DISCUSSION

Regarding the left ventricular function

Left ventricular systolic function (LVEDV, LVESV and EF)

After AMI, the feature that most adversely affects long-term survival is LV dilatation, which in some studies ranks even higher than the severity of CAD as a prognostic feature[125].

Our results showed that patients have higher LVEDV, LVESV and lower ejection fraction (169.83±49.53 ml, 94.2±34.54 ml and 45.8±8.8 %) respectively compared with controls (104.35±24.93 ml, 38.95±8.57 ml and 62.2±4.8 %) respectively (p=0.0001). This is in agreement with Zhang et al.[126] who found that LVEDV, LVESV and EF in patients group were (87.3± 15.0 ml, 44.2± 9.8 ml and 49.7±6.6%) versus (75.2±12.9 ml, 31.6± 7.8 ml and 64.9±6.2%) in the control respectively (P value <0.05).

Dogan et al.[2] reported that LVEDV was (110.1±18.4 ml in patients versus 107.0 ± 18.6 ml in control) (P= 0.5) LVESV was (59.4 ± 15.2 ml, in patients versus 41.7 ± 8.1 ml in control) (p=0.005), and EF was (46.6 ± 9.2 % in patients versus 61.0 ± 5.2 % in control) (P= 0.001).

Korup et al.[127] stated that major left ventricular dilation is present within three hours after onset of symptom of first acute myocardial infarction.

Picard et al.[128] described that all cases of infarct expansion were identified within the first 24 hours of clinical symptoms of AMI and enlargement was observed as early as 2 hours after onset of chest pain.

A lower LVEF may be a reflection of a larger infarct, more extensive CAD, less cardiac reserve, and poorer outcome[129].

Systolic velocity (Sa) of mitral annulus by tissue Doppler imaging;

Our study demonstrated that patients with MI had significantly reduced peak systolic velocity by tissue Doppler imaging (0.067±0.015 m/s) when compared to the control group (0.098 ± 0.019 m/s), with p=0.0001. This finding is in accordance with Alam et al.[130] who reported that compared with healthy subjects, the MI patients had a significantly reduced peak systolic velocity at the mitral annulus, especially at the infarction sites. A relatively good linear correlation was found between the ejection fraction and the mean systolic velocity from the 4 LV sites (r = 0.74, p < 0.001). When the patients were divided into 2 different groups with respect to an ejection fraction ≥0.50 or <0.50, a cutoff point of mean systolic mitral annular velocity of ≥7.5 cm/s had a sensitivity of 79% and a specificity of 88% in predicting a preserved global LV systolic function. In addition to the reduced systolic velocity at the infarction sites in both anterior and inferior MI, the systolic velocity at the noninfarction sites was also reduced compared with that in healthy subjects.

In another study by Alam et al.[131] it was found that the patients with MI had significantly reduced peak systolic velocity compared to Healthy group (8.6 v. 9.7 cm/s, p < 0.001).

Regarding the left ventricular diastolic function:

Mitral inflow velocities (E;A; E/A ratio and E wave deceleration time):

Doppler echocardiographic assessment of transmitral flow provides a noninvasive means of identifying patients with elevated LA pressures[(132)]. Left ventricular relaxation is an energy dependent process. Prolonged relaxation is the earliest abnormality in left ventricular dysfunction and is preceded by filling abnormality. Combinations of different degrees of systolic and diastolic myocardial dysfunction are prevalent among patients with AMI, and both components have a significant impact on patient outcome[(133)].

Our patient population have Doppler echo finding suggestive of different degree of diastolic dysfunction ranging from impaired relaxation 18(45.0%) to pseudonormal 16(40.0%) to restrictive filling pattern 6(15%)with E/A ratio ranging from 0.54 to 3.53 and DT ranging from 90-230 ms.

The prognostic importance of a restrictive filling pattern after AMI was initially reported by Oh et al.[(134)] in 1992 In a cohort of 62 patients, a restrictive filling pattern was associated with a high occurrence of in-hospital congestive heart failure.

