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Original Article

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Association of Preeclampsia with P-wave and R-wave Peak Times of Electrocardiogram

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Abstract

Background: Preeclampsia is a major contributor to maternal and neonatal morbidity and is increasingly linked to long-term cardiovascular risks. This study investigated the association between preeclampsia severity and electrocardiographic markers of atrial and ventricular conduction, specifically P-wave dispersion (Pd), P-wave peak time (PWPT), R-wave peak time (RWPT), and QT dispersion (QTd).

Methods: In this prospective observational study, 90 pregnant women receiving care at Kowsar Obstetrics and Gynecology Hospital, Urmia, Iran, were evaluated, comprising 60 preeclamptic cases (stratified by clinical criteria into mild, n=30, and severe, n=30) and 30 age- and gestational age-matched normotensive pregnant controls. Preoperative 12-lead electrocardiograms (ECGs) were obtained prior to scheduled cesarean section. Continuous ECG parameters were compared across groups using ANOVA and Tukey's post-hoc tests, with statistical significance defined as $p< 0.05$.

Results: Significant electrophysiological variations were observed across groups. The mean P-wave peak time in lead II progressively prolonged with preeclampsia severity, measuring 41.15±4.65 *ms* in severe preeclampsia, 36.17±8.13 *ms* in mild preeclampsia, and 30.10±7.85 *ms* in controls (p=0.001). Similarly, significant progressive prolongations were noted for PWPT in lead V1, as well as RWPT in leads V5 (38.70±7.27 *ms vs*. 32.38±5.65 *ms vs*. 28.34±6.89 *ms*; p=0.001) and V6 (36.72±7.62 *ms vs*. 32.04±5.62 *ms vs*. 25.90±6.65 *ms*; p=0.001).

Conclusion: Preeclampsia is associated with distinct alterations in atrial depolarization and ventricular repolarization, as evidenced by the prolongation of surface ECG parameters such as Pd, RWPT, and QTd. Early identification of these non-invasive ECG abnormalities in preeclamptic patients provides an opportunity for early risk stratification and timely cardiovascular monitoring.

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Introduction

Preeclampsia is a complex, multifactorial pregnancy disorder characterized by the onset of hypertension and organ dysfunction after 20 weeks of gestation (1). This condition affects approximately 2–8% of pregnancies globally (2). Despite significant advancements in obstetric care, preeclampsia remains a leading cause of maternal and fetal complications (3, 4), contributing to over 50,000 maternal deaths annually worldwide (5). Maternal complications can include ec-

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lampsia, HELLP syndrome (hemolysis, elevated liver enzymes, low platelets), placental abruption and hemorrhage, pulmonary edema, and disseminated intravascular coagulation. Fetal complications may include preterm delivery, perinatal death, intrauterine growth restriction, and neurological injury caused by hypoxia (3, 4). Furthermore, recent studies have demonstrated that preeclampsia is associated with an increased long-term risk of major adverse cardiovascular and cerebrovascular events, such as myocardial infarction, chronic hypertension, venous thromboembolism, and stroke (6). The precise mechanisms linking preeclampsia to this heightened cardiovascular risk have yet to be fully elucidated.

Numerous studies have documented electrocardiographic alterations during normal gestation, including an elevated resting heart rate, a shortened PR interval, a prolonged corrected QT interval (QT_c), inverted or flattened T-waves, and a leftward shift of the QRS and T axes (7). Beyond these normative physiological adaptations, specific electrocardiographic parameters, such as P-wave dispersion (Pd), defined as the difference between maximum and minimum P-wave durations across a 12-lead electrocardiogram (ECG), serve as key indices of altered cardiac conduction (8). Consequently, Pd functions as a well-established marker for identifying heterogeneous intra- and inter-atrial conduction (9). Pd values are influenced by various clinical states, including coronary artery disease, essential hypertension, obesity, and valvular heart disease (10), and elevated Pd levels predict prolonged atrial conduction times and paroxysmal atrial fibrillation (11, 12).

