

Research Article

Cardiac Characteristic in Preeclampsia and Normotensive Pregnant Women Attending Bugando Medical Centre Tanzania

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Abstract

Background: Hemodynamic changes brought on by pregnancy, may affect the heart either by physiological or pathological changes. Preeclampsia-eclampsia (PE-E) primarily affects the cardiovascular system which leads to low cardiac output and a high systemic vascular resistance, this lead to abnormal adaptive mechanisms that may be a result of changes in vascular tone and may have effects on a woman's life. **Methodology:** 273 women from an obstetric ward or prenatal clinic who were at least 28 weeks pregnant and those who are 10 days postpartum at BMC participated in the comparative cross-sectional study. Transthoracic echocardiograms (TTE) were performed along with interviews of the recruited subjects. A questionnaire was implemented to gather data, and STATA version 13 was used for analysis. A level of statistical significance was defined as less than 0.05 p-value in multivariate logistic regression. **Results:** 49.45% out of 273 women, had PE-E. 78.7% out of 273 participants were ≤ 35 years of age. The prevalence of abnormal structural cardiac characteristics was 60% and 28.57% in PE-E and normotensive pregnant women respectively, while the prevalence of abnormal functional cardiac characteristics between them was nearly the same. No difference in LA enlargement between PE-E and normotensive pregnant women. Interventricular septum and posterior wall thickness of the LV were more than 5 to 8-fold in PE-E compared to normotensive pregnant women. EF and diastolic dysfunction were 3 and 2 fold respectively, in PE-E compared to normotensive pregnant women. Abnormal structural cardiac characteristics were more common among women with severe forms of PE-E by 2 to 9 fold, but there is no statistically significant in functional cardiac characteristics with the severity of PE-E. **Conclusion and recommendations:** Structural and functional cardiac abnormalities were more common in PE-E than in normotensive pregnant women, hence TTE should include as a part of an investigation into PE-E and symptomatic normotensive. Cardiac structural and functional should be followed up in patients with PE-E even after termination of pregnancy. There is a need for a larger longitudinal study to determine the proper timing of cardiac evaluation in PE-E both during ANC and post-delivery.

Keywords

Pre-eclampsia-eclampsia, Structural Cardiac Characteristics, Functional Cardiac Characteristics, Transthoracic Echocardiogram

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Received: 17 October 2025; **Accepted:** 27 October 2025; **Published:** 3 December 2025



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1. Introduction

During pregnancy, 10% of women have high blood pressure, and pre-eclampsia complicates 2% to 8% of pregnancies. Maternal mortality caused by PE-E ranges from 9% to 26% in higher and low-income countries [1]. Similarly, the incidence of preeclampsia at BMC in Tanzania was found to be 1.4%, with a death rate of 7.89% [2, 3].

Normally, pregnancy causes cardiovascular changes that begin at five to eight weeks of gestation (GA) and then remain at their maximum at 28-32 weeks [4]. These changes include an increase in cardiac output (CO) and a decrease in systemic vascular resistance [5].

PE-E is a systemic condition with unknown causes but it is hypothesized that due to abnormal implantation results in numerous placenta-derived circulatory agents, which cause primary cardiovascular and global vascular endothelial damage [6]. The pathophysiology of PE-E to the heart depends on the severity of the disease and the resulting ventricle remodeling, diastolic, and systolic impairment, as keeping a balance between myocardial oxygen demand and its supply [7].

Contradictory results are mostly attributed to limitations in technology in lower and middle-income countries, patient selection, and data interpretation, so this study is needed to outline structural and functional cardiac characteristics in preeclampsia-eclampsia in comparison to normotensive pregnant women attending Bugando Medical Center, Tanzania'.

2. Materials and Methods

A hospital-based cross-sectional study was conducted at Bugando Medical Centre (BMC)-Tanzania in the Department of Obstetrics and Cardiology. BMC, teaching, and Consultant Tertiary Referral Hospital –Lake Zone, located in the Mwanza region at Nyamagana District. The average number of pregnant women who deliver is approximately 400 per month of which 10 cases were PE-E.