Williamson et al.[(135)] studied 60 patients within 24 hours of AMI by doppler echo. Of 54 patients who also underwent catheterization, 45 (83%) were successfully reperfused. A subgroup of 17 patients underwent a follow-up Doppler examination at 7 days after infarction, whereas 15 patients with stable exertional angina served as control subjects. The final recovery values at 7 days were not significantly different from those of the coronary artery disease group.

Chenzbraun et al.[(136)] studied LV filling patterns with Doppler echocardiography in 15 healthy subjects and 38 patients with recent acute myocardial infarction. LV end-diastolic pressures were low to normal in patients with an E/A ratio less than 1 and were usually greater than 15 mm Hg in those with normal or abnormally increased (greater than 2) E/A ratios. Thus, an apparently normal E/A ratio in patients after myocardial infarction may identify those with more severe LV diastolic dysfunction and increased LV filling pressure.

Moller et al.[(137)] studied echocardiography within 24 h, five days and one and three months after MI in 125 unselected consecutive patients. They concluded that pseudonormal or restrictive filling patterns are related to progressive LV dilation and predict cardiac death after a first MI.

Tissue Doppler imaging (Ea wave; Aa wave; Ea/Aa ratio and E/Ea):

Alam et al.[(130)] examined seventy-eight patients with a first MI before discharge. Peak systolic, peak early diastolic, and peak late diastolic velocities were recorded at 4 different sites on the mitral annulus corresponding to the septum, anterior, lateral, and inferior sites of the left ventricle. Nineteen age matched healthy subjects served as controls. Compared with healthy subjects, the MI patients had a significantly reduced peak systolic velocity at the mitral annulus, especially at the infarction sites. A relatively good linear correlation was found between the ejection fraction and the mean systolic velocity from the 4 LV sites ($r = 0.74$, $p < 0.001$). When the patients were divided into 2 different groups with respect to an ejection fraction ≥0.50 or <0.50, a cutoff point of mean systolic mitral annular velocity of ≥7.5 cm/s had a sensitivity of 79% and a specificity of 88% in predicting a preserved global LV systolic function. In addition to the reduced systolic velocity at the infarction sites in both anterior and inferior MI, the systolic velocity at the noninfarction sites was also reduced compared with that in healthy subjects. Similar to systolic velocities, the early diastolic velocity was also reduced, especially at the infarction sites. The peak mitral annular early diastolic velocity correlated well with LV ejection fraction ($r = 0.66$, $p < 0.001$).

In another study by Alam et al.[(131)] an echocardiogram was performed in 19 patients with acute MI who were previously healthy and had a normal ejection fraction and no wall motion abnormalities at echocardiogram. These 19 patients were compared with 16 age matched healthy subjects (HS). The longitudinal LV function was assessed using the mitral annular velocities (mean value from four different sites of the LV) determined by DTI. They showed that the patients with MI had significantly reduced peak systolic and peak early diastolic mitral annular velocities compared to Healthy group (8.6 v. 9.7 cm/s, $P < 0.001$ for systolic velocity, and 10.9 v. 12.3 cm/s, $P < 0.01$ for diastolic velocity, respectively). The patients had normal diastolic LV function assessed by the conventional Doppler echocardiogram (e.g. transmitral flow, IVRT and pulmonary venous flow patterns). To assess the LV filling pressure, the ratio of the transmitral early wave velocity assessed by conventional echo-Doppler and peak early diastolic mitral annular velocity determined by DTI (E/Edti) was used. The E/Edti was significantly higher in patients than in HS (7.0 vs. 5.7, $P < 0.05$).

Using the E/Em ratio, a close approximation of LV filling pressures can be obtained in a wide spectrum of patients[(138)]. The E/Em ratio is superior to other echocardiographic indices in this respect[(139)], and after AMI, an elevated ratio predicts higher mortality and an increased risk of adverse remodeling[(140)].

However, an E/Em ratio between 8 and 15, was associated with a very wide range of mean LV diastolic pressures in the study by Ommen et al.[(139)].

