ORIGINAL ARTICLE

The relationship between red-cell distribution width and abnormal left ventricle geometric patterns in patients with untreated essential hypertension

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Abnormal left ventricle (LV) geometric patterns are associated with an increased risk of vascular complications of hypertension (HT). The association between red-cell distribution width (RDW) and HT has been recently investigated; however, the relationship between abnormal LV geometric patterns and RDW has not been studied before. The aim of this study was to investigate the relationship between RDW and abnormal LV geometric patterns in patients with untreated essential HT. Measurements were obtained from 139 patients with untreated essential HT (mean age = 51.3 ± 16.3 years). Four different geometric patterns (NG, normal geometry; CR, concentric remodeling; EH, eccentric hypertrophy; CH, concentric hypertrophy) were determined according to the LV mass index (LVMI) and relative wall thickness (RWT). RDW, lipid parameters and other biochemical markers were measured in all patients. While the highest RDW values were detected in the CH group ($P < 0.05$, for all), it was similar among the NG, CR and EH groups ($P > 0.05$, for all). RDW was associated with age, LVMI and LV geometry in bivariate analysis ($P < 0.05$, for all). In a receiver operating characteristic (ROC) curve analysis, a level of RDW > 14.5 predicted CH with 81% sensitivity and 59% specificity. Age (beta = 0.309, $P = 0.001$), LV geometry type (beta = 0.228, $P = 0.01$) and RWT (beta = -0.278 ; $P = 0.25$) were independent predictors of high RDW in multiple linear regression analysis. In conclusion, the highest RDW values were observed in the CH group in the untreated essential HT patients. RDW seems to be a useful tool for the prediction of end-organ damage in patients with untreated essential HT.

Hypertension Research (2014) 37, 560–564; doi:10.1038/hr.2014.39; published online 6 March 2014

Keywords: essential hypertension; left ventricle geometric patterns; left ventricle mass index; red-cell distribution width

INTRODUCTION

Hypertension (HT) is an important health problem that is associated with considerable cardiovascular (CV) morbidity and mortality related to end-organ damage. It should be adequately treated according to the specific risk factors and clinical conditions of the patient.^{1,2} Left ventricular hypertrophy (LVH) is an important example of end-organ damage in patients with HT.^{3,4} The heart may adapt to HT by developing concentric LVH (CH), eccentric LVH (EH), concentric left ventricular remodeling (CR) or by retaining a normal left ventricular geometry (NG).³ In previous studies it has been shown that increased left ventricular mass (LVM) and abnormal geometry have a strong predictive value for CV death, myocardial infarction and stroke.^{3,4}

Red-cell distribution width (RDW) is a quantitative measure of the variability in the size of circulating erythrocytes, and higher values reflect a more heterogeneous cell population.^{5,6} Increased RDW is also related to oxidative stress and the release of cytokines in response to inflammation.⁷ It has been consistently shown that RDW is closely related to the prognosis and long-term adverse events of CV diseases.⁸

Additionally, previous studies have shown that RDW levels are significantly increased in patients with HT.^{9–11} However, the relation between the RDW level and LV geometric patterns in patients with HT has not been investigated previously. The goal of this study was to investigate the relation between RDW and different LV geometric patterns in patients with untreated essential HT.

METHODS

Study population

Participants were recruited from the HT outpatient clinic at the Izmir Tepecik Research and Training Hospital between September 2012 and April 2013. A total of 139 patients (60 male, mean age = 51.1 ± 16.3 years) who met the criteria of being newly diagnosed with essential HT were enrolled in this cross-sectional study. The patients with HT had three clinical blood pressure (BP) measurements ($> 140/90$ mm Hg) taken at 1-week intervals in the absence of any previous antihypertensive treatment to exclude any pharmacological effects on hemodynamics or ventricular hypertrophy and function.¹² Echocardiography examinations including 2D, spectral and tissue Doppler measurements were performed, and blood samples were obtained from all

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Received 31 July 2013; revised 16 September 2013; accepted 12 November 2013; published online 6 March 2014