Similarly, QT dispersion (QTd), measured as the inter-lead variability in the QT interval across surface ECG leads, serves as a direct, non-invasive index of spatial heterogeneity in ventricular repolarization (13). Alterations in ventricular repolarization parameters (QT and QT_c) have been demonstrated in preeclampsia (14), and QTd holds established prognostic utility for adverse cardiovascular events and mortality across several patient populations (15, 16). While recent investigations have utilized related parameters, such as P-wave peak time (PWPT) and R-wave peak time (RWPT), to examine localized atrial and ventricular conduction delays in cardiovascular conditions, their application alongside Pd and QTd in preeclampsia remains limited (17, 18). Given that the specific electrocardiographic manifestations of acute pressure overload in preeclampsia remain a subject of investigation, the purpose of the current study was to evaluate the relationship between Pd and QTd across varying levels of preeclampsia severity.

Methods

This prospective, observational study evaluated 90 pregnant women admitted to Kowsar Obstetrics and Gynecology Hospital, Urmia, Iran. Participants were recruited based on preeclampsia status and underwent preoperative ECG evaluation. They were classified as preeclamptic cases (n=60; subcategorized into mild, n=30, and severe, n=30) or age- and gestational age-matched normotensive pregnant controls (n=30). The study protocol received approval from the ethics committee of Urmia University of Medical Sciences (Number: IR.UMSU.REC.1399.132). All participants provided both verbal and written informed consent, and the study was conducted in compliance with the principles of the Declaration of Helsinki. To ensure comparability, individuals in all three groups were matched according to age and gestational age. A standard 12-lead ECG was performed on all participants as part of their preoperative management for cesarean section, using the Sina-100 electrocardiograph (Avecinna, Iran).

In healthy pregnant women, the indication for a cesarean section was based on standard obstetric guidelines. Participants were excluded from the study if they had specific cardiovascular, hepatic, or renal diseases; diabetes mellitus; acute or chronic inflammatory conditions; or thyroid disorders. Additionally, women taking medications known to affect ECG findings, such as beta-blockers, calcium channel blockers, or antiarrhythmic drugs, were also excluded.

The diagnostic criteria for preeclampsia were based on a new onset of hypertension and proteinuria after 20 weeks of gestation in a previously normotensive woman (19). Hypertension was defined as a systolic blood pressure of ≥140 *mmHg* or a diastolic blood pressure of ≥90 *mmHg*, measured on at least two occasions with a four-hour interval in a resting state. Proteinuria was defined as a 24-*hr* urine collection with a protein level of >300 *mg*, a random protein-to-creatinine ratio >0.3, or a urine dipstick reading of 1+ or greater (≥1+) on a standard random urine sample. The criteria for severe preeclampsia include a systolic blood pressure of ≥160 *mmHg* or a diastolic blood pressure of ≥110 *mmHg* on two separate measurements. Alternatively, severe preeclampsia

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can be diagnosed by the presence of one or more of the following clinical or laboratory signs. Clinical symptoms include headache, blurred vision, epigastric pain, and signs of pulmonary edema. Laboratory signs include a platelet count <100,000/ *µl*, elevated liver enzymes (AST and ALT >70 *IU/L*) or a doubling of baseline liver enzymes, serum creatinine >1.1 *mg/dL*, acute renal failure, and thrombocytopenia. Exclusion criteria for the study included a history of preeclampsia, preeclampsia superimposed on chronic hypertension, coronary artery disease, atrial flutter or fibrillation, atrioventricular conduction abnormalities, and pre-existing kidney, liver, or pulmonary disease. Additionally, patients with acute or chronic inflammatory conditions, hypo- or hyperthyroidism, hyperlipidemia, or diabetes mellitus were excluded. The presence of ECGs without a clearly identifiable P-wave or a measurable QT interval also constituted grounds for exclusion.

For all participants, comprehensive data were recorded on a standardized form. This included demographic information such as age, parity, and body mass index (BMI). Clinical data collected were gestational age, type of delivery, smoking status, systolic and diastolic blood pressure, heart rate, and family history of preeclampsia and cardiovascular disease. Laboratory parameters documented were proteinuria, creatinine, hemoglobin, and platelet count. Finally, all electrocardiogram findings were also recorded.

