# Impact of Chronic Kidney Disease on Left Ventricular and Left Atrial Deformation in Patients with Anterior Myocardial Infarction as Detected by 2D -Speckle Tracking

Hany H. Ebaid, Nagy E. Antar, Khaled E. Elrabat, Karim Elakabawi

Department of cardiovascular medicine, Benha faculty of medicine, Benha University, Egypt.

MEDICAL JOURNAL

**Correspondence to:** Nagy E. Antar, Department of cardiovascular medicine, Benha faculty of medicine, Benha University, Egypt.

#### Email:

nagyelsebaye86@gmail.com

Received:

Accepted:

#### Abstract

Background: Chronic kidney disease (CKD) and coronary artery disease (CAD) represent major causes of morbidity and mortality worldwide. The aim of the current study is to investigate the value of 2D-STE in assessing the abnormalities in LV and LA function after anterior MI in patients with and without CKD. Methods: This prospective, single-centre cohort study was conducted on 200 patients aged more than 18 years diagnosed with anterior wall myocardial infarction who were admitted to the coronary care unit at Benha University Hospital in the duration from February 2023 to January 2024. The study included 100 consecutive cases of anterior STEMI and mild to moderate (stages 2-3) CKD and were compared to 100 age and sex-matched cases of anterior STEMI with normal GFR ( $\geq 90$ ml/min/1.73m<sup>2</sup>). Results: Regarding the echocardiographic findings, EF, LV-GLS, LASr, LAScd, LASct, and TAPSE were significantly lower in patients with CKD (P<0.05), while LA vol and LAVI were significantly higher, with no significant difference between both groups regarding LVEDD and LVESD. There was a significant positive correlation between eGFR and EF (r= 0.507, P<0.001), LASr (r=0.547, P<0.001) and TAPSE

(r=0.377, P<0.001). There was a significant negative correlation between eGFR and LA  $_{Vol}$  (r= -0.409, P<0.001), LAVI (r=-0.379, P<0.001) and LV-GLS (r=-0.396, P<0.001), LAScd (r=-0.581, P<0.001) and LASct (r=-0.370, P<0.001).

**Conclusions:** STEMI patients with mild to moderate CKD had higher diuretics administration, CRP, troponin levels, and serum creatinine, while it has lower HR, SBP, DBP, and eGFR with prolonged hospitalization compared to STEMI patients without CKD.

High level of serum creatinine was associated with increased LA Vol, LV-GLS and LAScd and decreased EF, LVEDD and LASr. While high level of eGFR was associated with increased EF, LASr and TAPSE and decreased LA Vol, LAVI and LV-GLS, LAScd and LASct.

**Keywords:** Chronic Kidney Disease, Left Ventricular, Left Atrial Deformation Anterior Myocardial Infarction, 2D -Speckle Tracking.

## Introduction

CKD and coronary artery disease (CAD) represent major causes of morbidity and mortality worldwide, and the combination particularly of both diseases is unfavourable in terms of prognosis <sup>[1]</sup>. Besides that, many shreds of evidence show that renal function impairment indicated by reduced estimated glomerular filtration rate has a significant impact on the incidence and prognosis of CAD and outcomes after acute myocardial infarction affecting (AMI), success and complications of reperfusion therapy, the occurrence of heart failure, and mortality rates<sup>[2]</sup>.

This risk becomes evident as the GFR decreases below  $60 \text{ml/min}/1.73 \text{m}^2$ ; however, inconsistent results have been reported for the association between mild to moderate CKD and CV risk <sup>[3]</sup>.

Many studies showed that assessing left ventricular function using modalities like global longitudinal strain analysis that describe the cycle deformation (shortening or lengthening) of the myocardium allows better information about LV dysfunction than left ventricular ejection fraction and allows additional prognostic information <sup>[4]</sup>.

Therefore, the aim of the current study is to investigate the value of 2D-STE in assessing the abnormalities in LV and LA function after anterior MI in patients with and without CKD.

