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# Left Atrial Volume Index (LAVI) as a Predictor of Outcome for First Time ST Elevation Myocardial Infarction (STEMI), Compared to Other Conventional Echocardiographic Parameters of Left Ventricular (LV) Function

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## Abstract

*Background:* Acute Heart Failure (AHF) complicates acute Myocardial Infarction (AMI) as a result of complex interaction of structural, hemodynamic, neurohormonal, and genetic maladaptations. This study aims to analyse the role of Left Atrial Volume Index (LAVI) compared to other conventional parameters of systolic and diastolic Left Ventricular function (LV) in patients with first time ST Segment Elevation Myocardial Infarction (STEMI), in predicting in-Hospital Heart failure (HF), in-hospital mortality and development of heart failure and subsequent rehospitalisation over a follow-up period of 6 months.

*Material and Methods:* The present study is a prospective observational study conducted in the Cardiology Department of Assiut University Hospitals (AUH) on 70 STEMI patients admitted to Coronary Care Unit (CCU). Left Ventricular Ejection Fraction (LVEF), LV End-Systolic and End-Diatolic Dimensions (LVESD and LVEDD), LAVI, diastolic and systolic parameters were measured within 24 hours after admission and then 6 months later. These variables were correlated with the development of heart failure according to Killip classification on admission and 6 months later by NYHA classification.

*Results:* There was a statistically significant difference regarding development of in-hospital HF (Killip classification >II) with the following variables LAV, LAVI, LVESD, FS, EF, and the diastolic parameters used to evaluate diastolic function except Isovolumetric Relaxation Time (IVRT) and E/A ratio, whereas in-hospital mortality was related significantly to the same variables except LAVI, E/A ratio and EF by univariate analysis. ($p$-value <0.001).

After 6 months of follow-up, there was a statistically significant relation between mortality development and the following variables; LAVI, EF, Deceleration Time (DT), IVRT, TDI (tissue Doppler imaging) septal e` and E/e` by univariate analysis. ($p$-value <0.001).

A statistically significant correlation between development of HF symptoms according to NYHA classification and subsequent rehospitalization was observed with the previously mentioned variables except DT and IVRT by univariate analysis. ($p$-value <0.001).

LAVI, EF, TDI septal é and E/é ratio had a statistically significant correlation with development of in-hospital HF ($p$-value <0.001), but only E/é ratio and TDI septal e` were significantly related to in-hospital mortality ($p$-value <0.05). All the previously mentioned four variables were significant predictors of mortality or symptoms of HF and rehospitalization over six months follow-up. ($p$-value <0.05).

Using multiple regression analysis, LAVI ml/$m^2$, E/e` ratio, EF were the most significant predictors of in-hospital HF with E/e` ratio being the most powerful predictor. ($p$-value=0.001) and LAVI ml/$m^2$ was the most powerful predictor of mortality during the follow up period. ($p$-value=0.03).

*Conclusions:* LAVI and other determinants of systolic and diastolic functions of the heart played an important role in prediction of HF and mortality both in the in-hospital setting and after a follow-up period of 6 months.

LAVI, E/e` ratio and EF are the most significant predictors of in hospital heart failure with E/e` ratio being the most powerful predictor, LAVI and EF were the most powerful predictors of mortality and HF during a follow-up period of 6 months respectively.

***Key Words:*** *STEMI – Killip – Left ventricle diastolic dysfunction – LAVI.*

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## Introduction

**MYOCARDIAL** Infarction (MI) can be recognized by clinical features, including Electrocardiographic (ECG) findings, elevated values of biochemical markers of myocardial necrosis, and by imaging, or may be defined by pathology. It is a major cause of death and disability worldwide [1].

Myocardial ischemia induced by exercise or pharmacological stress causes myocardial dysfunc-

tion. In response to acute ischemia, diastolic dysfunction develops before systolic dysfunction becomes apparent. Thus, it is not surprising that stress-induced diastolic dysfunction has been repeatedly reported to be a more sensitive determinant for detection of Coronary Artery Disease (CAD) than measuring systolic dysfunction [2-6].