ECG analysis: All standard 12-lead ECGs were recorded with patients in a supine position following a 10-*min* rest period using 1-*mV/cm* voltage calibration. To optimize temporal resolution for precise manual interval measurements, specifically P-wave dispersion and peak times, ECGs were recorded at an expanded paper speed of 50 *mm/s* rather than the standard 25 *mm/s*. At 50 *mm/s*, each 1-*mm* small grid square represents 0.02 s (20 *ms*) rather than 0.04 s (40 *ms*), providing doubled horizontal magnification that significantly reduces measurement error during caliper assessment.

ECG parameter measurements: P-wave duration and dispersion were measured from the preoperative ECG. The P-wave was measured from the beginning of the first atrial deflection to its return to the isoelectric line. The maximum (Pmax) and minimum (Pmin) P-wave durations were identified across all twelve leads, and Pd was calculated as the difference between these two values (Pd= Pmax−Pmin). Heart rate, QRS complex duration, RR interval, and QT interval were also calculated from the preoperative ECG. QTc was determined using Bazett's formula (20). QTd was calculated as the difference between the maximum and minimum QT intervals measured from the onset of the QRS complex to the end of the T-wave across each ECG lead (21). PWPT was measured in lead II from the P-wave onset to its maximum positive peak, and in lead V1 from the P-wave onset to its maximum negative peak. RWPT was measured in leads V5 and V6 from the QRS complex onset to the peak of the R-wave. All measurements were performed manually using a magnifying glass and a caliper. The individual responsible for the measurements was blinded to the participants' study group assignments to minimize bias (Figure 1).

Sample size calculation: Sample size was determined based on primary mean differences reported by Kirbas et al. (18) (µ1=42.1, µ2=58, δ1=17, δ2=10.8). Assuming α=0.05 and 80% statistical power (β=0.20), a minimum of 24 participants per group was required. To account for a potential 20% attrition or unusable ECG recordings, 30 participants per group were recruited (n=90 total).

Statistical analysis: Continuous parameters were evaluated for normality using the Shapiro-Wilk

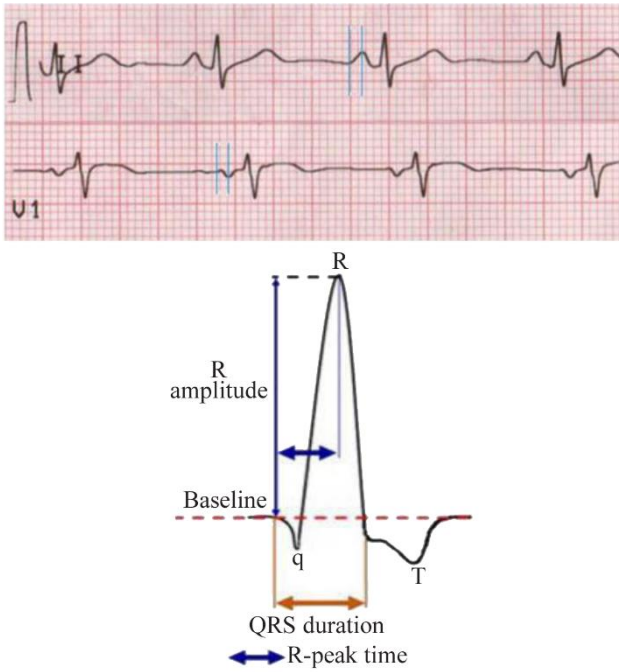


Figure 1. Representative surface 12-lead ECG tracing recorded at an expanded paper speed of 50 *mm/s* (top) alongside a schematic diagram illustrating the manual measurement of QRS duration and R-wave peak time (RWPT) from the onset of the QRS complex to the R-wave peak (bottom)

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test. Differences across the three groups were evaluated using One-Way Analysis of Variance (ANOVA). Upon establishing overall statistical significance ($p<0.05$), pairwise post-hoc comparisons were performed using Tukey's Honestly Significant Difference (HSD) test to identify specific group differences, reporting adjusted p-values.