Our results come in harmony with the previous studies where it demonstrated that patients with MI had significantly reduced peak early diastolic annular velocity (Ea) compared with control (0.069±0.021 vs. 0.136±0.042) ($p=0.0001$). The late diastolic mitral annular velocity (Aa) was lower in patients than controls but the difference did not reach statistical significance (0.093±0.02 in patients vs. 0.095±0.024 in the controls) ($p=0.734$). E/Ea was significantly higher in patients than in the control (10.08±4.32 vs. 6.26±1.88). Ea/Aa ratio in the patients group showed significant lower value (0.78±0.29) than in the control group (1.58±0.76). This difference was statistically highly significant ($p<0.01$).

Regarding the left atrial function:

The left atrial diameter:

Yurdakul et al.[(3)] studied Twenty-four patients with previous anterior MI (aged 63.8±4.2 years, 56% men) and 30 healthy controls (aged 60.7±5.3 years, 60% men). They found that LA horizontal diameter [cm] was (3.8±0.3) in patients group versus (3.6±0.3) in the control group, but did not reach statistical significance (p value 0.33).

Our results showed increased LA diameter in patients compared with controls where the left atrial diameter (cm) in patients group was (4.11±0.58) versus (3.52±0.44) in the control group ($p=0.0001$).

Kizer et al.[38] demonstrated that using a population based cohort, showed that LA diameter independently predicted cardiovascular events, including nonfatal stroke, coronary heart disease, heart failure and cardiovascular mortality (risk ratio 1.04/mm, 95% CI : 1.02-1.07, $p<0.002$).

The enlargement of the LA has been associated with an increased incidence of atrial fibrillation, stroke, congestive heart failure, and mortality, especially in elderly people[141].

Regarding LA volumes:

Myocardial ischemia causes inflammatory and neurohormonal changes, along with increased platelet activation with endothelial damage. All of these factors are associated with LV diastolic dysfunction[142,143].

Ischemic injury reduces LV compliance, leading to increased LA pressure and progressive dilatation of the LA to maintain LV filling[6,42]. Numerous studies have demonstrated an association between myocardial infarction and ischemia and LA dilatation consequent to impaired LV diastolic dysfunction[42,144,145].

Left atrial enlargement, specifically following ST elevation myocardial infarction, has been reported previously[146]. Additionally, LA enlargement has been shown to be a powerful predictor of adverse cardiovascular events and sudden cardiac death in patients following STEMI[38,144,145,147].

In a study by Beinart et al.[145] clinical and echocardiographic parameters were prospectively collected in 395 consecutive patients with acute MI. Patients with LA volume index (LAVI) >32 ml/m^2 (normal ± 2 standard deviations) were compared with those with LAVI ≤32 ml/m^2. Left atrial volume index >32 ml/m^2 was found in 63 patients (19%) who had a higher incidence of congestive heart failure on admission (24% vs. 12%, $p < 0.01$), a higher incidence of mitral regurgitation, increased LV dimensions, and reduced LV ejection fraction when compared with patients with LAVI ≤32 ml/m^2. They concluded that in patients with acute MI, increased LA volume, determined within the first 48 h of admission, is an independent predictor of five-year mortality with incremental prognostic information to clinical and echocardiographic data.

Dardas et al.[148] demonstrated a significant relationship between LA function and LV diastolic dysfunction following an anterior infarction, with an associated increase in cardiovascular morbidity and mortality.

LA volume (>32mL/m^2) following an acute myocardial infarction was shown to be an independent predictor of survival, independent of clinical variables and LV systolic and diastolic function[147].