The presence of diastolic dysfunction in patients with CAD with MI, Acute Coronary Syndrome (ACS), and chronic stable CAD has a prognostic impact regarding future events [7]. The presence of diastolic dysfunction in patients who have suffered an MI indicates a poor prognosis with a higher risk of in-hospital or early mortality and more marked Left Ventricular (LV) remodelling [8,9].

LV systolic function has been extensively studied in relation to development of HF; however it has now become apparent that LV diastolic function contributes to symptoms and signs of clinical Heart Failure (HF) [10].

Other echocardiographic parameters like Left Atrial Volume (LAV) and diastolic indices like E/A ratio, Deceleration Time (DT) are believed to be useful in predicting early in-hospital HF in patients with ST elevation MI (STEMI) [10].

This study aims to analyze the role of LAV compared to other conventional parameters of systolic and diastolic LV function in patients with first time STEMI, in predicting in-hospital HF, in-hospital mortality and development of HF, rehospitalization and mortality over a follow-up period of 6 months.

## Material and Methods

The present study is a prospective observational study conducted in the Cardiology Department of Assiut University Hospitals (AUH) on 70 STEMI patients admitted to Coronary Care Unit (CCU) between 1 st of February 2016 and 30 th of June 2016. They were all qualified after exclusion criteria.

We excluded patients with prior history of non STEMI, early reinfarction, previous Coronary Bypass (CABG), valvular heart disease, congenital heart disease, left bundle branch block, chronic HF.

*Demographic, medical and laboratory data:*

Demographic and medical data including age, gender, smoking, Diabetes Mellitus (DM), Hypertension (HTN), renal insufficiency, previous MI, Percutaneous Intervention (PCI) or CABG, family history and dyslipidemia.

The Body Mass Index (kg/m2) (BMI) and body surface area ($m^2$) were calculated.

*Clinical examination stressing on:*

Blood pressure, Pulse, cardiac examination, and Killip class [11] also recorded.

Clinical examination of the patients was repeated 6 months later with additional assessment of symptoms of heart failure using New York Heart Association (NYHA) classification [12].

*Management strategies:*

ACS subdivided into three subgroup patient with acute MI, whose underwent different management strategies as primary PCI, received Streptokinase (SK), SK then rescue PCI and medical treatment.

*Laboratory investigations:*

All patients investigated with routine work up as cardiac enzymes (Troponin I ng/ml), urea nitrogen (mg/dl), creatinine (mg/dl), serum sodium (mmol/l), potassium levels (mmol/l), magnesium (mmol/l), lipid profile mg/dl, complete blood picture, Prothrombin Concentration (PC) and INR.

*Echocardiography:*

All patients were examined by transthoracic Doppler echocardiography (Phillips i.e. 33 ultrasound system device) within 24 hours of admission using the following protocol:

- With M-mode, LV Ejection Fraction (EF), LV dimensions, diastolic and systolic dimensions were measured. Segmental wall motion defects were assessed by 2-dimensional echocardiography [13].
- LAV calculated, using an area length formula, (Equation 1) [13]:

*Equation 1:* Shows area length formula to calculate LAV and LAVI:

$$\text{Left artial volume (LAV) ml} = \frac{8}{3\pi}\left\{\quad\right\}\left\{\frac{(A1.\ A2)}{L}\right\}$$

$$\text{Left artial volume index (LAV) ml/m}^2 = \frac{\text{Left artial volume (LAV) ml}}{\text{Body surface area (BSA) m2}}$$

The area length A1 and A2 measured in apical four chambers (A4C) view and apical two chambers (A2C) view respectively, by tracing the area of LA. Length (L) is defined as the shortest of the

two long axes measured in the A4C view and A2C view at ventricular end-systole.

- *Grading of diastolic dysfunction (LVDD):* Patients who had LVDD was classified into three grades accordingly **[14]**:
  - *Grade 1:* Prolonged relaxation pattern.
  - *Grade 2:* Pseudo-normalization pattern; and.
  - *Grade 3:* Restrictive pattern.

*The following diastolic parameters was used for grading LV diastolic function:*

- Pulsed Doppler mitral flow velocity (E, A, E/A ratio, and deceleration time of early filling (DT), and the Isovolumic Relaxation Time (IVRT).
- Tissue Doppler Imaging (TDI) (e` and a` velocity at septal mitral valve annulus).
- The Valsalva manoeuvre was done if needed to confirm the grade of LVDD.