## **Patients and Methods:**

This observational single-centre cohort study was conducted on 200 patients aged more than 18 years diagnosed with anterior wall myocardial infarction who were admitted to the coronary care unit at Benha University Hospital in the duration from February 2023 to January 2024. An informed written consent was obtained from the patient or relatives of the patients. The study was done after approval from the Ethical Committee Tanta University Hospitals (MS 23-4-2023)

Inclusion criteria were age more than 18 years, both sexes, patients who were diagnosed with anterior myocardial infarction on the basis of typical ischemic: chest pain enduring for  $\geq 20$  minutes with electrocardiographic (ECG) changes (STsegment elevations of  $\geq 1$ mm in  $\geq 2$ contiguous precordial leads other than V2,3 or new onset left bundle branch block) with a rise in cardiac biomarkers, including troponins and CKMB<sup>[5]</sup>.

Exclusion criteria were patients with severe CKD (stages 4-5), patients with cardiogenic shock or mechanically ventilated, patients refused to participate or lost in follow-up, patient not received thrombolytic or underwent percutaneous coronary intervention (PCI).

The study was conducted on 100 consecutive cases of anterior STEMI with mild to moderate (stages 2-3) CKD, **Group I.** They were compared to 100 age and sex-matched patients with normal GFR ( $\geq$ 90ml/min/1.73m<sup>2</sup>), **Group II**.

Chronic kidney disease (CKD) is defined as the presence of an abnormality in kidney structure or function persisting for more than 3 months. This includes 1 or more of the following: GFR less than 60 mL/min/1.73 m2, albuminuria (i.e. urine albumin  $\geq$ 30 mg per 24 hours or urine albumin-to-creatinine ratio [ACR]  $\geq$ 30 mg/g), abnormalities in urine sediment, histology, or imaging suggestive of kidney damage, renal tubular disorders, and history of kidney transplantation. The stages of CKD related to GFR: Stage 1  $\geq$ 90 GFR normal kidney, Stage 2 60–89 GFR mild dysfunction, Stage 3a 45–59 GFR mild to moderate dysfunction, Stage 3b 30–44 GFR moderate to severe dysfunction, Stage 4 15–29 GFR severe dysfunction, and Stage 5 <15 GFR kidney failure <sup>[6]</sup>.

All patients were subjected to history taking, complete examination included general and local examination, and full laboratory investigations. The in-hospital management of the study population was also evaluated, including medications used during hospitalization, reperfusion method, comprehensive echocardiographic examination, and in-hospital clinical outcomes. Primary PCI was the preferred reperfusion strategy. However, when primary PCI was not technically feasible, we used thrombolytic therapy with planned rescue PCI in case of failed thrombolysis.

The echocardiographic examinations were performed by two experienced Echocardiographers using a system (EPIQ 7C; Philips) equipped with a broadband S5-1 transducer. Two-dimensional, Mmode and Doppler echocardiography were acquired according to the guidelines of the American Society of Echocardiography (ASE) and stored digitally on magnetooptical discs and on an EchoPAC server (Image Vault 5.0 system; General Electric Company, Horten, Norway). LAS values were measured for all the patients as recommended bv the "EACVI/ASE/Industry Task Force" in apical 4-and 2-chamber views and were reported separately for the three phases of LA cycle: reservoir, conduit. and contraction phase: LASr = strain duringreservoir phase, measured as the strain value from the ventricular end-diastole to the mitral valve opening at ventricular end-systole (positive value), LAScd = strain during conduit phase, measured as the strain value from the mitral valve opening to the onset of atrial contraction (negative value). In patients with atrial fibrillation, LAScd has the same value as LASr, but with a negative sign, and LASct = strain during contraction phase, measured only in patients in sinus rhythm as the strain value from the onset of atrial contraction to ventricular end-diastole (negative value).

## Statistical analysis:

Statistical analysis was done by SPSS v28 (IBM©, Armonk, NY, USA). Shapiro-Wilks test and histograms were used to evaluate the normality of the distribution of data.

Quantitative parametric data were presented as mean and standard deviation (SD) and were analyzed by unpaired student t-test. Quantitative non-parametric data were presented as the median and interquartile range (IQR) and were analyzed by Mann Whitney-test.

Qualitative variables were presented as frequency and percentage (%) and analyzed using the Chi-square test or Fisher's exact test when appropriate. Pearson correlation was performed to estimate the degree of correlation between two quantitative variables. Multiple regression was used to analyze the relationship between a single dependent variable and several independent variables. Receiver operating characteristic curve (ROC-curve) was conducted to analyze the cut-off values for the parameters that independently predicted for eGFR<45. A two-tailed P value < 0.05 was considered statistically significant.