Table 1 Comparison of baseline, clinical and laboratory characteristics

Variables	NG group (n = 36)	CR group (n = 35)	EH group (n = 30)	CH group (n = 38)	f	P-value
AGE (year)	45.2 ± 17.6	54.7 ± 16.8	52.8 ± 16.4	52.0 ± 13.8	2.313	0.079
Gender (male)	15 (42%)	20 (57%)	14 (48%)	11 (29%)	2.117	0.101
SBP (mm Hg)	135.8 ± 10.5 ^a	138.8 ± 14.9	140.3 ± 9.9	145.9 ± 15.5	3.824	0.011
DBP (mm Hg)	84.1 ± 7.2 ^b	89.8 ± 10.7	89.1 ± 9.6	90.6 ± 11.6	3.197	0.026
Pulse (per min)	78.4 ± 9.7	79.3 ± 10.1	75.9 ± 10.8	78.1 ± 10.9	0.587	0.624
Glucose (mg dl ⁻¹)	93.1 ± 20.6	97.9 ± 22.1	103.7 ± 28.1	105.7 ± 42.1	1.111	0.348
BUN (mg dl ⁻¹)	28.2 ± 11.2	35.4 ± 13.8	32.8 ± 10.3	33.7 ± 16.7	1.587	0.197
Creatinine (mg dl ⁻¹)	0.91 ± 0.21	1.06 ± 0.48	0.98 ± 0.2	0.93 ± 0.25	1.288	0.282
TChol (mg dl ⁻¹)	214.1 ± 60.4	219.3 ± 51.4	196.0 ± 35.7	203.4 ± 49.2	0.988	0.402
TG (mg dl ⁻¹)	128.3 ± 56.3 ^c	172.0 ± 83.1	143.0 ± 67.5	169.8 ± 73.3	2.582	0.058
HDL-C (mg dl ⁻¹)	48.5 ± 10.5	44.4 ± 10.6	42.6 ± 15.6	43.4 ± 9.6	1.464	0.229
LDL-C (mg dl ⁻¹)	140.0 ± 53.4	140.5 ± 42.8	124.2 ± 30.2	126.1 ± 45.0	0.966	0.412
HBG (mg dl ⁻¹)	13.7 ± 1.6	13.9 ± 1.7	13.5 ± 1.6	13.1 ± 1.9	1.190	0.317
HTC (%)	40.5 ± 4.3	42.6 ± 8.8 ^d	39.6 ± 4.4	38.6 ± 5.1	2.469	0.066
WBC (K ul ⁻¹)	86.6 ± 3.8	88.8 ± 5.6	8.0 ± 2.9	7.9 ± 3.2	0.442	0.724
RDW (%)	13.7 ± 1.0	13.7 ± 1.1	13.7 ± 0.8	14.7 ± 1.5 ^e	5.225	0.002
Platelet (× 10 ⁹ l ⁻¹)	277.5 ± 75.2	247.8 ± 73.7	276.6 ± 51.6	256.0 ± 91.5	1.118	0.345
MPV (fL)	8.6 ± 1.2	8.6 ± 1.1	8.6 ± 0.7	8.6 ± 0.9	0.021	0.996

Abbreviations: BUN, blood urea nitrogen; CH, concentric hypertrophy; CR, concentric remodeling; DBP, diastolic blood pressure; EH, eccentric hypertrophy; f, f distribution; HBG, Hemoglobuline; HDL-C, high density lipoprotein cholesterol; HTC, Hemotocrite; LDL-C, low density lipoprotein cholesterol; MPV, mean platelet volume; NG, normal geometry; RDW, Red blood cell distribution width; SBP, systolic blood pressure; TChol, total cholesterol; TG, triglyceride; WBC, White blood cell.

^aP = 0.002 vs. CH group.

^bP = 0.005 vs. CH group. P = 0.01 vs. CR group. P = 0.02 vs. EH group.

^cP = 0.018 vs. CH group. P = 0.03 vs. CR group.

^dP = 0.04 vs. CH group.

^eP = 0.004 vs. EH group. P = 0.005 vs. NG group. P = 0.007 vs. CR group.