*Statistical analysis:*

The data were tested for normality using the Anderson-Darling test and for homogeneity variances prior to further statistical analysis. Categorical variables were described by number and percent (N, %), where continuous variables were described by mean and standard deviation (mean ± SD). Chi-square test and Fisher exact test were used to compare between categorical variables whereas comparison was done between continuous variables by *t*-test and ANOVA. Pearson correlation coefficients were used to assess the correlation between continuous variables. Multiple regression analysis was used for multivariate analysis. A two-tailed $p < 0.05$ was considered statistically significant. All analyses were performed with the IBM SPSS 20.0 software.

Informed oral consent was obtained from all participants after explanation of all steps of the study. It was explained to all the participants that the collected data is confidential and for the purpose of the scientific research only, all investigations regarding electrocardiographic, echocardiographic and, laboratory investigations were free without any cost to the participant, any faulty habits towards treatment and lifestyle modification were advised through health education by the researcher himself. The Ethical Committee of Assiut Faculty of Medicine approved the study.

## Results

A total of 70 patients were included in this study, who were admitted in Cardiology Department of Assiut University Hospital, they presented with acute STEMI, 54 patients (77.1%) were males with a mean age of 55.6±13.1 years. Diabetics were (35.7%), hypertensives were (21.4%) and 62.8% were smokers.

Table (1): Baseline characteristics of patients.

| | No. | % |
|---|---|---|
| *Age:* | | |
| Range | 20-85 | |
| Mean ± SD | 55.6±13.1 | |
| *Gender:* | | |
| Male | 54 | 77.1 |
| Female | 16 | 22.9 |
| *BMI:* | | |
| Range | 18.8-41.5 | |
| Mean ± SD | 27.9±4.5 | |
| *Smoking:* | | |
| Yes | 44 | 62.9 |
| No | 26 | 37.1 |
| *DM:* | | |
| Yes | 25 | 35.7 |
| No | 45 | 64.3 |
| *HTN:* | | |
| Yes | 15 | 21.4 |
| No | 55 | 78.6 |
| *Previous IHD:* | | |
| Yes | 10 | 14.3 |
| No | 60 | 85.7 |
| *Family history:* | | |
| Yes | 4 | 5.7 |
| No | 66 | 94.3 |
| *History of dyslipidemia:* | | |
| Yes | 3 | 4.3 |
| No | 67 | 95.7 |
| *Diagnosis:* | | |
| Anterior MI | 14 | 20.0 |
| Extensive anterior MI | 27 | 38.6 |
| Extensive inferior MI | 12 | 17.1 |
| Inferior MI | 15 | 21.4 |
| Lateral MI | 2 | 2.9 |
| *Management strategy:* | | |
| PPCI | 33 | 47.1 |
| Ant ischemic | 2 | 2.9 |
| SK | 35 | 50.0 |
| *Killip class:* | | |
| I | 37 | 52.9 |
| II | 10 | 14.3 |
| III | 20 | 28.6 |
| IV | 3 | 4.3 |

Patients who received thrombolysis by SK were 35 (50%), while 33 patients underwent primary PCI (47.1%) and only two patients had neither due to late presentation.

The number of patients who developed in hospital HF with Killip class >II were 23 (32.9%), 20 of them being in class III (28.6%) and 3 (4%) patients in class IV. A total of four patients died during admission where in-hospital mortality of 5.7%.

On follow-up 6 months later, further seven patients died due to cardiac causes (10%) and twenty patients were admitted again within six months due to cardiac causes (development of heart failure and new ischemic insult).

There was a statistically significant correlation regarding development of in-hospital heart failure (Killip classification >II) with the following variables:

LAV, LAVI, LVESD, EF, and the diastolic parameters used to evaluate diastolic function.

However, there was a statistically significant correlation regarding development of in-hospital mortality with the same variables except LAVI, E/A ratio and EF.

Six months later, follow-up of our patients revealed a statistically significant correlation between mortality development and the following variables:

LAVI, EF, E/A ratio, DT, IVRT, TDI Septal e`, E/e` .