#### **Results:**

Baseline characteristics (age, sex, weight, height, and BMI) were insignificantly different between both groups, there was insignificant difference between both groups regarding the risk factors including (smoking, HTN, DM, dyslipidemia, IHD, and CVD). **Table 1** 

Regarding medications, administration of diuretics was significantly higher in group I compared to group II (P=0.042). Other medication including Amiodarone, OAC, BB, ACE/ARB, ASA, statin and P2Y12 were insignificantly different between both groups). HR, SBP were significantly lower in group I compared to group III (P=0.025, <0.001, <0.001). DBP was insignificantly different between both groups. CRP and troponin levels were significantly higher in group I compared to group II (P<0.001, <0.001). There was a significant difference between both groups regrading Killip class (P<0.001), the total duration of hospitalization (P<0.001), with no significant difference between both

groups regarding Hb, PLT and WBCs. **Table 2** 

Regarding the echocardiographic findings, EF, LV-GLS, LASr, LAScd, LASct, and TAPSE were significantly lower in group I compared to group II (P<0.05), while LAVol and LAVI were significantly higher in group I compared to group II (P<0.05), with no significant difference between both groups regarding LVEDD and LVESD. **Table 3** 

There was a significant positive correlation between eGFR and EF (r= 0.507, P<0.001), LASr (r=0.547, P<0.001) and TAPSE (r=0.377, P<0.001). There was a significant negative correlation between eGFR and LA <sub>Vol</sub> (r= -0.409, P<0.001), LAVI (r=-0.379, P<0.001) and LV-GLS (r=-0.396, P<0.001), LAScd (r=-0.581, P<0.001) and LASct (r=-0.370, P<0.001). There was an insignificant correlation between eGFR and LVEDD. **Table 4** 

The multiple regression analysis revealed that among the echocardiographic parameters, EF, LA <sub>Vol</sub>, LASr, LAScd and TAPSE were the only significant predictors for reduced eGFR. **Table 5** 

LAScd was a significant predictor for eGFR<45 with sensitivity of 80%., 62.16% specificity, 228.7% PPV and 98.1% NPV. LASr was a significant predictor for eGFR<45 with 93.33% sensitivity, 83.51% Specificity, 60.0% PPV, 98.3% NPV. **Figure 1** 

		Total (n=200)	Group I (n=100)	Group II (n=100)	P value	
Age (	years)	$64.5\pm7.33$	$65.5\pm7.75$	$64.4\pm6.77$	0.128	
Sex	Male	159 (79.5%)	84 (84%)	75 (75%)	0.115	
	Female	41 (20.5%)	16 (16%)	25 (25%)		
BMI (	Kg/m <sup>2</sup> )	$28.97 \pm 4.43$	$29.2\pm4.58$	$28.7\pm4.28$	0.392	
Smo	oking	75 (37.5%)	42 (42%)	33 (33%)	0.188	
H	TN	109 (54.5%)	57 (57%)	52 (52%)	0.478	
D	M	71 (35.5%)	40 (40%)	31 (31%)	0.183	
Dyslip	oidemia	99 (49.5%)	52 (52%)	47 (47%)	0.479	
II	łD	27 (13.5%)	16 (16%)	11 (11%)	0.301	
C	VD	12 (6.0%)	7 (7%)	5 (5%)	0.552	

Table 1: Baseline characteristics of the studied groups

BMI: body mass index, HTN: hypertension, DM: diabetes mellitus, CVD: coronary vascular disease. \*: statistically significant as p value <0.05