In our study we demonstrated that maximum, minimum and reservoir left atrial volumes were increased whereas the LAEF is decreased in MI patients compared to controls where the LAV max. (ml) (57.57±27.06) in patients group versus (37.74±16.84) in the control ($p=0.004$); LAV min. (ml) was (30.95±17.99) in patients group versus

(14.64±7.54) in the control (p=0.0001); LAV res. (ml) was (28.94±9.61) in patients group versus (21.02±7.36) in the controls (p=0.002) and LAEF (%) in patients group was (45.9±18.3) versus (60±14) in the control (p=003). This goes in hand with Dogan et al.[2] who demonstrated that LAV max. (ml) was (47.6±12.5) in patients group versus (41.1±10.4) in the control (p=0.001); LAV min. (ml) was (23.2±16.3) in patients group versus (16.3±5.6) in the control (p=0.001); LAV res. (ml) was (26.1±8.7) in patients group versus (21.7±5.3) in the control (p=0.010) and LAEF (%) was (51.7±8.3) in patients group versus (60.4±10.3) in the control (P= 0.001).

In our study the LAV max. index and LAV min. index were increased in patients compared with controls where the LAV max. index (ml/m^2) was (30.15±14.45) in patients group versus (20.07±9.04) in the control group (P=0.006) and LAV min. index (ml/m^2) in patients group was (16.23±9.56) versus (7.75±4.02) (P=0.0001) in the control group and this finding comes in concordance with Yurdakul et al.[3] who demonstrated that LAV max. index (ml/m^2) was (28.83±7.20) in patients group versus (19.72 ± 6.27) in the control (p=0.0001) and LAV min. index (ml/m^2) was in patients (10.21±1.07) versus (8.33±1.80) in the control (p=0.0001).

Increased LA static volumes may be the manifestation of LA remodelling which is the result of LV diastolic dysfunction due to MI. LA size predicts prognosis in patients with heart disease[149] and LA volume is an important marker of diastolic dysfunction and LV filling pressure[150]. LV filling pressure increases following acute MI, with increases in LA wall tension and LA pressure. This is a compensatory mechanism of the LA to overcome reduced compliance of the LV.

Regarding left atrial strain and strain rate:

Doppler-based strain imaging, which has emerged as adjunctive to conventional TVI, is a quantitative technique that estimates myocardial contractility relatively independent of changes in load, cardiac rotational motion and tethering effects[121].

Left atrial function and volume curves include three phases known as reservoir, conduit and booster pump[11]. LAs-strain is closely related to the LA reservoir function, besides the LA strain during atrial systole is related to the booster pump function[151]. The LA reservoir function is assessed in two consecutive phases as early and late. While the early reservoir function depends on LA relaxation, the late reservoir function depends on LV contraction through the descent of the base during systole[47]. Therefore, both LA relaxation and LV systolic function might affect the LA reservoir function and so LAs-strain.

Inaba et al.[112] found that systolic strain rate corresponded to reservoir function and early diastolic strain rate corresponded to conduit function, while late diastolic stain rate corresponded to booster pump function.

Wakami et al.[151] investigated the effect of LVEDP on LAs-strain during LV systole. They found that elevated LVEDP is associated with a decrease of LA-s strain. In addition to the LAs-strain being an index of the reservoir function, it is also closely related to LV systolic function as mentioned previously [47].

Cameli et al.[81] reported a good correlation between the peak LA strain and PCWP in patients with advanced heart failure.

Antoni et al.[79] used atrial strain to assess patients with relatively preserved LV function following acute myocardial infarction treated with percutaneous revascularization (n=320). This study reported that the evaluation of reservoir function, by atrial strain in addition to LA volume, were independent predictors of mortality, reinfarction and hospitalisation for heart failure. Furthermore, atrial strain increased prognostic value, compared to LA volume, at predicting adverse cardiovascular outcomes following infarction.

Dogan et al.[2] studied 90 STEMI patients who were treated with primary percutaneous coronary intervention and 22 healthy control subjects. STEMI patients had echocardiographic examination within 48 hours after the PCI procedure. In addition to conventional echocardiographic parameters, LA strain curves were obtained for each patient. Average peak LA strain values during left ventricular systole (LAs-strain) were measured. They found that mean LAs-strain (%) in control group was higher than MI group (30.6±5.6% vs. 21.6±6.6%; p=0.001). LAs-strain had significant correlation with LVEF (r=0.51, p=0.001), also significant inverse correlations between LAs-strain and E/Em ratio (r= −0.30, p=0.001), LA maximal volume (r= −0.41, p=0.001), LA minimal volume (r = −0.50, p=0.001) and LV end systolic volume (r = −0.37, p=0.001) were detected.

