

Results: Male female ratio was 4:1 and age group (24-58y). The mean ESD was 63 (56-78) Mean EDD was 50 (46-54). However at the mean ESD of 50 mm the EDD was quiet variable between (58-72mm). We observed EDD to scatter much from the mean and ESD showed less variation. The LVEF was linearly correlating with both EDD and ESD. Severe LV dysfunction (< 25 %) was more related to larger ESDs. When mitral regurgitation was present ESD were better correlated with LV dysfunction. The impact of intensive medical management on LV dimension was also studied in 5 patients. The regression of EDD was more rapid than ESD .An empirical reduction > 5mm in ESD was considered significant. It was achieved only in two, while EDD regression was documented in all. Functional class improvement and 6 minute walk test showed a positive trend only in patients whose ESD regressed.

Conclusions: We conclude LVESD is a more scientific parameter to diagnose DCM than LVEDD. It is also a better index to assess and follow-up these patients. In our series, a mean LVESD of more than 50 mm predicted all significant DCMs .We suggest there is a need for a larger study on this issue. We may universalize the definition of DCM based on LVESD rather than LVEDD.

Is the pleotropic effect of ranolazine is due to its antioxidant action?

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Background: Ranolazine is a unique anti anginal drug with undefined mechanisms of action. In the present study we were aimed to identify the role of ranolazine in improving left ventricular function by various echocardiographic parameters and to identify its effect on a unique oxidative stress marker Malonaldehyde in patients with post myocardial infarction left ventricular dysfunction who underwent revascularization by percutaneous interventions.

Methods: Prospectively one hundred (100) cases and forty (40) controls with LV dysfunction were recruited in this study with 6 months of follow up and all baseline and demographic parameters, clinical features and symptomatology with blood chemistry parameters were collected for all patients. All patients underwent detailed echocardiography at baseline and at 6 months and serum Malonaldehyde levels were also analyzed at baseline and 6 months. The echocardiographic parameters studied were ejection fraction [(LV volume in end diastole (LVEDV) - LV volume in end systole (LVESV))/ LVEDV], fractional shortening ((LVEDd-LVESd) / LVEDd expressed as a percentage - d is dimension) and peak mitral annular velocity (degree of movement of mitral annulus during systole - PMAV) by Doppler. Malondialdehyde (MDA) is one of the most frequently used indicators of lipid peroxidation. We used plasma MDA levels to know the degree of oxidative stress. MDA concentration will be determined by using the method described by Draper and Hadley based on TBA reactivity. Normal Range of MDA is 3.60 ± 0.90 nanomole/ml . Controls received standard medical therapy alone after PCI where as cases, in addition, received 500 milligrams of Ranolazine twice a day for 6 months. All Echocardiographic parameters were reassessed after 6 months and malonaldehyde concentrations were repeated. Minitab 16 version is used for statistical analysis.

Results: Male: Female ratio was 3.4:1 in cases and in controls. The mean age of cases was 56.8 ± 9.6 yrs in cases and 55.1 ± 12 yrs in controls. Basal clinical demographic features and risk factors are

comparable between cases and controls. After 6 months of Ranolazine therapy there was a statistically significant improvement in EF{(46.1±10.5) to (48.32±9.63%)} and {(44.2±10.8) to (43.85±8.38%)} (p=0.008) by diameter and volume [(43.8±9.3) to (46.72±9.34ml)] and [(41.8±9.52) to (42.55±9.1 ml)] p=0.018) in cases and controls respectively in comparison to baseline and there was improvement in MAV [(9.5±2.1) to (9.79±1.84)] and [(8.8±1.8) to (8.65±1.8)] (p=0.001) in cases and controls respectively indicating the positive remodeling effects of drug on LV parameters Plasma MDA levels also showed improvement in cases than in controls. MDA at 6 months in cases was decreased from 3.6 nanomole/dl to 3.4 ± 0.8 nanomole/dl where as in controls increased 3.8 to 3.9 ± 0.7 nanomole/dl. Analysis of variance (ANOVA) was performed to check whether there is any significant difference between the basal values, 6 months control & with drug. It can be inferred from the study that the effect of Ranolazine on left ventricular remodeling in obstructive coronary artery disease patients is highly significant (p=0.001) and has the potential to reduce the risk considerably.

Conclusions: Peak Mitral velocities can be used to follow the cases in clinical practice to know the improvement in LV contractility like standard EF. There is significant improvement in EF in cases when compared to controls at 6 months, reconfirming the results from the PMAV analysis. There is reduction in oxidative stress levels in cases than controls at 6 months, may be possible mechanism of action of ranolazine induced positive LV remodeling.

Risk stratification of women with peripartum cardiomyopathy at initial presentation: A dobutamine stress echocardiography study

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Background: Peripartum cardiomyopathy is a rare disorder effecting women in their prime years of life. There appears to be an initial high-risk period with 25% to 50% of women dying within the first 3 months postpartum. Early risk stratification and prognostication are, thus, crucial. However, only limited data are available.

Objectives: We sought to determine the prognostic use of inotropic contractile reserve on risk stratification and prognostication of women with peripartum cardiomyopathy.

Methods: In all, 10 women (mean age 20.7 years) with peripartum cardiomyopathy and severe left ventricular (LV) dysfunction (mean LV ejection fraction [LVEF] $27.3 \pm 6.5\%$) were studied. Of these, 8 underwent dobutamine stress echocardiography at baseline and a follow-up resting echocardiogram at a mean of 3.6 ± 0.9 months after initial presentation. Resting and peak inotropic contractile reserve, and follow-up LVEF, were computed.

Results: The mean LVEF improved significantly from baseline ($27.3 \pm 6.5\%$) to maximal inotropic contractile reserve ($52.6 \pm 11.2\%$) (P = .0004) and at follow-up ($54.2 \pm 14.3\%$) (P = .006). Importantly, LVEF at maximal inotropic contractile reserve and at follow-up (3.6 months) did not differ significantly. The mean LVEF at maximal inotropic contractile reserve correlated well with the follow-up (LVEF R = 0.79). However, the baseline LVEF did not correlate with follow-up LVEF.

Conclusions: In patients presenting with peripartum cardiomyopathy, inotropic contractile reserve during dobutamine stress