Figure 1 RDW levels along with left ventricle geometric patterns. A full color version of this figure is available at *Hypertension Research* online.

patients. Our Hospital Ethics Committee approved the study, and written informed consent for participation in the study was obtained from all of the participants. Patients with a history of chronic HT receiving appropriate antihypertensive medications, a history of secondary HT, pregnancy-induced HT and white coat HT, echocardiographic (left ventricle ejection fraction < 50%) and clinical (≥ New York Health Association class II) evidence of heart failure, a history of acute coronary syndrome, ischemic ECG findings, significant coronary artery disease in coronary angiography (> 50% luminal stenosis), organic valvular heart disease, chronic renal insufficiency (serum creatinine > 1.5 mg dl⁻¹ in men, > 1.3 mg dl⁻¹ in women) and a history of major noncardiovascular diseases such as autoimmune diseases, hematological diseases, cancer, thrombocytopenia and systemic inflammatory conditions were excluded from this study.

Anthromorphometry, BP measurement

After a 30-minute resting period, BP was measured twice in both arms while sitting using a mercury sphygmomanometer and ~ sized cuffs. The average of the two highest BP measurements was used to calculate systolic and diastolic BP. Patients with a systolic BP ≥ 140 mm Hg and/or a diastolic BP ≥ 90 mm Hg were considered to have HT.

Body mass index was computed as weight divided by height squared (kg m⁻²). The body surface area (BSA) was calculated using the Dubois formula of BSA = 71.84 W^{0.725} H^{0.725} where W is weight in kilograms and H is height in centimeters.¹³ Waist circumference (in centimeters) was measured from the midpoint between the lowest rib and the iliac crest, while the subject was standing.

Blood samples

Blood samples were drawn in the morning after a 20-minute rest following a fasting period of 12 h. Serum glucose, creatinine and lipid profiles were analyzed for each patient. RDW was measured from tripotassium ethylenediaminetetraacetic acid-based anticoagulated blood samples stored at + 4 °C and assessed by a Sysmex K-1000 autoanalyzer (Block Scientific, Bohemia, NY, USA) within 30 min of sampling.

Echocardiography

An echocardiographic examination was performed in all study subjects using a commercially available system (Vivid 7R GE Medical System, Horten, Norway) with a 2–3.5 MHz transducer. M-mode echocardiographic measurements were obtained on the basis of the standards of the American Society of Echocardiography.¹⁴ Left atrial diameter, left ventricular diameter in end diastole and end systole (LVEDD, LVESD), interventricular septal thickness and posterior wall thickness (PWT) were measured. LV ejection fraction was determined by the biplane Simpson's method.¹⁵ LVM (g) was calculated using the Devereux formula: LVM (g) = 0.8 × 1.04 × [(LviDD + IVS + PWT)³ – LviDD³] + 0.6.¹⁶ Then, the LV mass index (LVMI, g m⁻²) was obtained using the following formula: LVM/body surface area. LVH was defined according to more stringent criteria as LVMI values exceeded 125 g m⁻² in men and 110 g m⁻² in women.¹⁷ Relative wall thickness (RWT) was measured at end diastole as the ratio of (2 × PWT)/LVEDD. Increased