	Total(n=200)	Group I (n=100)	Group II (n=100)	P value	
Amiodarone	48 (24%)	26 (26%)	22 (22%)	0.508	
OAC	51 (25.5%)	31 (31%)	20 (20%)	0.074	
BB	132 (66%)	61 (61%)	71 (71%)	0.136	
ACE/ARB	152 (76%)	72 (72%)	80 (80%)	0.185	
Diuretics	78 (39%)	46 (46%)	32 (32%)	0.042*	
ASA	192 (96.0%)	94 (94%)	98 (98%)	0.279	
Statin	192 (96.0%)	95 (95%)	97 (97%)	0.279	
HR (beat/min)	$83.1\pm7.6$	$81.9\pm7.81$	$84.3\pm7.22$	0.025*	
SBP (mmHg)	$106.9 \pm 15.5$	$100.1\pm11.5$	$113.9 \pm 16.03$	<0.001*	
DBP (mmHg)	$77.8 \pm 10.76$	$68.75 \pm 11.41$	$69.51 \pm 12.15$	0.649	
Killip class 1	126 (63%)	51 (51%)	75 (75%)	<0.001*	
Hb (g/dL)	$12.8 \pm 1.03$	$12.7 \pm 1.05$	$12.9 \pm 1.01$	0.163	
PLT (*10 <sup>9</sup> /L)	$247.7\pm43.5$	$247.6\pm42.5$	$247.9 \pm 44.7$	0.961	
WBCs (*10 <sup>9</sup> /L)	$7.00 \pm 1.44$	$6.98 \pm 1.48$	$7.01 \pm 1.41$	0.861	
	101	143		0.001	
CRP (mg/L)	(25.7 – 184)	(87 - 173.5)	31 (19 – 184)	<0.001*	
	92	225	75	<0.001*	
Troponin (pg/mL)	(54.5 - 472)	(59.5 - 767.5)	(52.25 - 100)		
Hospitalization (days)	5 (4 – 8)	8 (5-16)	4 (4-5)	<0.001*	

Table 2: Medications and clinical examination of the studied groups

HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, Hb: hemoglobin, PLT: platelets, WBCs: white blood cells, CRP: C - reactive protein, \*: statistically significant as p value <0.05.

	Total (n=200)	Group I (n=100)	Group II (n=100)	P value
<b>EF (%)</b>	$48.9 \pm 5.59$	$45.3\pm3.98$	$52.5\pm4.57$	<0.001*
LVEDD (mm)	$48.98 \pm 4.94$	$48.4\pm4.86$	$49.5\pm4.99$	0.113
LVESD (mm)	$34.7\pm5.25$	$35.3\pm6.12$	$33.99 \pm 4.13$	0.071
LA <sub>Vol</sub> (mL)	$55.4 \pm 16.38$	$62.1 \pm 18.74$	$48.7\pm9.88$	<0.001*
LAVI (mL/m <sup>2</sup> )	$28.4\pm8.12$	$32.1 \pm 8.64$	$24.7\pm5.45$	<0.001*
LV-GLS (%)	$-16.6 \pm 2.97$	$-15.0 \pm 2.5$	$-18.2 \pm 2.53$	<0.001*
LASr (%)	$28.4\pm7.18$	$23.4 \pm 5.78$	$33.4 \pm 4.43$	<0.001*
LAScd (%)	$-15.6 \pm 4.84$	$-12.01 \pm 3.83$	$-19.2 \pm 2.5$	<0.001*
LASct (%)	$-12.9 \pm 3.51$	$-11.03 \pm 3.68$	$-14.8 \pm 2.04$	<0.001*
TAPSE (mm)	$1.8\pm0.26$	$1.6 \pm 0.24$	$1.9 \pm 0.21$	<0.001*

Table 3: Echocardiography of the studied groups

EF: ejection fraction, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, LA Vol: left atrial volume, LAVI: Left Atrial Volume Index, LV-GLS, left ventricular global longitudinal strain, LASr, left atrial strain during reservoir phase, LAScd: left atrial strain during conduit phase, LASct: left atrial strain during contraction phase, TAPSE: tricuspid annular plane systolic excursion, \*: statistically significant as p value <0.05.

Table 4: Correlation between eGFR and echocardiographic parameters
--------------------------------------------------------------------

	eGFR (ml/min/1.73 m <sup>2</sup> )		
	r	Р	
EF (%)	0.507	<0.001*	
LVEDD (mm)	0.057	0.427	
LVESD (mm)	-0.078	0.270	
LA <sub>Vol</sub> (mL)	-0.409	<0.001*	
LAVI (mL/m <sup>2</sup> )	-0.379	<0.001*	
LV-GLS (%)	-0.396	<0.001*	
LASr (%)	0.547	<0.001*	
LAScd (%)	-0.581	<0.001*	
LASct (%)	-0.370	<0.001*	
TAPSE (mm)	0.377	<0.001*	

r: correlation coefficient, eGFR: estimated glomerular filtration rate, EF: ejection fraction, LVEDD: Left ventricular enddiastolic diameter, LVESD: Left ventricular end-systolic diameter, LV-GLS, left ventricular global longitudinal strain, LASr, left atrial strain during reservoir phase, LAScd: left atrial strain during conduit phase, LASct: left atrial strain during contraction phase, TAPSE: tricuspid annular plane systolic excursion, \*: statistically significant as p value <0.05.