The study was carried out from January 2022 to January 2023, on all pregnant women attending antenatal and admitted labor wards, who voluntarily consented. Two groups of singleton pregnant women were recruited, where PE-E and those who are normotensive pregnant women both groups are taken ≥ 28 weeks' GA based on LNMP and those who are 10 days post-delivery. Participants' confidentiality was ensured. But Women with chronic hypertension, diabetic Mellitus, those in an active phase of labor, and pre-existing cardiovascular diseases are excluded from this study.

The sample size was estimated by using the Kish Leslie formula [8] and a minimum number of 113 participants were obtained in each group. Participants were recruited by a serial random sampling method until the required sample size was met in each group.

A structured questionnaire form was used for data collec-

tion and filled out by the principal investigator and research assistants. Socio-demographic data (age, weight, height, BMI, gravidity, parity, number of abortions, GA at the date of interview, and education status) were recorded. History taking and physical examination (cardiovascular and abdominal) were done and the clinical severity of PE-E was determined.

The diagnosis of PE-E was made by using BP measurements, urine for protein, and other investigations extracted from the patient's file. BP was measured twice at least 4 hours apart and one reading was recorded. All of the measurements were performed in either the left or right arm, with the arm at the level of the heart, a systolic BP $\geq 140/90$ mmHg, plus proteinuria of $\geq 1+$ on dipstick assessment. The BP measurement was done using an Automatic Arm BP machine (Omron HEM 7120) and urine for protein was measured with URINOX- 10 (Urinalysis Test Strips). Preeclampsia without severe features was when the BP is 140/90 to 160/110 mmHg without proteinuria and no signs of organ damage. And preeclampsia with a severe feature was when BP $\geq 160/110$ mmHg with proteinuria on dipstick of $\geq 1+$ and signs of organ damage (low platelet, elevated serum creatinine, renal insufficiency, pulmonary edema, new onset of headache, visual disturbance). Apart from that, Eclampsia patients will include those participants with pre-eclampsia and grand mal seizure, altered mental status, or loss of consciousness. For the second group of normotensive pregnancy, women also were taken. The weight measurements were done using a weighing scale (capacity 160 kg) with an incorporated stadiometer (maximum height 190 cm) and BMI was calculated as weight (kg) divided by (height)² (m²).

The TTE was performed at the BMC. General Electric VIVID IQ machine was used for examination and a report was recorded in a patient's file and a questionnaire. A participant was lying in a left lateral supine position, the cardiac probe produce images of the heart to identify cardiac structure and function. No risks were obtained in a standard TTE, except the feeling of some discomfort from the transducer which was held very firmly against the chest.

Firmness was important to produce the best images of the heart, no special preparations are necessary for a standard TTE. The patient was asked to remove clothing from the waist up and lie on an examination bed. The cardiologist was attach sticky electrodes to the body to detect and conduct the heart's electrical. A gel was applied to improve the conduction of sound waves and record images of sound-wave.

All echocardiographic examinations were performed by an experienced cardiologist, and the principal investigator got a chance to learn how TTE is done. LV ejection fraction was determined using the Teicholz method from motion-mode guided parasternal long-axis images of the left ventricle and was taken as a measure of LV systolic function. An EF of $<50\%$ was considered a systolic dysfunction [9].

LV filling was recorded at the level of the tips of the mitral leaflets. The leading edge of the mitral flow pattern will be traced to derive peak early (E) and atrial (A) velocities, E/A ratio, and E-deceleration time. Isovolumic relaxation time will be measured from the leading edge of the aortic valve closure spike to the leading edge of the mitral valve opening spike. The medial early diastolic mitral annular velocity (E') was measured by spectral tissue Doppler imaging in apical four-chamber views. The ratio of E to E' velocity (E/E' ratio) was taken as an estimation of LV filling pressure and was considered increased when it is ≥ 14 [10]. LV diastolic dysfunction was defined as mild (impaired relaxation), moderate (pseudonormal pattern), and severe (restrictive pattern) based on transmitral inflow in combination with the diastolic mitral annular velocities [11, 12]. LV mass was calculated using the anatomically validated formula by Devereux et al and indexed to body surface area. LV hypertrophy was defined as LV mass index >95 kg/m² in women [9].

Data were analyzed by computer using STATA version 13.0. Nominal data were described as frequency and percentage and compared using Chi-Square tests. Univariate and

multivariate logistic regression were used to determine the factors associated with the outcome (structural and functional cardiac characteristics). Factors with p-value less than 0.05 on univariate were subjected to multivariate. A p-value less than 0.05 was considered significant.