Also, a statistically significant correlation between development of HF symptoms according to NYHA classification and subsequent rehospitalization and the following variables; LAVI, EF, E/A ratio, TDI septal é and E/é ratio.

It was found that LAVI correlated significantly with other echocardiographic indices of systolic and diastolic function using Pearson correlation coefficients.

LAVI ml/$m^2$, E/e` ratio, EF were the most significant predictors of in-hospital heart failure with E/e` ratio being the most powerful predictor and LAVI was the most powerful predictor of mortality over that period.

However, EF appeared to be the most important predictor of development of symptoms of heart failure and need for rehospitalization over the same follow-up period.

Table (2): Effect of different echocardiographic variables on Killip classification grading on admission.

| | Killip class | | | | *p*-value |
|---|---|---|---|---|---|
| | I (n=37) Mean ± SD | II (n=10) Mean ± SD | III (n=20) Mean ± SD | IV (n=3) Mean ± SD | |
| Left atrium A1 | 15.5±2.9 | 17±4.9 | 20.3±2.9 | 20.3±2.1 | 0.000** |
| Lt atrium A2 | 15.4±3.2 | 17.2±4.6 | 20.4±3.4 | 21±3.6 | 0.000** |
| LAV ml | 47.7±13.4 | 60.8±25.4 | 77.9±16.4 | 78.2±13.4 | 0.000** |
| LAVI ml/$m^2$ | 24.6±7.3 | 26.9±10.3 | 36.2±3.9 | 38.9±4.9 | 0.000** |
| LV ESD | 3.6±0.5 | 3.7±0.5 | 4.1±0.6 | 4.2±0.7 | 0.002** |
| FS | 27.9±5.1 | 25.5±6.3 | 22±3.9 | 23.3±6 | 0.001** |
| EF | 52.4±7 | 46.2±11.8 | 41.6±6.5 | 44.7±10.3 | 0.000** |
| E/A ratio | 0.9±0.3 | 1.1±0.5 | 1.5±0.9 | 1.4±0.7 | 0.012* |
| DT | 209.3±25.7 | 195.7±26.9 | 175.5±28.7 | 166.7±23.1 | 0.000** |
| IVRT | 93.9±18.3 | 82±19.3 | 84.5±18.6 | 70±10 | 0.046* |
| TDI Septal e` | 7.5±1.6 | 6.3±1 | 6±0.9 | 5.1±0.4 | 0.000** |
| E/e` | 7.9±2 | 9.9±3.5 | 13.2±3.1 | 14.9±4.5 | 0.000** |

LAV : Left Atrial Volume.
LAVI : Left Atrial Volume Index.
LVESD : Left Ventricular Endsystolic Diameter.
FS : Fractional Shortening.
EF : Ejection Fraction.
DT : Deceleration Time.
IVRT : Isovolumetric Relaxation Time.
TDI : Tissue Doppler Imaging.

Table (3): Relation of in-hospital mortality with different echocardiographic variables.

| | In-hospital mortality | | *p*-value |
|---|---|---|---|
| | Yes (n=4) Mean ± SD | No (n=66) Mean ± SD | |
| LAV ml | 88.2±14 | 57.7±20.3 | 0.004** |
| LAVI ml/m$^2$ | 36.8±1.9 | 28.4±8.8 | 0.059 |
| FS | 25.3±2.6 | 25.7±5.7 | 0.877 |
| EF | 46.3±6.7 | 48.2±9.2 | 0.679 |
| E/A ratio | 1.6±0.5 | 1.1±0.6 | 0.080 |
| DT | 162.5±20.6 | 197.9±30 | 0.024* |
| IVRT | 70±8.2 | 89.6±19 | 0.045* |
| TDI Septal e` | 5.3±0.6 | 6.9±1.5 | 0.042* |
| E/e` | 16.8±2.1 | 9.6±3.3 | 0.000** |

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Table (4): Relation of mortality follow-up with different echocardiographic variables.