	Coefficient	SE	t	Р	r <sub>partial</sub>	r <sub>semipartial</sub>
EF (%)	0.778	0.206	3.771	<0.001*	0.265	0.195
LVEDD (mm)	0.004	0.187	0.024	0.981	0.002	0.001
LVESD (mm)	0.291	0.188	1.546	0.124	0.112	0.080
LA vol (mL)	-0.215	0.060	-3.587	<0.001*	-0.253	0.186
LAVI (mL/m <sup>2</sup> )	-0.237	0.124	-1.919	0.057	-0.138	0.099
LV-GLS (%)	-0.611	0.354	-1.726	0.086	-0.125	0.089
LASr (%)	0.630	0.155	4.079	<0.001*	0.285	0.211
LAScd (%)	-0.766	0.253	-3.028	0.003*	-0.216	0.153
LASct (%)	-0.208	0.293	-0.711	0.478	-0.052	0.037
TAPSE (mm)	8.649	3.917	2.208	0.028*	0.159	0.114

**Table 5:** Multiple regression analysis of echocardiographic parameters for prediction of reduced eGFR

eGFR: estimated glomerular filtration rate, EF: ejection fraction, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, LV-GLS, left ventricular global longitudinal strain, LASr, left atrial strain during reservoir phase, LAScd: left atrial strain during conduit phase, LASct: left atrial strain during contraction phase, TAPSE: tricuspid annular plane systolic excursion, SE: standard error, \*: statistically significant as p value <0.05.

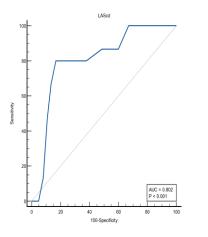


Figure 1A:ROC analysis of LAScd for prediction of eGFR<45

#### 100-80-60-40-0-20-0-20-40-0-20-40-0-20-40-0-0-0-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-100-

LAS

Figure 1B: ROC analysis of LASr for prediction of eGFR<45

#### Discussion

Left ventricular hypertrophy, dilatation, and dysfunction are the most common cardiac abnormalities in CKD patients. Previous studies found that left ventricular hypertrophy was the first prominent cardiac impairment due to a consistently high level of plasma urea, which is progressively more severe across CDK stages, earlier than dilatation and dysfunction<sup>[7]</sup>. We found that CRP and troponin levels were significantly higher in group I compared to group II (P<0.001, <0.001), with no significant difference between both groups regarding Hb, PLT and WBCs.

On the other hand, Krishnasamy et al., <sup>[8]</sup>, stated that patients with renal impairment had lower Hb levels compared with those with eGFR  $\geq 60$  mL/min/1.73 m<sup>2</sup>.

However, Nguyen et al., <sup>[9]</sup> noted that lower hemoglobin (g/L) and RBC results (T/L) (p<0.001) were related to more severe stages, this difference between our findings and the previous study may be due to their different comparison from our study.

In the present study, serum creatinine before and after was significantly higher in group I compared to group II (P<0.001, <0.001), while eGFR before and after was significantly lower in group I compared to group II (P<0.001, <0.001). In both groups, serum creatinine after was significantly increased compared to before. In group I, eGFR after was significantly decreased compared to before (P<0.001), while in group II, eGFR after was significantly increased compared to before (P<0.001).

This previous finding was confirmed by Nguyen et al., <sup>[9]</sup> who stated that there was a statistical association between more severe stages of CKD and lower GFR (p<0.001), and a higher concentration of creatinine (mmol/L) (p<0.001).

Regarding the echocardiographic findings, EF, LV-GLS, LASr, LAScd, LASct, and TAPSE were significantly lower in group I compared to group II (P<0.05), while LA Vol and LAVI were significantly higher in group I compared to group II (P<0.05), with no significant difference between both groups regarding LVEDD and LVESD. There was a significant difference between both groups regarding Killip class (P<0.001). The hospitalization was significantly prolonged in group I compared to group II (P<0.001). The atrial fibrillation incidence was insignificantly different between both groups.