To ensure the internal validity of the study the data-collecting tool was pre-tested and the principal investigator ensured the completeness and consistency of the edited data collected.

Ethical clearance was sought from the Joint CUHAS/BMC Ethics and Review Committee before the commencement of the study with certificate number: CREC/517/2022. Permission to carry out the study was obtained from BMC hospital authorities.

3. Results

A total of 273 participants were enrolled and analyzed to answer our study objectives (Figure 1).

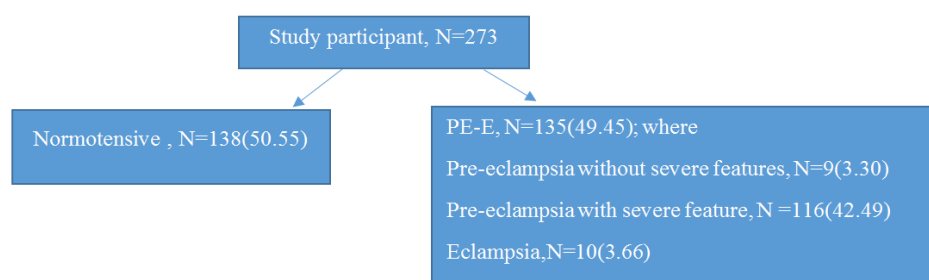


Figure 1. Flowchart of study participants.

Social Demographic and clinical characteristics of study participants

Among women enrolled, 78.7% out of 273 participants were ≤ 35 years of age, and 54.2% come from their self-initiatives (Table 1).

Table 1. Social demographic and clinical characteristics of study participants.

Variable		Total N=273(%)
Age category	≤ 35	215(78.7%)
	>35	58(21.12%)
Education status	Formal	251(91.9%)
	Informal	22(8.06%)
Marital status	married	243(89.0%)
	not married	30(10.9%)
Mode admission	Referred	125(45.8%)
	From home	148(54.2%)
Occupation	Formal employed	175(64.1%)

Variable		Total N=273(%)
Residence	Informal employed	98(35.9%)
	Rural	100(36.6%)
	Urban	173(63.4%)
BMI status	normal=18.8 -24.9	75(27.5%)
	over weight=25-29.9	106(38.8%)
	obese> or=30	92(33.7%)
Duration of sampling	≤34 weeks	57(20.9%)
	>34 weeks	113(41.4%)
Parity category	After delivery	104(37.7%)
	Prime	70(25.6%)
	Parous	203(74.4%)

Prevalence of abnormal cardiac characteristics among pregnant women attending BMC

Abnormal structural cardiac characteristics in PE-E are 60% and normotensive is 28.57%. (Figure 2)

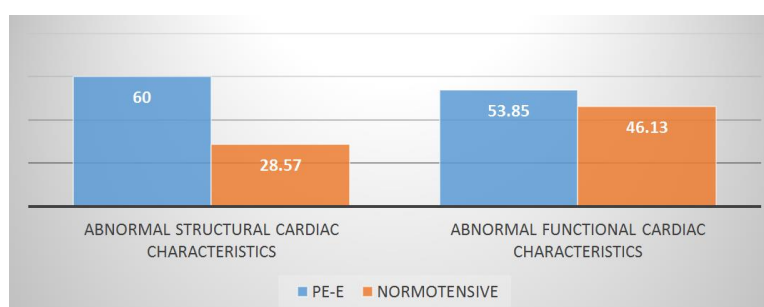


Figure 2. Prevalence of abnormal cardiac characteristics (structural and functional) among pregnant women attending BMC.

Association between pregnancy hemodynamic state to structural and functional cardiac characteristics among pregnant women attending BMC.

On univariate and multivariate logistic regression the vari-

able hemodynamics state is associated with intraventricular septum thickness, left ventricle posterior wall, reduced ejection fraction, and diastolic dysfunction. (Table 2)

Table 2. Association between pregnancy hemodynamic state to structural and functional cardiac characteristics among pregnant women attending BMC.