Yurdakul et al.[3] studied 24 patients with previous anterior MI (aged 63.8±4.2 years, 56% men) and 30 healthy controls (aged 60.7±5.3 years, 60% men). LA volume measurements and VVI-derived LA peak systolic strain (S), strain rate (SRs), early diastolic (ESRd) and late diastolic strain rate (LSRd) were measured. LV diastolic function was analysed by pulsed wave-Doppler and tissue velocity imaging. They found that VVI-derived LA peak systolic S, SRs and ESRd were impaired in the patients group. LA LSRd was similar between the groups.

Our results come in agreement with the previous studies where the global left atrial peak systolic strain, strain rate, early diastolic strain rate and late diastolic strain rate in patients with MI were impaired compared with control. Where the global PSS was significantly lower in patients with MI than in the controls (11.69±4.25% vs. 22.43±3.74%) with statistically significant difference (p=0.0001). The global peak systolic strain rate was significantly lower in the studied patients than in the control (1.04±0.37 S^{-1} vs. 1.81±0.28 S^{-1}) with statistically significant difference (p=0.0001), early diastolic strain rate was significantly lower among the studied patients than in the controls (-0.84±0.44 S^{-1} vs. -1.98±0.60 S^{-1}) with significant statistical difference (p=0.0001) and the global late diastolic strain rate was significantly lower among patients group than in the control group (-1.43±0.29 S^{-1} vs. -1.62±0.37 S^{-1}) with significant statistical difference (p=0.034).

Kurt et al.[82] reported that LA strain during atrial systole is significantly reduced in diastolic HF patients secondary to LA stiffness.

In agreement with the previous study the mean strain during left atrial contraction was also significantly lower in patients than in the controls where the global strain during left atrial contraction was significantly lower in patients with MI than in the control (-6.10±3.49% vs. -8.06±2.40%) with statistically significant difference (p=0.029).

Our results suggest reduction in left atrial reservoir, conduit functions and LA contractile functions in the STEMI patients as atrial contractile function is determined by preload, afterload and LA contractility[152]. In the active contractile phase, the LA contracts and projects blood into the LV in late diastole. When the LV is affected by ischemia, LV end diastolic pressure, transmitral pressure gradient and LA afterload increase. Enhanced LA wall stretch results in an increase of the contractile force with the effect of the Frank-Starling law on the LA myocardium.

In accordance with previous studies, our study showed that the global PSS of left atrium in the studied patients with MI showed significant negative correlation to E/Ea ratio (r= - 0.457, p=0.0001), and the minimum left atrial volume (r= - 0.359, p=0.005). There was a negative correlation between the PSS of left atrium and the maximum left atrial volume (r = - 0.195), however, this correlation was not statistically significant (p=0.135). A significant positive correlation was found between the PSS of left atrium in the studied patients with MI and the left ventricular ejection fraction (r= 0.338, p=0.033). Also there were positive correlations between the PSS of left atrium and the left atrial reservoir volume (r=0.246) and left atrial ejection fraction (r=0.216); however, these were not found statistically significant (p=0.127, p=0.181 respectively).

Study limitations

- We performed our study within 24 hour of hospital admission and after they receive their reperfusion strategy and not on presentation.
- Strain and strain rate have been shown to be influenced by preload and it is time consuming to be assed bedsides in STEMI patients.
- We include all the atrial segments in the analysis and we did not exclude the low quality strain and strain rate curves.
- The patients were not followed prospectively to assess the prognostic impact of decreased LA strain after MI.
- The LV filling pressure was measured by echocardiography only and we did not measure PCWP during cardiac catheterization.