Results

Demographic and clinical baseline characteristics were comparable across all three groups. There were no statistically significant differences in mean age (29.70±6.70 years in severe preeclampsia, 30.06±6.54 years in mild preeclampsia, and 29.60±6.36 years in controls; p=0.95), gestational age (30.36±3.16 weeks, 31.39±3.03 weeks, and 31.90±3.90 weeks, respectively; p= 0.34), or parity (1.83±0.83, 1.73±0.90, and 2.20± 1.50, respectively; p=0.17) (Table 1).

The mean peak time of the P-wave in lead II was found to be 41.15±4.65 *ms* in the severe preeclampsia group, 36.17±8.13 *ms* in the mild preeclampsia group, and 30.10±7.85 *ms* in the healthy control group. A one-way ANOVA test revealed a significant difference in this measurement among the three groups (p=0.001), as detailed in table 2.

Post-hoc analysis using Tukey's test confirmed significant differences in pairwise comparisons. A significant difference was observed between the severe and mild preeclampsia groups for mean P-wave peak time (p=0.01). A significant difference was also observed between the severe preeclampsia group and the healthy control group (p=0.001). Additionally, a significant difference was found between the mild preeclampsia group and the healthy control group (p=0.01). These results indicate that the P-wave peak time in lead II progressively increases with the severity of preeclampsia. The mean peak time of the P-wave in lead V1 was 44.13±18.22 *ms* in the severe preeclampsia group, 36.43±9.80 *ms* in the mild preeclampsia group, and 74.29±7.48 *ms* in the healthy control group. A one-way ANOVA indicated a significant difference in this parameter among the three groups (p=0.001), as shown in table 2.

Post-hoc analysis using Tukey's test revealed significant differences in pairwise comparisons. A significant difference was observed between the severe and mild preeclampsia groups (p=0.03). A significant difference was also found between the severe preeclampsia and healthy control groups (p=0.001). Additionally, a significant difference was observed between the mild preeclampsia and healthy control groups (p=0.01). These results suggest that the P-wave peak time in lead V1 varies significantly with the presence and severity of preeclampsia. The mean peak time of the R wave in lead V5 was 38.70±7.27 *ms* in the severe preeclampsia group, 32.38±5.65 *ms* in the mild preeclampsia group, and 28.34±6.89 *ms* in the healthy control group. A one-way ANOVA test demonstrated a significant difference in this measurement across the three groups (p=0.001), as detailed in table 2.

Post-hoc analysis using Tukey's test revealed significant differences in pairwise comparisons. A significant difference was observed between the severe and mild preeclampsia groups (p=0.03). A significant difference was also found between the

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Table 1. Frequency distribution of demographic and clinical characteristics of mothers in the three study groups

	Severe preeclampsia	Mild preeclampsia	Healthy group	p-value
Mean age (year)	29.70±6.70	30.06±6.54	29.60±6.36	0.95
Mean gestational age (weeks)	30.36±3.16	31.39±3.03	31.90±3.9	0.34
Mean parity	1.83±0.83	1.73±0.90	2.2±1.5	0.17

Data presented as Mean ± Standard Deviation. P-values calculated via One-Way Analysis of Variance (ANOVA)

Table 2. Comparison of P-wave and R-wave peak times across study groups

Parameter (*ms*)	Severe preeclampsia (n=30)	Mild preeclampsia (n=30)	Healthy controls (n=30)	p-value*
PWPT lead II	41.15±4.65	36.17±8.13	30.10±7.85	0.001
PWPT lead V1	44.13±18.22	36.43±9.80	74.29±7.48	0.008
RWPT lead V5	38.70±7.27	32.38±5.65	28.34±6.89	0.001
RWPT lead V6	36.72±7.62	32.04±5.62	25.90±6.65	0.001

PWPT = P-wave peak time; RWPT = R-wave peak time. All units reported in milliseconds (*ms*). * P-values calculated via one-way ANOVA

severe preeclampsia and healthy control groups (p=0.001). Additionally, a significant difference was observed between the mild preeclampsia and healthy control groups (p=0.04). These results indicate that the R-wave peak time in lead V5 progressively increases with the severity of preeclampsia. The mean peak time of the R-wave in lead V6 was 36.72±7.62 *ms* in the severe preeclampsia group, 32.04±5.62 *ms* in the mild preeclampsia group, and 25.90±6.65 *ms* in the healthy control group. A one-way ANOVA test demonstrated a significant difference in this measurement across the three groups (p=0.001), as detailed in table 2.