| | Mortality follow-up | | *p*-value |
|---|---|---|---|
| | Yes (n=7) Mean ± SD | No (n=59) Mean ± SD | |
| LAV ml | 79.5±14.3 | 55.8±20.1 | 0.000** |
| LAVI ml/m$^2$ | 38±3 | 27.1±8.4 | 0.000** |
| FS | 22.7±3.3 | 26.2±5.7 | 0.049* |
| EF | 42.2±6 | 49.2±9.1 | 0.017* |
| E/A ratio | 1.5±0.6 | 1.1±0.6 | 0.042* |
| DT | 171.8±24 | 200.3±29.7 | 0.004** |
| IVR | 70.5±14.2 | 91.8±18 | 0.000** |
| TDI Septal e` | 5.5±1 | 7±1.5 | 0.003** |
| E/e` | 14.1±3.7 | 9.2±3.1 | 0.000** |

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Table (5): Relation of NYHA classification grading and subsequent rehospitalization with different echocardiographic variables.

| | LAVI ml/m$^2$ (mean ± SD) | EF (mean ± SD) | E/A ratio (mean ± SD) | TDI septal e` (mean ± SD) | E/e` (mean ± SD) |
|---|---|---|---|---|---|
| *NYHA-F:* | | | | | |
| I | 24.44±7.78 | 53.19±7.79 | 0.94±0.3 | 7.59±1.56 | 8.2±2.23 |
| II | 28.61±8.18 | 44.58±9.2 | 1±0.37 | 6.24±1.26 | 9.73±2.97 |
| III | 34.35±6.49 | 41.09±4.78 | 1.53±1.09 | 6.08±0.65 | 12.04±3.82 |
| *p*-value | 0.001 ** | <0.001 ** | 0.010* | 0.001 ** | 0.001 ** |
| *Rehospitalization:* | | | | | |
| Yes | 33.85±6.12 | 42.25±6.99 | 1.29±0.86 | 5.94±0.8 | 11.51±3.21 |
| No | 23.69±7.36 | 52.74±8.03 | 0.95±0.32 | 7.59±1.52 | 8.06±2.24 |
| *p*-value | <0.001 ** | <0.001 ** | 0.033* | <0.001** | <0.001 ** |

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LAVI : Left Atrial Volume Index.
LVESD : Left Ventricular Endsystolic Diameter.
FS : Fractional Shortening.
EF : Ejection Fraction.
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TDI : Tissue Doppler Imaging.

Table (6): Correlation between different echocardiographic variables.

| | LAVI ml/m$^2$ | EF | E/A ratio | TDI septal e` | E/e` | DT |
|---|---|---|---|---|---|---|
| *LAVI ml/m$^2$:* | | | | | | |
| *r* | 1 | | | | | |
| *p* | | | | | | |
| *EF:* | | | | | | |
| *r* | –0.443 | 1.0 | | | | |
| *p* | <0.001** | | | | | |
| *E/A ratio:* | | | | | | |
| *r* | 0.356 | –0.317 | 1.0 | | | |
| *p* | 0.002** | 0.008** | | | | |
| *TDI Septal e`:* | | | | | | |
| *r* | –0.478 | 0.473 | –0.236 | 1.0 | | |
| *p* | <0.001** | <0.001** | 0.049* | | | |
| *E/e`:* | | | | | | |
| *r* | 0.626 | –0.459 | 0.709 | –0.652 | 1.0 | |
| *p* | <0.001** | <0.001** | <0.001** | <0.001** | | |
| *DT:* | | | | | | |
| *r* | –0.659 | 0.521 | –0.669 | 0.478 | –0.780 | 1.0 |
| *p* | <0.001** | <0.001** | <0.001** | <0.001** | <0.001** | |

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Table (7): Multivariate analysis to assess the most powerful predictors of Killip classification.

| | Beta | *t* | *p*-value |
|---|---|---|---|
| LAVI ml/m$^2$ | 0.28 | 2.43 | 0.018* |
| EF | –0.24 | –2.44 | 0.017* |
| E/A ratio | –0.18 | –1.32 | 0.191 |
| TDI Septal e` | 0.09 | 0.71 | 0.479 |
| E/e` | 0.76 | 4.10 | 0.001** |
| DT | 0.23 | 1.50 | 0.139 |

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Table (8): Multivariate analysis to assess the most powerful predictors of mortality follow-up.

| | ODDS (95% CI) | *p*-value |
|---|---|---|
| LAVI ml/m$^2$ | 0.68 (0.48-0.96) | 0.030* |
| EF | 0.98 (0.84-1.14) | 0.756 |
| E/A ratio | 1.31 (0.14-11.84) | 0.811 |
| TDI Septal e` | 1.36 (0.26-7.2) | 0.714 |
| E/e` | 0.62 (0.36-1.06) | 0.081 |
| DT | 0.97 (0.91-1.03) | 0.334 |

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EF : Ejection Fraction.
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TDI : Tissue Doppler Imaging.