SUMMARY

Myocardial infarction (MI) is a major cause of death and disability worldwide. MI may be the first manifestation of coronary artery disease or it may occur, repeatedly, in patients with established disease. It can be recognized by clinical features, electrocardiographic (ECG) findings, elevated values of biochemical markers (biomarkers) of myocardial necrosis, and by imaging, or may be defined by pathology. Acute MI results in left ventricular LV both systolic and diastolic dysfunction in survivors.

The left atrium plays a major role in LV performance. LA function is a surrogate marker of LV diastolic dysfunction. LA mechanical dysfunction occurs in LV systolic and diastolic dysfunction and coronary artery disease.

Traditionally, assessment of LA function has been performed by measuring LA size or volume with two-dimensional (2D) echocardiography and Doppler echocardiographic measurements. Currently, a method known as strain imaging is used for the quantitative assessment of myocardial deformation. Doppler-based strain imaging, which has emerged as adjunctive to conventional TVI, is a quantitative technique that estimates myocardial contractility relatively independent of changes in load, cardiac rotational motion and tethering effects.

The aim of our study was to study, left atrial function and LA myocardial deformation in patients presenting with acute ST segment elevation myocardial infarction and to show the relationship between left ventricular function and LA deformation parameters.

We enrolled 60 subjects in our work distributed as:

- **Acute STEMI group**: 40 patients with acute ST elevation myocardial infarction.
- **Control group**: 20 normal subjects forming the control group.

All patients were evaluated by history taking, clinical examination, routine laboratory investigation and cardiac enzymes (CKMB and troponin) and 12-lead ECG. Echocardiography was performed within 24 hours of admission after they received their reperfusion strategy whether PCI or thrombolytic therapy and was also performed for the control group and it included: conventional M-mode, and 2-D transthoracic echocardiographic examination and Doppler study using standard parasternal and apical views to assess left ventricular diastolic function via transmitral mitral inflow velocities, left atrial volumes and left ventricular chamber dimensions, volume and function. Pulsed wave tissue Doppler echocardiography was acquired at the mitral annulus.

Evaluation of LA deformation parameters (strain and strain rate) were carried out by tissue Doppler imaging using a frame rate of 160-200 s^{-1} and three consecutive beats were recorded from apical 4- and 2- chamber views. LA strain and strain rate were measured offline from colour tissue Doppler images of the LA obtained in the apical four and two chamber views. A narrow (10 x 2 mm) sample volume was selected due to the thin atrial walls.

The following parameters show statistically significant differences between STEMI patients group and the control group:

Left ventricular dimensions, volumes and systolic functions:

- The LVEDD and LVESD had significantly higher values in STEMI patients group than control group (p value <0.01).
- The LVEDV and The LVESV had significantly higher values in STEMI patients group than in the control group (p value <0.01).
- The LVEF was significantly lower in the studied patients with MI than in the control group (p value <0.01).

Conventional Doppler echocardiography:

- The E-wave was found to be significantly lower in the studied patients than in the control group ($p< 0.05$).
- The A wave was significantly higher in STEMI patients than in the control group ($p< 0.01$).
- The E-wave deceleration time was significantly shorter in STEMI patients group than in the control group ($p<0.01$).
- The E/A ratio is significantly lower in the STEMI patients group than in the control group ($p<0.05$).

Pulse wave tissue Doppler imaging at the mitral annulus:

- The Sa wave velocities showed significantly lower values in the in the STEMI patients group than in the control group ($p=0.0001$).
- The Ea wave velocities showed significantly lower values in the in the STEMI patent group than in the control group ($p<0.01$).
- The Aa wave showed statistically insignificantly lower value in patients than in the control ($p>0.05$).
- The Ea/Aa in the patients group showed significantly lower values than in the control group ($p<0.01$).
- The E/Ea in the patients group showed significantly higher values than in the control group ($p<0.01$).