Post-hoc analysis using Tukey's test revealed significant differences in all pairwise comparisons. A significant difference was observed between the severe and mild preeclampsia groups (p=0.02). A significant difference was also found between the severe preeclampsia and healthy control groups (p=0.001). Similarly, a significant difference was found between the mild preeclampsia and healthy control groups (p=0.001). These results indicate that the R-wave peak time in lead V6 progressively increases with the severity of preeclampsia.

Discussion

In 2011, the American Heart Association (AHA) recognized preeclampsia as an independent, gender-specific risk factor for cardiovascular disease (22). Studies have estimated that women with a history of preeclampsia have a 31% greater ten-year cardiovascular disease (CVD) risk based on the Framingham score, while those with gestational hypertension have a 27% greater risk, when compared to women without a history of pregnancy-related complications (23). Therefore, in this study, the relationship between Pd and QTd with the presence and severity of preeclampsia was investigated.

Current evidence suggests that both pre-pregnancy risk factors and the preeclampsia itself may contribute to the development of long-term CVD risk. Preeclampsia is now considered a significant cardiovascular risk factor. The primary pathophysiological mechanisms involved in this condition include endothelial dysfunction, severe inflammation, hypercoagulability, increased oxidative stress, and reduced uteroplacental blood flow (24). One recent study found that the 10-year CVD risk, as calculated by the Framingham score, is 31% higher in women with a history of preeclampsia and 27% higher in those with a history of gestational hypertension (25).

In our study, there was no significant difference in terms of gestational age between the three study groups. Also, the results of the present study showed that there was no significant difference in terms of parity between the three study groups.

Various cardiovascular risk factors have been reported to influence Pd (26). Abnormal changes in P-wave morphology during pregnancy have been identified as potential predictors for hypertensive disorders in previous studies (27, 28). Specifically, the P-wave duration, including Pd, was found to be significantly increased in pregnant women with preeclampsia compared to healthy controls (17). Meta-analyses have shown that increased PWV, an indicator of vascular stiffness linked to endothelial dysfunction, is a significant feature of preeclampsia (29). A study of women 16 months postpartum following a preeclamptic pregnancy showed a trend toward increased aortic-to-brachial PWV, suggesting persistent vascular stiffness, particularly in smaller arteries (30).

Our study's findings are consistent with these observations, as clear differences were found in women with severe preeclampsia compared to those with mild preeclampsia and healthy controls. A significant difference was observed in the mean peak time of the P-wave in lead II, which was notably longer in women with severe preeclampsia than in both the mild preeclampsia and healthy control groups. Furthermore, a recent study has proposed that identifying abnormal P-wave morphology in lead V1 during pregnancy may serve as a potential predictor for the development of a hypertensive disorder (31).

In agreement with the findings of Kirbas et al. (18), our study found that Pd values were significantly prolonged in pregnant women with preeclampsia, with the extent of prolongation correlating to disease severity. Pd is a marker that reflects atrial size and is influenced by various cardiovascular risk factors. It has been independently linked to an elevated risk of atrial fibrillation (AF), recurrent transient ischemic attacks (TIA), and stroke (32). These alterations in P-wave parameters are thought to stem from electrophysiological and electromechanical abnormalities caused by exaggerated systemic inflammation, ischemia, increased oxidative stress, and enhanced sympathetic activity (7), all of which are closely associated with preeclampsia (33). This research is con-

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sistent with the analysis by Raffaelli et al. (19), who also found a significantly longer P-wave duration in preeclamptic women compared to a normal pregnancy control group. Furthermore, Brown (34) has underscored the multifactorial nature of cardiovascular changes in preeclampsia, highlighting that ECG parameters can offer valuable insights into cardiac function and prognosis.