Table 2 Comparison of echocardiographic parameters

Variables	NG group (n = 36)	CR group (n = 35)	EH group (n = 30)	CH group (n = 38)	f	P-value
LVDD (mm)	44.6 ± 3.3 ^a	42.6 ± 3.3 ^b	49.8 ± 2.4 ^c	46.3 ± 3.5	24.92	<0.001
LVSD (mm)	26.1 ± 4.6 ^d	25.4 ± 4.4 ^e	30.4 ± 2.8 ^f	28.5 ± 3.7	9.05	<0.001
IVST (mm)	9.4 ± 0.8 ^g	10.9 ± 1.3	11.2 ± 0.9	12.6 ± 1.2 ^h	47.47	<0.001
PWT (mm)	9.2 ± 0.8 ⁱ	10.7 ± 1.1	10.7 ± 0.7	12.2 ± 1.2 ^j	51.95	<0.001
RVDD (mm)	24.5 ± 4.1	24.8 ± 3.5	27.2 ± 3.9 ^k	25.5 ± 3.3	2.53	0.06
LV MASS (g)	139.6 ± 29.1 ^l	159.9 ± 36.6 ^m	206.4 ± 29.1	218.2 ± 44.5	36.61	<0.001
LVMI (g m ⁻²)	79.6 ± 12.3 ⁿ	87.0 ± 15.6 ^o	113.7 ± 12.1 ^p	126.2 ± 20.1	67.82	<0.001
RWT (mm)	0.41 ± 0.03 ^q	0.50 ± 0.04 ^r	0.43 ± 0.02 ^s	0.53 ± 0.07	42.02	<0.001
LAD (mm)	35.9 ± 6.8	35.1 ± 6.2 ^t	38.2 ± 3.9	37.9 ± 4.4	2.39	0.07
LAVI (ml m ⁻²)	31.0 ± 0.7	28.1 ± 0.4	31.0 ± 0.6	30.1 ± 0.6	1.94	0.25
LVEF (%)	61.6 ± 3.5	60.8 ± 2.7	62.0 ± 4.4	60.5 ± 6.7	0.66	0.57
Assendan aorta (mm)	28.4 ± 4.0 ^u	30.2 ± 4.7	30.2 ± 4.3	31.3 ± 5.1	2.57	0.05
E/A	0.98 ± 0.1	1 ± 0.04	1 ± 0.08	0.95 ± .16	1.03	0.38
Dec T (ms)	266.8 ± 23.1	261.5 ± 20.3	260.7 ± 23.5	265.6 ± 35.1	0.26	0.84
e' (cm s ⁻¹)	0.1 ± 0.01	0.1 ± 0.01	0.11 ± 0.01	0.1 ± 0.01	0.89	0.45
a' (cm s ⁻¹)	0.1 ± 0.01	0.11 ± 0.01	0.11 ± 0.01	0.11 ± 0.01	0.49	0.68
Sm' (cm s ⁻¹)	0.11 ± 0.01	0.11 ± 0.01	0.1 ± 0.01	0.1 ± 0.01	0.83	0.48
E/e'	6.8 ± 0.8	6.7 ± 0.8	6.3 ± 0.6	6.4 ± 0.7	1.48	0.22
e'/a'	1 ± 0.1	0.99 ± 0.1	1.01 ± 0.1	0.96 ± 0.1	0.517	0.67

Abbreviations: A, Mitral late diastolic velocity; a', Mitral annular late diastolic velocity; CH, concentric hypertrophy; CR, concentric remodeling; Dec T, deceleration time; E, Mitral early diastolic velocity; e', Mitral annular early diastolic velocity; EH, eccentric hypertrophy; f, f distribution; IVST, interventricular septal thickness; LAD, Left atrial diameter; LAVI, Left atrial volume indices; LVDD, left ventricle diastolic diameter; LVEF, Left ventricle ejection fraction; LV MASS, Left ventricle mass; LVMI, left ventricular mass index; LVSD, Left ventricle systolic diameter; NG, normal geometry; PWT, posterior wall thickness; RVDD, Right ventricle diastolic diameter; RWT, relative wall thickness; Sm', Mitral annular systolic velocity.

^aP<0.001 vs. EH and CR groups. P=0.03 vs. CH group.

^bP<0.001 vs. CH and EH groups.

^cP<0.001 vs. CH group.

^dP<0.001 vs. EH group. P=0.02 vs. CH group.

^eP<0.001 vs. EH and CR groups.

^fP<0.03 vs. CH group.

^gP<0.001 vs. EH, CR and CH groups.

^hP<0.001 vs. EH and CR groups.

ⁱP<0.001 vs. CH, EH and CR groups.

^jP<0.001 vs. EH, CH groups.

^kP=0.02 vs. EH. P=0.03 vs. CR group.

^lP<0.001 vs. EH and CH groups. P=0.01 vs. CR group.

^mP<0.001 vs. EH and CH groups.

ⁿP<0.001 vs. EH and CH groups. P=0.03 vs. CR group.

^oP<0.001 vs. EH and CH groups.

^pP=0.008 vs. CH group.

^qP<0.001 vs. CR and CH groups. P=0.04 vs. EH group.

^rP<0.001 vs. EH group. P=0.04 vs. CH group.

^sP<0.001 vs. CH group.

^tP=0.03 vs. NG group.

^uP<0.08 vs. CH group.