Outcomes	Normotensive, N=138	PE-E, N=135	Univariate analysis		Multivariate analysis	
			cOR [95% CI]	P-value	aOR [95% CI]	P-value
Left atrium						
Normal	18(13.04)	18(13.33)	1.0			
Abnormal	120(86.96)	117(86.66)	0.9 [0.48-1.96]	0.944	-	-
Left ventricle internal diameter						
Normal	137(99.28)	130(96.29)	1.0			

Outcomes	Normotensive, N=138	PE-E, N=135	Univariate analysis		Multivariate analysis	
			cOR [95% CI]	P-value	aOR [95% CI]	P-value
Abnormal	1(0.72)	5(3.71)	5.2 [0.60- 45.70]	0.132	-	-
<i>Intraventricular septum thickness</i>						
Normal	122(88.41)	82(60.74)	1.0			
Abnormal	16(11.59)	53(39.26)	4.9 [2.63-9.20]	<0.001	4.9 [2.6-9.1]	<0.001
<i>Left ventricular posterior wall</i>						
Normal	127(92.03)	80(59.26)	1.0			
Abnormal	11(7.97)	55(39.86)	7.9 [3.9-16.0]	<0.001	7.8 [3.8-15.9]	<0.001
<i>Ejection fraction EF%</i>						
Normal	131(94.93)	119(88.15)	1.0			
Abnormal	7(5.07)	16(11.85)	2.5 [1.0-6.3]	0.050	2.6 [1.0-6.4]	0.045
<i>Grading of EF%</i>						
Normal	99(71.74)	86(63.70)	1.0			
Hyerdynamic	32(23.19)	31(22.96)	1.1 [0.6-1.9]	0.709	1.1 [0.6-1.9]	0.723
Reduced	7(5.07)	18(13.33)	2.9 [1.1-7.4]	0.021	3.0 [1.2-7.6]	0.018
<i>Diastolic function</i>						
Normal	110(79.71)	93(68.89)	1.0			
Abnormal	28(20.29)	42(31.11)	1.7 [1.0-3.0]	0.042	1.7 [1.0-3.1]	0.039
<i>Grade of diastolic function</i>						
Grade 2(normal)	109(78.99)	93(68.89)	1.0			
Grade 1	3(2.17)	4(2.96)	1.5 [0.3-7.1]	0.565	1.5 [0.3-7.3]	0.546
Grade 3	25(18.12)	38(28.15)	1.7 [1.0-3.1]	0.049	1.8 [1.0-3.2]	0.044

Association between the cardiac characteristics and severity of PE-E

On univariate and multivariate logistic regression, the severity of BP was seen to be associated with abnormal structural cardiac (Table 3).

Table 3. Association between the cardiac characteristics and severity of PE-E in our study participants.

Structural						
Severity of PE-E	Abnormal n (%)	Normal n (%)	Univariate OR [95%CI]	P-value	Multivariate AOR [95%CI]	P-value
SBP						
≤139	33(20)	132(80)	1.0			
140-159	25(40.98)	36(59.02)	2.8 [1.46-5.25]	<0.001	2.3 [1.11-4.95]	0.020
≥160	26(55.32)	21(44.68)	4.9 [2.48-9.87]	<0.001	2.3 [0.83-6.62]	<0.001
DBP						

Structural						
Severity of PE-E	Abnormal n (%)	Normal n (%)	Univariate OR [95%CI]	P-value	Multivariate AOR [95%CI]	P-value
<90	39(22.16)	137(77.84)	1.0			
91-109	28(38.36)	45(61.64)	2.3 [1.21-3.94]	<0.001	1.3 [0.6-2.76]	0.520
>110	17(70.83)	7(29.17)	8.5 [3.30-22.04]	<0.001	4.3 [1.15-15.66]	0.030
Functional						
Severity of PE-E	Abnormal	Normal	Univariate OR [95%CI]	P-value	Multivariate AOR [95%CI]	P-value
SBP						
≤139	13(7.88)	152(92.12)	1.0			
140-159	7(11.48)	54(88.52)	1.5 [0.57-3.99]	0.400	1.5 [0.5-4.0]	0.300
≥160	41(87.23)	41(87.23)	1.7 [0.61-4.78]	0.300	1.7 [0.6-5.0]	0.200
DBP						
<90	17(9.66)	159(90.34)	1.0			
91-109	6(8.22)	67(91.74)	0.8 [0.31-2.21]	0.720	0.8 [0.3-2.2]	0.700
>110	3(9.52)	21(87.50)	1.3 [0.36-4.94]	0.660	1.3 [0.3-5.07]	0.640

4. Discussion

Preeclampsia-Eclampsia is a multisystem disorder that can affect almost every organ. Association between PE-E and cardiovascular morbidity has been highlighted where structural and functional cardiac characteristics were thoroughly investigated in this study.

