications (PMPCs)

		Periodontal disease				
	Total	Present	Absent	Crude odds ratio	95% Confidence interval	P value
No complications	132	99 (75.0%)	33 (25%)			
PMPC	130	117 (90%)	13 (10%)	3.000	1.497–6.013	<0.05
Spontaneous preterm birth	69	63 (91.3%)	6 (8.7%)	3.500	1.387–8.831	<0.05
Preeclampsia	42	37 (88.1%)	5 (11.9%)	2.467	0.895–6.797	0.074
Small for gestational age	57	50 (87.7%)	7 (12.3%)	2.381	0.984–5.761	0.766
Placental abruption	11	9 (81.8%)	2 (18.2%)	1.500	0.308–7.297	0.888
Pregnancy loss	2	2 (100.0%)	0 (0.0%)	0.667	0.059–7.592	0.567

"No complications" indicates women without PMPCs.

as means and SDs or medians and interquartile ranges and were compared using Student's t test or the Mann–Whitney U test.

For univariate and multivariate analyses, maternal age was dichotomized as <35 and ≥35 years, and parity was dichotomized as primiparous and multiparous. The magnitude of associations of potential risk factors with PMPCs was quantified by the ORs with 95% CIs, which were calculated by logistic regression analysis.

Multivariate logistic regression analyses were used to identify significant independent risk factors associated with PMPCs. Independent variables included in the multivariate logistic model were maternal age, parity, periodontal disease, other complications, and history of PMPCs. For the final model, all independent variables were selected via a stepwise procedure. Two-tailed P-values <0.05 were considered to indicate statistical significance. All statistical analyses were performed using SPSS software version 23.0 (IBM Corporation, Armonk, NY, USA) and R version 3.5.2 (The R Foundation for Statistical Computing, Vienna, Austria).

Results

In total, data from 262 inpatients were analyzed (**Figure 1**). Participants were classified into two groups according to the presence or absence of PMPCs. The clinical characteristics of the study population are presented in **Table 1**.

The prevalence of periodontal disease in women with and without PMPC was 90.0% and 75.0%, respectively, and was significantly higher in those with PMPC (P = 0.002; **Table 1**). Exploratory analyses of the associations of periodontal disease with individual PMPC components are shown in **Table 2**. "No complication" refers to women without PMPCs. In these analyses, a significant association was observed for spontaneous preterm birth (present vs absent: 91.3% vs 8.7%, P < 0.05), but no significant associations were found for the other components.

Multivariate logistic regression analyses were performed to identify risk factors independently associated with PMPCs (**Table 3**). The model included periodontal disease, maternal age, parity, medical comorbidities, and history of PMPCs. Periodontal disease was identified as an independent risk factor for PMPCs (adjusted OR [aOR], 2.89; 95% CI, 1.41–5.88). Younger maternal age (<

Table 3 Multivariate logistic regression analysis of risk factors for placenta-mediated pregnancy complications (PMPCs)

Variable	Adjusted odds ratio	95% CI	P value
Maternal age <35 years	1.75	1.04–2.92	0.034
Periodontitis	2.89	1.41–5.88	0.004
History of PMPCs	3.27	1.47–7.27	0.004

The reference category comprised women aged ≥35 years with no periodontitis and no history of PMPCs.

35 years; aOR, 1.75; 95% CI, 1.04–2.92) and history of PMPCs (aOR, 3.27; 95% CI, 1.47–7.27) were also significantly associated with PMPCs.

Discussion

In this retrospective case-control study, periodontal disease was independently associated with PMPCs. Women with periodontal disease had a significantly higher risk of developing PMPCs, even after adjustment for maternal age, parity, medical comorbidities, and history of PMPCs. These findings suggest that periodontal disease should be regarded as a common risk factor underlying placenta-mediated disorders rather than as a condition associated with isolated obstetric outcomes. Pregnant women have a high prevalence of periodontal disease[23], perhaps because hormonal changes during pregnancy promote inflammatory responses that facilitate development of periodontal disease. Increased plasma levels of progesterone and estrogen during pregnancy can affect periodontal structures by interfering with the composition of subgingival microflora and the maternal immune system, as well as by inducing proinflammatory mediator production. Because of hormonal changes, 50%–70% of pregnant women develop periodontitis[24]. A Japanese study reported that the prevalence of periodontal disease in pregnant women was 50% in 2015[25]. However, because periodontal disease was defined as the presence of periodontal pockets measuring ≥4 mm in that report, prevalence was likely underestimated.