Table (9): Multivariate analysis to assess the most powerful predictors of NYHA class and rehospitalization.

| | Beta | *t* | *p*-value |
|---|---|---|---|
| LAVI ml/m$^2$ | 0.24 | 1.60 | 0.115 |
| EF | –0.36 | –2.77 | 0.008** |
| E/A ratio | 0.21 | 1.29 | 0.204 |
| TDI Septal e` | –0.23 | –1.52 | 0.134 |
| E/e` | –0.12 | –0.51 | 0.614 |
| DT | 0.02 | 0.13 | 0.900 |

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TDI : Tissue Doppler Imaging.

## Discussion

The main purpose of this study was to analyse the role of LAVI compared to other conventional parameters of systolic and diastolic LV function in patients with first time STEMI, in predicting early HF and mortality during in-hospital evolution by echocardiography.

LV diastolic dysfunction progresses due to progressive LV remodelling after AMI and leads to a rise in LV End Diastolic Pressure (LVEDP). Because the left atrium is influenced by LVEDP while the mitral valve is open, LA pressure increases due to the persistent rise of LVEDP, and LAVI increases. After AMI progressive diastolic dysfunction due to LV remodelling and increasing LVEDP have been reported to indicate a poor prognosis **[15]**.

Alashetty et al., analyzed the role of LAVI compared to other conventional parameters of systolic and diastolic LV function in patients with first time STEMI, in predicting early HF during in-hospital evolution by echocardiography **[16]**. Their results analyzed LAVI, the mean LAVI in patients with HF Killip >II was 28.96ml/m$^2$ and those without HF was 22.55ml/m$^2$ which was statistically significant ($p$ 0.01) **[16]**.

Likewise, our study showed elevated LAVI in patients with Killip class III and IV, it had a mean value of 36.2±3.9 in patients with Killip class III patients and 38.9±4.9 in Killip class IV patients compared to 24.6±7.3 in Killip class I patients and 26.9± 10.3 in Killip class II patients. The relation was statistically significant with $p$-value <0.001.

Souza et al., did not observe any significant result between HF and LAVI with a mean LAVI of 18.7±4.8 in patients with no CHF, and 20.6±5.7 in patients with CHF following first STEMI with $p$-value of 0.1 **[17]**.

This difference may be attributed to the fact that in that study, LA volume Index was not significantly different between patients with or without CHF in the univariate analysis. This result was expected, since LA remodeling could not occur within 48 hours of initial presentation of a first AMI, because it is not a marker of acute changes in diastolic function and/or increased filling pressures and echocardiographic examination was performed within 30 hours of onset of chest pain **[10]**.

Moller et al., also analyzed the role of LAVI and other indices of diastolic and systolic function after AMI in 314 patients, they observed a significant association between LAVI and Killip class with a $p$-value <0.001 **[18]**.

In our study, LAVI was not significantly related to development of in-hospital mortality with a mean of 36.8± 1.9 in patients who died during admission and 28.4±8.8 in patients who didn't and a $p$-value of 0.059.

This result is not correspondent with other studies analyzing the role of LAVI as a predictor of mortality following AMI, for example Moller et al., in 2003 concluded that LAVI is a predictor

of survival after AMI [18]. Furthermore, LAVI provides prognostic information incremental to clinical data and standard echocardiographic predictors of outcome. They had a mean of LAVI of more than 32mL/m2 in 104 out of 268 surviving patient and 38 out of 46 deceased patients following AMI with a $p$-value of 0.001, this difference might be due to the smaller size of our research sample of patients and the fact that Moller's study included all-cause mortality over a median follow-up period of 15 months.