A significant finding of our study was the novel demonstration of a simultaneous association between both P-wave and QT interval duration parameters, specifically Pd and QTd, and the presence and severity of preeclampsia. Our findings demonstrate progressive prolongations in both PWPT and RWPT alongside increased Pd and QTd in women with preeclampsia, correlating directly with clinical severity. These results align with supportive evidence from Kirbas et al. (18) and Raffaelli et al. (19), who reported significant prolongations of Pd and QTd in preeclamptic pregnancies relative to healthy controls, attributing these alterations to exaggerated autonomic activation, microvascular ischemia, and systemic inflammation. Similarly, Angeli et al. (27, 28) underscored the utility of novel P-wave parameters in identifying subclinical atrial conduction delays secondary to acute hypertensive load.

QTd is a well-established marker of heterogeneous ventricular repolarization (35) and has been tested for its predictive value for cardiovascular mortality in several clinical conditions (36, 37). Our findings confirm that women with preeclampsia exhibit greater QT interval dispersion compared to the control group, suggesting an alteration in ventricular repolarization in this population. The discriminative effect of Pd in distinguishing between mild and severe preeclampsia may hold clinical utility. The clinical utility of QTd as a predictor of cardiovascular mortality has been reported in various conditions, including hypertension, chronic renal failure requiring dialysis, type 2 diabetes, acute ST-elevation myocardial infarction, metabolic syndrome, and obstructive sleep apnea (38, 39). Consistent with these findings, our study revealed a significant difference in the average R-wave peak time in leads V5 and V6. The average R-wave peak time was significantly longer in women with severe preeclampsia compared to both the mild preeclampsia and healthy control groups.

Impaired uteroplacental perfusion in preeclampsia may alter cardiac electrophysiology and ventricular repolarization prior to the clinical manifestation of overt systemic symptoms (39). In support of this mechanism, Raffaelli et al. (19) documented significant alterations in ventricular repolarization, including QTc prolongation and elevated QTd, in preeclamptic pregnancies compared to normotensive controls. Conversely, our findings contrast with those of Loukrakpam et al. (40), who observed minimal variation in basic electrocardiographic parameters during mild gestational hypertension, reporting that pathological prolongations were restricted to severe preeclampsia or eclampsia. These discrepancies likely stem from methodological differences, particularly our use of a higher paper speed resolution (50 *mm/s vs*. standard 25 *mm/s*), variations in gestational age at evaluation, or differing clinical threshold criteria for mild disease.

Furthermore, altered cardiac electrical activity in preeclampsia reflects heightened sympathetic tone and delayed myocardial conduction. Murphy et al. (41) demonstrated that women with a history of preeclampsia exhibit persistent postpartum autonomic dysregulation, evidenced by reduced heart rate variability, alongside prolonged P-wave and QRS durations, with QRS prolongation persisting independently of blood pressure. Aligning with these observations, our antenatal data reveal significant, severity-dependent prolongations in both P-wave and R-wave peak times across leads II, V1, V5, and V6 in women with severe preeclampsia relative to mild cases and healthy controls. However, whereas Murphy et al. noted persistent postpartum delays, our demonstration of acute antenatal RWPT prolongation in lateral precordial leads (V5 and V6) suggests that acute left ventricular pressure overload induces transient intraventricular conduction delays during late gestation. If sustained, these electrophysiological alterations may predispose to chronic structural remodeling and an elevated risk of postpartum arrhythmogenesis.

This study has several limitations. First, the relatively small sample size may limit the generalizability of our findings. Despite this, a significant relationship between Pd, QTd, and the presence of preeclampsia was demonstrated. Second, the manual calculation of P-wave and QT interval parameters using a magnifying lens, without the aid of computer-assisted software or Holter monitoring and electrophysiological evaluation, likely introduced increased inter- and intra-observer variability.

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Conclusion

The findings of this study reveal a significant relationship between preeclampsia and the peak times of the P- and R-waves of ECG. The observed changes suggest that increased cardiac sympathetic activity and delayed cardiac conduction in pregnant women with preeclampsia may lead to long-term structural changes in the heart and a heightened risk of arrhythmias. Therefore, the early identification of these electrocardiogram abnormalities in patients with preeclampsia offers a valuable opportunity to mitigate future cardiac complications through timely diagnosis and intervention.

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Conflict of Interest

The authors have no competing interests to declare that are relevant to the content of this article.

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