All our previous findings were partially confirmed by Wang et al., [10] who assessed the risk factors for acute kidney injury in patients with acute myocardial infarction. Their logistic regression analysis showed that the independent risk factors for AKI in patients with AMI included (>60 years old). age hypertension, chronic kidney disease, Killip class ≥3, extensive anterior myocardial infarction, use of furosemide, and non-use of ACEIs/ARBs. These factors provided an accurate tool to identify patients at high risk of developing AKI. However, they focused on AKI, whereas we focused on CKD; despite that, our results were almost similar.

We observed that there was a significant positive correlation between serum creatinine and LA Vol, LV-GLS, and LAScd. There was a significant negative correlation between serum creatinine and EF, LVEDD, and LASr. There was an insignificant correlation between serum creatinine and LVESD, LAVI, LASct, and TAPSE.

In agreement, Nguyen et al., <sup>[9]</sup> illustrated a significant reduction in the LA reservoir strain (LASr; %) (P<0.001), while a remarkable increase (less negative) was observed in the LA conduit strain (LAScd; %) (P<0.001) among different CKD groups. The alteration of LASct across CKD stages was not significant (p=0.2144).

In our study, there was a significant positive correlation between eGFR and EF, LASr, and TAPSE. There was a significant negative correlation between eGFR and LA Vol, LAVI, LV-GLS, LAScd, and LASct. There was an insignificant correlation between eGFR and LVEDD.

LV-GLS association was supported by Krishnasamy et al., <sup>[8]</sup> who concluded that GLS is an important predictor of all-cause mortality in CKD patients. Traditional and non-traditional risk factors such as phosphate are important determinants of GLS. Strain assessment in CKD patients may provide greater cardiovascular risk stratification.

In the present study, multiple regression analysis revealed that among the echocardiographic parameters, EF, LA Vol, LASr, LAScd, and TAPSE were the only significant predictors for reduced eGFR. We found that LA speckle is a rapid, easy tool that can predict patients who will develop moderately severe renal impairment after STEMI and who need close follow-up of renal functions.

In alignment with our results, Nakanishi et al., <sup>[11]</sup>found that there was no significant difference in LAVImax between the CKD and non-CKD groups, whereas significant differences were observed for LAVImin and LAEF. Multivariate regression analysis revealed that eGFR was significantly associated with LAEF independent of age, LV mass index, and diastolic dysfunction (all p<0.05).

Our results regarding LV parameters, especially LV ejection fraction, were confirmed by Hensen et al., <sup>[12]</sup>, who investigated the prognostic implications of LV GLS in predialysis and dialysis patients. They noted that LV GLS and LV fraction ejection were significantly associated with all-cause mortality together with age, male gender, albumin fibrillation, levels. atrial and renal transplantation. LV GLS and LV ejection fraction were independently associated with all-cause mortality after correcting for age, male gender, albumin levels, atrial fibrillation, and renal transplantation.

Paoletti and Zoccali<sup>[13]</sup> also found that LA has the potential to be elected as a surrogate endpoint in CKD patients, but the issue remains to be tested in specifically designed clinical studies.

Our study hypothesis and results were confirmed by Essig et al., <sup>[14]</sup>, who investigated cardiovascular remodeling and extracellular fluid excess in early stages of chronic kidney disease. They concluded that CV remodeling and extracellular fluid (ECF) excess occurred at a very early stage of CKD. The independent association between ECF excess and cardiac and vascular remodeling and hypertrophy may be instrumental in the increased cardiovascular risk in CKD patients. Early therapeutic control of ECF may reduce CV events in CKD patients.

In contrast, Krishnasamy et al., <sup>[15]</sup> showed very different results from ours. They

investigated the prognostic value of GLS over EF in patients with advanced chronic kidney disease and showed that the allcause and CV mortality rates were substantially higher for patients with impaired GLS compared to preserve GLS. They further concluded that, in patients with advanced CKD, GLS is a more sensitive predictor of overall and CV mortality compared to EF, which differs from our study findings.

Our present study had limitations such as a relatively small sample size and a short follow-up period, which may contribute to insignificant results. We need more variable measurements and more statistical analysis methods, such as diagnostic analysis and measuring all-mortality associated risk factors.

#### Conclusions

STEMI patients with mild to moderate CKD had higher diuretics administration, CRP, troponin levels, and serum creatinine, while it has lower HR, SBP, DBP. and eGFR with prolonged compared hospitalization **STEMI** to patients without CKD. EF, LA Vol, LASr, LAScd and TAPSE can be used as predictors for reduced eGFR in STEMI patients with mild to moderate CKD.