However, LAVI was an important predictor of subsequent development of heart failure symptoms and rehospitalization due to cardiac causes after a follow-up of 6 months with a mean value of 36.6±7 in patients with NYHA class III symptoms, 33.2±7 in patients with NYHA class II symptoms and 25.3±7.9 in patients with NYHA class I, $p$-value was <0.001. LAVI had a mean value of 36.7±7.2 in patients who were admitted due to cardiac causes and 25±6.8 in patients who were not with a $p$-value of <0.001.

Many studies analyzed the role of EF as a predictor for in hospital HF like Alashetty et al., in 2014 showed that EF is a powerful index of systolic function and also its role in predicting in hospital heart failure has been well established [16].

Mean EF in patients with CHF present was 36% and in those without CHF was 45%, which was statistically significant $p$<0.001 [16].

Souza et al., in their 2011 study concluded that EF <40% was a powerful independent variable associated with development of CHF killip >II with a mean of 0.51±0.07 in patients with no CHF and 0.44±0.07 in patients with CHF ($p$-value <0.001), it was superior to other indices of systolic and diastolic function [17].

Poulsen et al., also observed that EF <45% was significant prognostic parameter in assessing LV function and predicting in hospital HF with mean EF of 50±10 in patients without CHF and 41±10 in patients with CHF following first acute STEMI [19].

Our study confirmed the previous data with EF having a statistically significant relation with development of in hospital HF with mean values of 52.4±7 and 46.2±11.8 in Killip I and Killip II patients respectively, and 41.6±6.5 and 44.7±10.3 in patients with Killip III and IV patients respectively and a $p$-value of <0.0001.

However, EF doesn't appear to be a significant predictor of in hospital mortality with a mean of 46.3±6.7 in patients with in hospital mortality, and 48.2±9.2 in surviving patients ($p$-value=0.68).

Of course, EF played an important role as a predictor of mortality over a follow-up period of 6 months with a mean ± SD value of 42.2±6 in patients who died within this period and 49.2±9.1 in surviving patients. ($p$-value=0.017). It also predicted development of HF symptoms and rehospitalization due to cardiac causes with follow-up with a mean ± SD value of 39.2±6.2 in NYHA III patients, 46.6±11.2 in NYHA II patients and 58.2±7 in NYHA I patients. ($p$-value <0.001). It had a mean ± SD value of 41.4±8 in patients who were admitted due to cardiac causes and 57.8±7.6 in patients who were not with a $p$-value of <0.001.

DT is an important determinant of diastolic function, Alashetty et al., demonstrated that Deceleration time (DT) showed a significant association with a presence of heart failure ($p$-value 0.04) [16]. Souza et al., observed no significant association between DT and heart failure (killip >II) [17] whereas Poulsen et al., found that a DT of <130ms identified a risk of developing HF [19].

Our study showed that DT had a statistically significant relation with development of in-hospital HF with mean ± SD values of 209.3±25.7 and 195.7±26.9 in Killip I and Killip II patients respectively, and 175.5±28.7and 166.7±23.1 in patients with Killip III and IV patients respectively, and a $p$-value of <0.0001. It was also a significant predictor of in-hospital mortality with a mean ± SD of 162.5±20.6in patients with in-hospital mortality, and 197.9±30 in surviving patients ($p$-value=0.024).

DT played an important role as a predictor of mortality over a follow-up period of 6 months with a mean ± SD value of 171.8±24 in patients who died within this period and 200.3±29.7 in surviving patients. ($p$-value=0.004).

It also predicted development of HF symptoms and rehospitalisation due to cardiac causes with follow-up with a mean ± SD value of 175±33.5 in NYHA III patients, 215.7±58.9 in NYHA II patients, and 209.6±19 in NYHA I patients. ($p$-value =0.007). It had a mean ± SD value of 183.3±31 in patients who were admitted due to cardiac causes, and 215.2±33.4 in patients who were not with a $p$-value of <0.001.

E/é ratio had a statistically significant relation with development of in-hospital HF with mean ± SD values of 7.9±2 and 9.9±3.5 in Killip I and

Killip II patients respectively, and 13.2 ±3.1 and 14.9±4.5 in patients with Killip III and IV patients respectively and a *p*-value of <0.001.