Left atrial functions:

- The left atrial diameter in patients group was significantly higher than in the control group ($p< 0.01$).
- The LAV max. in patients group was significantly higher than in the control group ($p< 0.01$).
- The LAV max. index was significantly higher among patients than in the control ($p< 0.01$).

- The LAV min. in patients group was significantly higher than in the control group ($p < 0.01$).
- The LAV min. index in patients group was significantly higher than in the control group ($p < 0.01$).
- The LAV res. in patients group was significantly higher than in the control group ($p < 0.01$).
- The LAV res. index in patients group was significantly higher than in the control group ($p < 0.05$).
- The LAEF in patients group was significantly lower than in the control group ($p < 0.01$).

Tissue Doppler derived strain and strain rate:

- Regional left atrial strain was significantly lower in patients than in the control.
- The global left atrial PSS was significantly lower in patients with MI than in the control (p=0.0001).
- The global strain during left atrial contraction was significantly lower in patients with MI than in the control (p=0.029).
- The global peak systolic strain rate of left atrium was significantly lower in the studied patients than in the control (p=0.0001).
- The global early diastolic strain rate (ESr) of left atrium was significantly lower among the studied patients than in the control (p=0.0001).
- The global late diastolic strain rate (ASr) of left atrium was significantly lower among patients group than in the control group (p=0.034).
- The PSS of left atrium in the studied STEMI patients showed significant negative correlation to E/Ea ratio (p=0.0001); and the LAV min. (p=0.005), and significant positive correlation to the LVEF (p=0.033).
- In the studied STEMI patients, there was a negative correlation between the global PSS of left atrium and the LAV max. (p=0.135), and positive correlations with LAV res. and LAEF, however, these correlation were not statistically significant (p=0.127, p=0.181 respectively).
- The global strain during LA contraction in the studied STEMI patients showed insignificant negative correlations with LAV max., LAV res., and E/Ea ratio (p=0.619, p=0.060, p=0.466) respectively, and insignificant positive correlation to the LAV min., LAEF and LVEF (p=0.734, p=0.091, p=0.971 respectively).
- The global PSSR in the studied STEMI patients showed significant positive correlations with LAV res. (p=0.014), LAEF (p=0.0001) and LVEF (p=0.048), and insignificant positive correlations with LAV max. (p=0.559). But insignificant negative correlation to the LAV min. (p=0.265) and E/Ea ratio (p=0.610).

- The global ESr in the studied STEMI patients showed insignificant negative correlations with LAV max. (p=0.546), LAV res. (p=0.161), E/Ea ratio (p=0.796) and significant positive correlation to LVEF (p=0.011). But insignificant positive correlation to the LAV min. (p=0.936) and LAEF (p=0.109).
- The global ASr in the studied STEMI patients showed insignificant positive correlations with LAV max. (p= 0.930), LAV min. (p= 0.346), and the E/Ea ratio (p=0.529), and insignificant negative correlations to LAV res. (p=0.065) and LVEF (p=0.993). But significant positive correlation to LAEF (p=0.036),
- The peak systolic strain of left atrium in the STEMI patients who were underwent primary PCI was significantly higher than in patients who were received thrombolytic therapy (p= 0.0001).

CONCLUSIONS

From the current study, we conclude that:

- Our study had demonstrated significant increase in left atrial volumes in STEMI patients in comparison to control.
- Reduced left atrial reservoir function (PSS, PSSR), conduit function (ESr), and booster function (ASr) in STEMI patients in comparison to control.
- The left atrial reservoir function, and conduit functions (PSS, PSSR, ESr) in STEMI patients were decreased consistently with deteriorating systolic and diastolic left ventricular functions.
- The left atrial booster function (ASr) was negatively correlated with the LVEF and positively correlated to the left ventricular filling parameter (E/Em) and to the LAEF suggesting a compensatory response of the LA in STEMI patients.
- The peak systolic strain of left atrium in STEMI patients treated by primary percutanous interventions was higher than those treated by thrombolytic therapy.
- The left atrial peak systolic strain was not related to the site of infarction nor to the number of stenotic coronary arteries in STEMI patients.