Table 3 Bivariate relationships of red-cell distribution width

Variables	Pearson correlation coefficient	P-value
AGE (year)	0.293	0.001
Systolic BP (mm Hg)	-0.01	0.911
Diastolic BP (mm Hg)	-0.106	0.256
IVST (mm)	0.199	0.036
LVMI (g m ⁻²)	0.189	0.047
RWT (mm)	0.06	0.532
Hypertrophy type	0.275	0.003
Dec T (ms)	0.133	0.257
E/A	-0.177	0.131
E/e'	0.194	0.098
e'/a'	-0.193	0.1

Abbreviations: A, Mitral late diastolic velocity; a', Mitral annular late diastolic velocity; BP, Blood Pressure; Dec T, Deceleration time; E, Mitral early diastolic velocity; e', Mitral annular early diastolic velocity; HBG, Hemoglobuline; HTC, Hemotocrite; IVST, interventricular septal thickness; LVMI, left ventricular mass index; RWT, relative wall thickness.

RWT was defined as >0.45.¹⁸ The left atrial volume was calculated using the area-length method. Using this method, the area of the left atrium was measured by planimetry of both apical views (A1 and A2). A linear dimension was measured from the center of the mitral annulus to the superior border of the chamber (litre). The left atrial volume was then calculated as [(0.85 × A1 × A2) ÷ l].^{19,28} The left atrial volume index was calculated by dividing the left atrial volume by the body surface area.

All of the echocardiography measurements were repeated in a second observer who was blinded to the values obtained from the first observer. Inter-observer variability was assessed by calculating the coefficient of variation. The coefficient of variation was <8% for all measurements. Any discrepancy was resolved by consensus. All echocardiography measurements were repeated 1 week later in an observer blinded to the results of the previous measurements; intra-observer variability was <5% for all measurements.

Patterns of left ventricular geometry

Geometric patterns were based on the upper normal limits for LVMI and RWT: (I) NG (normal LVMI and normal RWT); (II) CR (normal LVMI and

increased RWT); (III) CH (increased LVMI and increased RWT) and (IV) EH (increased LVMI and normal RWT).¹⁸

Statistical analysis

Statistical analyses were performed using SPSS for Windows version 20.0 (SPSS, Chicago, IL, USA). The comparison of categorical variables between the groups was performed using the χ^2 -test. The Kolmogorov-Smirnov test was performed to evaluate the normality of distribution of all continuous variables. Analysis of variance was used in the analysis of continuous variables. Stratified *post-hoc* analyses of echocardiographic, clinical and laboratory variables were performed according to the LV geometric patterns. The correlations between RDW and laboratory, hemodynamic and echocardiographic parameters were assessed by the Pearson correlation test. To avoid over-fitting and collinearity in assessing the multivariate model, independent variables have been tested for inter-correlation. The cutoff value of the RDW for predicting the LV geometric pattern of CH with corresponding sensitivity and specificity was assessed using the receiver operating characteristic (ROC) curve analysis. The stepwise multiple linear regression analysis was performed to identify the independent associations of RDW. A two-tailed $P < 0.05$ was considered to be statistically significant.

RESULTS

Four different geometric patterns were determined according to the LVMI and RWT; (1) 36 patients with NG, (2) 35 patients with CR, (3) 30 patients with EH and (4) 38 patients with CH. Comparisons of baseline, clinical and laboratory characteristics are shown in Table 1. In comparison with the other groups, the highest RDW values were determined to be in the CH group ($P < 0.01$, for all) (Figure 1). The RDW values were similar among the NG, CR and EH groups. A comparison of the echocardiography parameters is illustrated in Table 2. LVMI and RWT values were highest in the CH group, followed by the EH, NG and CR groups, respectively ($P < 0.05$, for all). The prevalence of abnormal diastolic function showed nonsignificant stepwise increase in the CH group. We compared the

effects of dipping and nondipping patterns on RDW and no significant difference was detected (13.9 ± 1.1 , 14 ± 1.3 ; $P = 0.45$).

RDW was significantly correlated with age ($r = 0.293$, $P < 0.01$), LVMI ($r = 0.189$, $P = 0.04$) and interventricular septal thickness ($r = 0.199$, $P = 0.03$) (Table 3). It was observed that age ($\beta = 0.309$, $P < 0.001$), RWT ($\beta = -0.278$, $P = 0.02$) and type of hypertrophy ($\beta = 0.408$, $P < 0.001$) were independent predictors of increased RDW value.

In receiver operating characteristic curve analysis, a level of RDW > 14.5 predicted CH with 81% sensitivity and 59% specificity, and its area under curve: 0.636, 95% CI = 0.518–0.755, $P = 0.02$ (Figure 2).