PMPCs is a general term for obstetric complications associated with placental hypoplasia and dysfunction. Inappropriate remodeling of maternal spiral arteries during early pregnancy leads to sustained elevation of vascular resistance and low uteroplacental circulation, resulting in placental ischemia, reperfusion injury, and oxidative stress. When present early in a pregnancy, these disorders disrupt the balance between angiogenic and antiangiogenic factors, leading to obstetric complications in late pregnancy[26].

PMPCs can recur and may share intergenerational characteristics. Previous large cohort studies reported increased recurrence rates of PMPCs across successive pregnancies[27]. Intergenerational associations of PMPCs were reported in population-based studies[9], and PMPCs may increase long-term cardiovascular risk in mothers and offspring[7,8].

Periodontal disease is associated with several obstetric complications. Chambrone et al.[28] reported an association between periodontitis and preterm birth and/or low birth weight. A systematic review and meta-analysis of case-control studies reported an association between periodontal disease and adverse pregnancy outcomes, including preterm birth and low birth weight[29]. A significant association between periodontal disease and preeclampsia was identified in a random-effects meta-analysis of 13 case-control studies involving 1,089 women with preeclampsia and two cohort studies (OR, 2.79; 95% CI, 2.01–3.01; $P < 0.0001$)[30]. Sgolastra et al.[31] reported a positive association between preeclampsia and periodontal disease in a meta-analysis of 15 studies, including three cohorts and 12 case-control studies (OR, 2.17; 95% CI, 1.38–3.41; $P = 0.0008$). Although associations of specific obstetric complications with periodontal disease have been established, no report has examined the relationship between such conditions when collectively classified as PMPCs and periodontal disease, as was done in this study. In the present exploratory analyses of individual PMPC components, a significant association was observed for spontaneous preterm birth; however, associations with other components were not significant. Because these analyses were exploratory and the number of cases was limited for several individual outcomes, the absence of statistical significance should be interpreted with caution.

Periodontal disease causes systemic inflammation and is known to be associated with cardiovascular disease. Cytokines produced during periodontal disease pathogenesis may cause vascular endothelial damage[32]. *Porphyromonas gingivalis*, a cause of periodontal disease, hindered remodeling of the uterine spiral artery in a rat

model[33] and degraded plasminogen activator inhibitor-1 (PAI-1) produced by vascular endothelial cells, slowing wound healing in vascular endothelial cells[34]. These findings suggest that periodontal disease is related to vascular endothelial disorders and remodeling failure. Periodontal disease can cause vascular endothelial dysfunction, which in turn causes PMPCs. PMPCs and periodontal disease share a common feature: vascular endothelial dysfunction.

This study had several limitations. First, it was a single-center, retrospective case-control study including only inpatients at a tertiary medical center, which may limit the generalizability of our findings. Second, periodontal status was assessed using partial-mouth recording, which may have led to misclassification and did not allow evaluation of periodontal disease severity or extent. Third, because the timing of periodontal examination relative to the onset of PMPCs could not be precisely determined, causal relationships cannot be inferred. Fourth, information on important potential confounders, such as smoking status, socioeconomic status, oral hygiene behaviors, and periodontal treatment during pregnancy, was unavailable or incomplete and could not be fully adjusted for in the analyses. Finally, although PMPCs were analyzed as a composite outcome, analyses of individual components were exploratory and may have been underpowered.

In conclusion, periodontal disease was independently associated with PMPCs in this study. These findings suggest that periodontal disease has a role in placental vascular dysfunction. Thus, management of periodontal disease during pregnancy might help prevent PMPCs.

Author Contributions: Akihito Nakai conceived and designed the study. Eriko Kikuchi and Asami Suzuki collected the data. Eriko Kikuchi and Masako Hayashi performed the statistical analyses. Eriko Kikuchi and Masako Hayashi interpreted the data. Eriko Kikuchi drafted the manuscript, and Atsuko Sekiguchi and Shunji Suzuki critically revised it for important intellectual content. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Acknowledgments: The authors thank E. Uchiyama for assistance with data collection.

Funding: No funding was received for this work.

Conflict of Interest: The authors declare no conflict of interest.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: The authors declare that no generative AI or AI-assisted technologies were used in the writing process of this manuscript.

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