This study found that the prevalence of abnormal structural cardiac characteristics in PE-E is higher compared to normotensive pregnant women. This could be explained by PE-E accompanied by an increase in PVR where the heart especially the LV will hypertrophy to compensate for the ongoing resistance and end with structural changes. Also, the observed structural changes in normotensive might be caused by physiological plasma volume expansion occurring in the 2nd and early 3rd trimesters [13], as previously reported [14].

Moreover, the prevalence of abnormal functional cardiac characteristics among PE-E and normotensive pregnant mothers was nearly the same. The observed minimum variation of 7.7% between the aforementioned groups could be due to the timely intervention provided in our PE-E mothers such as early diagnosis, use of vasodilators, and termination of pregnancy based on the severity of preeclampsia, which preserved the cardiac function, as previous reported [15, 16].

Our investigation found that normotensive and PE-E women had a comparable proportion of LA enlargement. It

should be highlighted that LA functions independently of hemodynamic condition; LA acts as a reservoir by enhancing its size and function over time to maintain enough blood flow; this physiology occurs in both normotensive and PE-E patients, which is why we noticed a similarity in their proportions. However, it was expected that in PE-E, their LA would be dilated more than in normotensive patients. This is not the case because, in our PE-E, there is placental insufficiency and vascular reactivity, which can result in systemic complications, increasing LV pressure, decreased valve compliance, and a rise in atrial wall tension to overcome that resistance. Our findings agree with previous reports [17-19].

Furthermore, our investigation spared the internal diameter of LV of normotensive patients while seeing a significant enlargement of the internal diameter of LV, septum thickness, and posterior wall in PE-E patients. This is because LV enlargement is directly related to an increase in workload. However, in normotensive pregnant women, despite significant physiological changes in cardiac remodeling, the ratio of ventricular wall thickness to radius did not change. Our findings are the same as previously reported [20-22] because both studies excluded metabolic risk, which causes endovascular disease and abnormal cardiovascular findings. Unfortunately, our findings contrast with the other study [23], which might be attributed to the sample criteria, which include gestational age from 9 to 42 weeks and diverse ethnicities. In contrast to our normal pregnancy, PE-E patients ex-

perience global vasoconstriction and vascular resistance, resulting in LV remodeling with concentric hypertrophy rather than normative eccentric remodeling [24].

Unfortunately, 2-3 fold of our PE-E indicated a drop in EF% compared to normotensive pregnancy women, the observed finding is due to weakening and injured cardiac muscles, which make contractions incomplete, as the heart may adjust by pumping, causing ventricle hypertrophy, which reduces the ventricle's capacity to fill and pump. This finding was consistent with other research [25, 26]. These similarities were brought by a compensatory mechanism for low cardiac output, which result in an increased heart rate and initially in an increase of BP, later on BP may drop and a patient may become hypotensive. However, our results could be attributed to being overweight, obese, multiparous, and black, all of which are risk factors for serious adverse cardiovascular events [27], resulting in a one-fold reduction of EF% in our normotensive pregnant women.

Furthermore, as compared to our normotensive pregnant women, there is a more than one-fold increase in LV diastolic dysfunction in PE-E. This is because PE-E is a unique illness that is hypothesized to cause hypo-perfusion; our PE-E participants have increased vascular resistance and arterial hypertension, as well as visible structural alterations; however, our normotensive patients experienced physiological events of increased preload and decreased afterload due to peripheral vasodilation caused by hormonal effect. Our results agree with those previously published studies [16, 28-30], with the outcome of our study being related to advanced maternal age, severe PE-E from other institutions, and early disease of fewer than 34 weeks of gestation. These have been linked to increased diastolic dysfunction in PE-E [31-33]. Furthermore, the degree of PE-E was related to the grade of diastolic dysfunction in our participants, showing that preload variations during normal pregnancy result in reversible physiological LV dilation rather than PE-E, as previously reported [16].