It was also a significant predictor of in-hospital mortality with a mean ± SD of 16.8±2.1 in patients with in hospital mortality and 9.6 ±3.3 in surviving patients (*p*-value <0.001).

E/é ratio played an important role as a predictor of mortality over a follow-up period of 6 months with a mean ± SD value of 14.1 ±3.7 in patients who died within this period and 9.2 ±3.1 in surviving patients. (*p*-value <0.001).

It predicted development of heart failure symptoms admission due to cardiac causes with follow-up with a mean ± SD value of 12.6±3.5 in NYHA III patients, 9.8 ±3.9 in NYHA II patients, and 8.6±2.7 in NYHA I patients. (*p*-value=0.002). It had a mean ± SD value of 11.7±3.8 in patients who were admitted due to cardiac causes and 8.6 ±2.6 in patients who were not with a *p*-value of <0.001.

*Conclusions:*

LAVI, E/e` ratio and EF are the most significant predictors of in hospital heart failure with E/e` ratio being the most powerful predictor.

LAVI was the most powerful predictor of mortality follow-up period and EF is the most important predictor of development of symptoms of heart failure over the same follow-up period.

*Conflicts of interest notification:*

The authors have no conflicts of interest to declare.

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# الدلالات المشتقة من الآشعة التليفزيونية على القلب وعلاقتها بحدوث فشل مبكر بعضلة القلب بعد الإصابة بإحتشاء عضلة القلب المصحوب بإرتفاع قطاع الاس تى آثناء الإقامة بالمستشفى

المقدمة: إحتشاء عضلة القلب الحاد هو مشكلة صحية عامة ومتزايدة فى البلدان النامية. فشل عضلة القلب الناتج عن الإحتشاء هو سبب هام من آسباب الوفيات وآمراض القلب والآوعية الدموية بشكل عام كما يساهم بشكل كبير فى العبء العالمى لآمراض القلب والآوعية الدموية. وقد زادت حالات الإصابة بضعف عضلة القلب بشكل كبير بسبب الدور المتزايد لآمراض الشرايين التاجية ولهذا فإن الكشف المبكر عن مرضى إحتشاء عضلة القلب الحاد المعرضين لخطر فشل عضلة القلب آثناء فترة حجزهم بالمستشفى هو ضرورى للحد من إصابة عضلة القلب وإختلال وظيفتها.

المرضى والطرق: إلتحق فى هذه الدراسة ذات المقطع المستعرض ٧٠ شخصا من المصابين بإحتشاء عضلة القلب الحاد وقد تم قياس الوظيفة الإنقباضية لعضلة القلب مع حجم الآذين الآيسر (LAV) ومؤشر حجم الآذين الآيسر (LAVI) وقياسات عوامل الوظيفة الإنبساطية للبطين الآيسر. وقد تم تصنيف درجات إختلال الوظيفة الإنبساطية للبطين الآيسر كما يلى: الدرجة الآولى الضعف الإسترخائى، الدرجة الثانية الضعف شبه الطبيعى الكاذب والدرجة الثالثة نمط التدفق الميترالى المقيد.

الإستنتاجات: لعبت مؤشر حجم الآذين الآيسر (LAVI) وغيرها من العوامل المحددة للمهام الإنقباضية والإنبساطية لعضلة القلب دورا مهما فى التنبؤ بفشل عضلة القلب ومعدل الوفيات آثناء فترة الإقامة بالمستشفى وآيضا بالتنبؤ بمعدل الوفيات وفشل عضلة القلب خلال فترة متابعة لمدة ٦ آشهر.

وقد آظهرت الدراسة آن مؤشر حجم الآذين الآيسر وبفة العضلة الإنقباضية ونسبة E/e` خاصة هى آكثر المؤشرات التى تتنبآ بحدوث فشل عضلة القلب آثناء الإقامة بالمستشفى.

بينما كان مؤشر حجم الآذين الآيسر مؤشرا قويا لحدوث وفيات آثناء فترة المتابعة البالغة ستة آشهر وكانت وظيفة عضلة القلب الإنقباضية هى آهم مؤشر لحدوث آعراض قصور القلب بنفس فترة المتابعة.