DISCUSSION

The present study provides the first data on the relationship between RDW and abnormal LV geometric patterns in patients with untreated essential HT independent of anemia. We demonstrated that RDW increased in the CH group but was similar in the NG, CR and EH groups. In addition, we demonstrated a significant stepwise increase in LVMI and RWT within the CH group.

Arterial HT is associated with a spectrum of cardiac geometric adaptations matched to systemic hemodynamic and ventricular load.²⁰ LVH is an important example of end-organ damage in patients with HT.³ Increased LVM predicts a greater risk of CV events, independent of other risk factors.^{15,21} The LV may adapt to HT by developing CH, EH, CR or by retaining NG.²² Each geometric pattern is associated with a distinct combination of pressure and volume stimuli, contractile efficiency (reduced in those with CH or CR) and prognosis. CR and EH are more common than the typical pattern of CH in patients with untreated HT.²³ Our results were comparable with previous data. In our study, LVMI and RWT were increased in the CH group. According to the previous data, subjects with CH had the worst prognosis, followed by those with EH, CR and NG in a population-based sample of subjects without CV disease.^{3,24}

Increased RDW is also related to oxidative stress and the release of cytokines in response to inflammation. Oxidative stress directly damages erythrocytes and causes shortening of erythrocyte survival, thereby resulting in the elevation of RDW.⁷ It has been consistently shown that RDW is closely related to the prognosis and long-term adverse events of CV diseases and heart failure.^{8,25,26} It was also previously shown that an increased RDW value was related to HT.^{10,11} Tanindi *et al.*⁹ demonstrated that RDW was higher in patients with prehypertension and HT compared with healthy controls independent of age, inflammatory status and anemia. The relationship between RDW and LV geometric patterns has not been studied in patients with untreated essential HT before. In our study, we demonstrated that RDW was increased in the CH group but was similar in the NG, CR and EH groups. RDW was independently associated with types of LVH and RWT. Unlike other studies, we demonstrated that RWT was related to concentric hypertrophy regardless of the BP level. There may be several possible explanations for the relation between red blood cells and LVH in HT. One tentative explanation is that red-cell Li^+/Na^+ exchange is increased in patients with essential HT. High Li^+/Na^+ counter transport reflects the abnormal kinetic properties of red-cell membrane Na/H exchange. A widespread abnormality of Na/H exchange could have a major role in the pathogenesis of CV diseases. Na/H exchange is involved in the regulation of cell pH and cell volume in the cellular responses to hormones, mitogens and growth factors and in the renal reabsorption of Na and bicarbonate.²⁷ Increased Li^+/Na^+ exchange has been found with cardiac hypertrophy in resistant HT.²⁸

Figure 2 Receiver operating characteristic (ROC) curve analysis of the RDW predicting the concentric hypertrophy in patients with untreated essential hypertension. A full color version of this figure is available at *Hypertension Research* online.

Previously, it has been shown that increased RDW was related to echocardiographic parameters in patients with diastolic heart failure.²⁹ However, in our study abnormal diastolic functions showed a stepwise increase in the CH group but this change did not reach statistical significance; we also did not find any relationship between RDW and diastolic function. LV diastolic functions are known to be deteriorated in hypertensive patients whether their LV mass is increased or normal.³⁰ Therefore, the reason for finding no significant differences between the groups may be owing to the deterioration of diastolic functions in all of the patients because every patient in our study group had untreated essential HT.

Limitation

The small number of participants and lack of a control group are the main limitations of this study. This is a cross-sectional study, meaning it is evident whether higher RDW levels in patients with CH is a cause or a result. Larger prospective trials should be performed to explore this issue. We already know that RDW has been shown to be an inflammatory marker that is closely related to the prognosis and long-term adverse events of CV diseases.^{6,7} However, the lack of assessment of inflammatory markers such as hs-CRP and their relation to RDW is another limitation of the current study. Finally, LV mass and ejection fraction were calculated based on M-mode measurements. It is possible that three-dimensional ultrasound recordings or myocardial MRIs may give more appropriate results.

In conclusion, in patients with untreated essential HT, the highest RDW values were observed in the CH group when compared with other geometric patterns independent of anemia. RDW could be a predictor of end-organ damage in these patients. Further prospective studies with larger sample sizes are needed to shed light on the mechanisms underlying this association.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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