Abnormal structural cardiac characteristics in our study, were more common among women with severe forms of PE-E by 2 to 9 fold. This was brought by localized myocardial ischemia and fibrosis which affected myocardial contractility and relaxation and led to unfavorable biventricular remodeling and hypertrophy [34]. Fortunately, in our results, there is no statistically significant in functional cardiac characteristics with the severity of PE-E, because prolonged vascular constriction, in PE-E results, is a compensatory mechanism of increased hemoconcentration, and maintaining a cardiac output for a while. Apart from that early intervention in our Tertiary Centre help our patients to minimize cardiac morbidity although we lack immediate assessment of maternal cardiac function postpartum period of the same patients.

5. Conclusion

The prevalence of structural abnormality in PE-E was

higher than in normotensive clients, while functional abnormalities were almost the same. MACEs were 2 to 8 times higher in PE-E than in normotensive pregnant women.

Recommendations

Structural and functional cardiac abnormalities were more common in PE-E than in normotensive pregnant women, hence TTE should include as a part of an investigation into PE-E and symptomatic normotensive. Cardiac structural and functional should be followed up in patients with PE-E even after termination of pregnancy. There is a need for a larger longitudinal study to determine the proper timing of cardiac evaluation in PE-E both during ANC and post-delivery.

Abbreviations

BMC	Bugando Medical Centre
BMI	Body Mass Index
CHD	Coronary Heart Disease
CI	Cardiac Index
CO	Cardiac Output
CUHAS	The Catholic University of Health and Allied Science
CVD	Cardiovascular Disease
DD	Diastolic Dysfunction
EF	Ejection Fraction
GA	Gestation Age
HDP	Hypertensive Disorders in Pregnancy
HEELP	Hemolysis, Elevated Liver Enzyme, and Low Platelets Count
HF	Heart Failure
HR	Heart Rate
LA	Left Atrium
LAP	Left Atria Pressure
LV	Left Ventricle
LVDD	left Ventricular Diastolic Dysfunction
LVH	Left Ventricle Hypertrophy
LVM	Left Ventricle Mass
MACEs	Major Adverse Cardiovascular Events
MFI	Myocardial Performance Index
MI	Myocardial Infarction
MMR	Maternal Mortality Rate
OBGYN	Obstetrics and Gynecology
PE-E	Preeclampsia –eclampsia
RV	Right Ventricle
TR	Total Resistance
TTE	Transthoracic Echocardiogram
TVRI	Total Vascular Resistance Index
VH	Ventricle Hypertrophy

Acknowledgments

The authors extend their sincere gratitude to enrolled mothers for their readiness to participate in the study. We also thank the Catholic University of Health and allied sci-

ences, the Department of Obstetrics and Gynecology, and the cardiology department of Bugando Medical Centre, for their support and contribution to our study.

Author Contributions

Fatma Said Mohammed: Investigation, Writing – original draft, Writing – review & editing

Eva Felician Mujuni: Conceptualization, Data curation, Formal Analysis, Writing – original draft, Writing – review & editing

Edgar Ndaboine: Data curation, Formal Analysis, Investigation, Methodology, Supervision, Writing – original draft

Richard Kiritta: Investigation, Writing – original draft, Writing – review & editing

Funding

The health cost offered to individual patients was partly waived by the Hospital administration by considering the patient's economic status as required by the health policy of the United Republic of Tanzania. The cost of data collection and manuscript preparation was covered by the authors.

Availability of Data and Material

The datasets used and analyzed during the current study are available from the corresponding author upon request.

Ethical Approval and Consent to Participate

Ethical clearance and permission to conduct this study were sought from the joint CUHAS/BMC Research Ethics and Review Committee, and research clearance certificate number CREC/517/2022 was granted. All enrolled study participants voluntarily signed an informed consent to participate in this study after being explained the objectives of the study.

Consent for Publication

Written informed consent to publish these study findings was obtained from the patients, copies of which are available for review by the Editor-in-chief of this journal. Additionally, consent was sought and granted by the Catholic University of Health and Allied Sciences Directorate of Research and Publication to publish this work. A copy of the clearance document is also available for review by the Editor-in-Chief of this journal.

Conflicts of Interest

All authors declare that they have no competing interest.

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