High level of serum creatinine was associated with increased LA Vol, LV-GLS and LAScd and decreased EF, LVEDD and LASr. While high level of eGFR was associated with increased EF, LASr and TAPSE and decreased LA Vol, LAVI and LV-GLS, LAScd and LASct.

#### References

- Nardi E, Palermo A, Mulè G, Cusimano P, Cottone S, Cerasola G. Left ventricular hypertrophy and geometry in hypertensive patients with chronic kidney disease. J Hypertens. 2009;27:633-41.
- Sarnak MJ. Cardiovascular complications in chronic kidney disease. Am J Kidney Dis. 2003;41:11-7.
- Hosseinpanah F, Barzin M, Golkashani HA, Nassiri AA, Sheikholeslami F, Azizi F. "Association between moderate renal insufficiency and cardiovascular events in a general population: Tehran lipid and glucose study". BMC Nephrology. 2012;13:59.
- Potter E, Marwick TH. Assessment of Left Ventricular Function by Echocardiography: The Case for Routinely Adding Global Longitudinal Strain to Ejection Fraction. JACC Cardiovasc Imaging. 2018;11:260-74.
- Kimura K, Kimura T, Ishihara M, Nakagawa Y, Nakao K, Miyauchi K, et al. JCS 2018 guideline on diagnosis and treatment of acute coronary syndrome. Circulation Journal. 2019;83:1085-196.
- Chen TK, Knicely DH, Grams ME. Chronic Kidney Disease Diagnosis and Management: A Review. JAMA. 2019;322:1294-304.
- Park M, Hsu CY, Li Y, Mishra RK, Keane M, Rosas SE, et al. Associations between kidney function and subclinical cardiac abnormalities in CKD. J Am Soc Nephrol. 2012;23:1725-34.
- Krishnasamy R, Isbel NM, Hawley CM, Pascoe EM, Leano R, Haluska BA, et al. The association between left ventricular global longitudinal strain, renal impairment and allcause mortality. Nephrol Dial Transplant. 2014;29:1218-25.
- Nguyen HTT, Do CV, Dang DTV, Do LD, Doan LH, Dang HTV. Progressive alterations of left atrial and ventricular volume and strain across chronic kidney disease stages: a speckle tracking echocardiography study. Front Cardiovasc Med. 2023;10:119-24.
- Wang C, Pei YY, Ma YH, Ma XL, Liu ZW, Zhu JH, et al. Risk factors for acute kidney injury in patients with acute myocardial infarction. Chin Med J (Engl). 2019;132:1660-5.

- Nakanishi K, Jin Z, Russo C, Homma S, Elkind MS, Rundek T, et al. Association of chronic kidney disease with impaired left atrial reservoir function: A community-based cohort study. Eur J Prev Cardiol. 2017;24:392-8.
- Hensen LCR, Goossens K, Delgado V, Rotmans JI, Jukema JW, Bax JJ. Prognostic Implications of Left Ventricular Global Longitudinal Strain in Predialysis and Dialysis Patients. Am J Cardiol. 2017;120:500-4.
- Paoletti E, Zoccali C. A look at the upper heart chamber: the left atrium in chronic kidney disease. Nephrol Dial Transplant. 2014;29:1847-53.

- 14. Essig M, Escoubet B, de Zuttere D, Blanchet F, Arnoult F, Dupuis E, et al. Cardiovascular remodelling and extracellular fluid excess in early stages of chronic kidney disease. Nephrol Dial Transplant. 2008;23:239-48.
- 15. Krishnasamy R, Isbel NM, Hawley CM, Pascoe EM, Burrage M, Leano R, et al. Left Ventricular Global Longitudinal Strain (GLS) Is a Superior Predictor of All-Cause and Cardiovascular Mortality When Compared to Ejection Fraction in Advanced Chronic Kidney Disease. PLoS One. 2015;10:127-44.

**To cite this article:** Hany H. Ebaid, Nagy E. Antar, Khaled E. Elrabat, Karim Elakabawi. Impact of Chronic Kidney Disease on Left Ventricular and Left Atrial Deformation in Patients with Anterior Myocardial Infarction as Detected by 2D -Speckle Tracking. BMFJ XXX, DOI: 10.21608/bmfj.2024.305380.2136