Original Article

COLOUR DOPPLER IMAGING OF LV INFLOW IN NORMAL VERSUS LV DIASTOLIC DYSFUNCTION AND ITS CORRELATION WITH FINDINGS DERIVED FROM PULSE DOPPLER ESTIMATION AND LV SYSTOLIC FUNCTION

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Abstract

Aim and Background: The present study concentrates on colour flow pattern over the left ventricular (LV) inflow from the annulus to the apex and the angle that it makes towards the lateral wall of the LV, which was measured, analyzed and compared amongst various degrees of diastolic dysfunction. Most operators avail just to eyeballing colour flow pattern across the AV valve. The angles that the flow stream makes with the central direction of flow vary with different degrees of LV diastolic dysfunction (LVDD).

Materials and Methods: A Prospective Observational Study comprising of 100 patients consulting at department of Cardiology in a medical college in South India., further these patients were divided into 5 groups of normal and various degrees of LVDD (Group 1: Normal; Group 2: Grade 1; Group 3: Grade 2; Group 4: Grade 3; Group 5: Grade 4); each group having 20 patients each.

Results: Examination of LV colour flow propagation angle values, as expected, revealed maximum values noted in both LVDD patient group 4 and group 5 when compared to patients with normal LV diastolic function($P < 0.001$).**Conclusion:** In the present study we conclude that routine observation of color doppler filling pattern by M Mode might add useful information in certain grey areas of decision making as in for surgery and other interventions. Wider the deviation of color flow pattern more the seriousness of ventricular dysfunction.

Keywords: Left ventricular, Diastolic pressure, Colour Doppler.

Introduction

To maintain homeostatic cardiovascular hemodynamics it is essential to maintain normal cardiac output, venous return and appropriate filling pressures in both the ventricles, in the resting state as well as during increased physical effort; in the normal and diseased state. In the last two decades there has been increasing accumulation of evidence on diastolic function of the ventricles being as important as the systolic function in maintaining tissue perfusion and also to respond to further extraneous factors like increased preload, afterload, heart rate and myocardial performance.[1-2]

Recognition of this entity and consensus among various stakeholders revealed that progressive alteration due to various molecular lased changes in relaxation of ventricles may result in profound heart failure with preserved systolic function of the ventricles. Hence, assessment of LV diastolic function becomes a pertinent part of imaging study in the clinical evaluation of cases as well as monitoring critical care patients in any institution [3-4].

Since any investigation finding should be validated by other modality of investigation, to see whether there is diastolic dysfunction inferred by colour doppler, other methods such as conventional pulse wave doppler echo, tissue doppler imaging measurements which have proven their merit in terms of sensitivity and specificity has been measured and compared in this study.

Materials and Methods

A Prospective Observational Study comprising 100 patients; 20 patients in each arm classified according to degree of LV diastolic dysfunction (normal, Grade 1, Grade 2, Grade 3 and Grade 4) were studied in department of Cardiology at a medical college in South India. Each patient was studied with a combination of M-mode and wide angle (90°) two-dimensional echocardiography at 8, 12, and 24 cm depths using a 5 MHz transducer with GE Vivid E9 series (model BT12) advanced echo sonography machine.

Results

The study population included 100 patients, further divided into 5 groups of normal and various degrees of LVDD (Group 1 : Normal; Group 2 : Grade 1; Group 3 : Grade 2; Group 4 : Grade 3; Group 5 : Grade 4); each group having 20 patients each. Descriptively out of all the patients involved in this study percentage of male patients were 77% and female were 23% patients; the gender distribution according to degree of diastolic dysfunction is depicted in **Figure.1.** The minimum and maximum age of the individuals of this study was 22 and 80 years. The age distribution according to degree of diastolic dysfunction is depicted in **Figure.2.** In the study population 75% patients had diabetes, 60% had hypertension and 51% had dyslipidaemia as depicted in **Figure.3 and Figure.4**respectively. There was significant statistical difference in terms of age between the 4 groups of LVDD when compared to the normal (p value <0.05).

Figure 1 : Gender distribution of various grades of diastolic dysfunction.

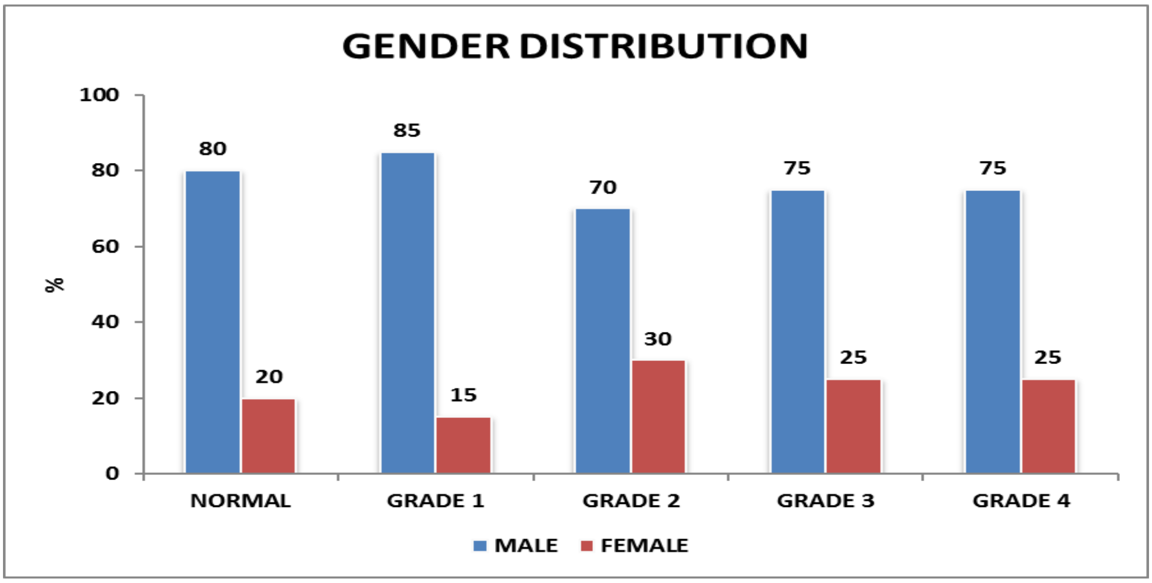


Figure 2 : Age distribution of various grades of diastolic dysfunction.

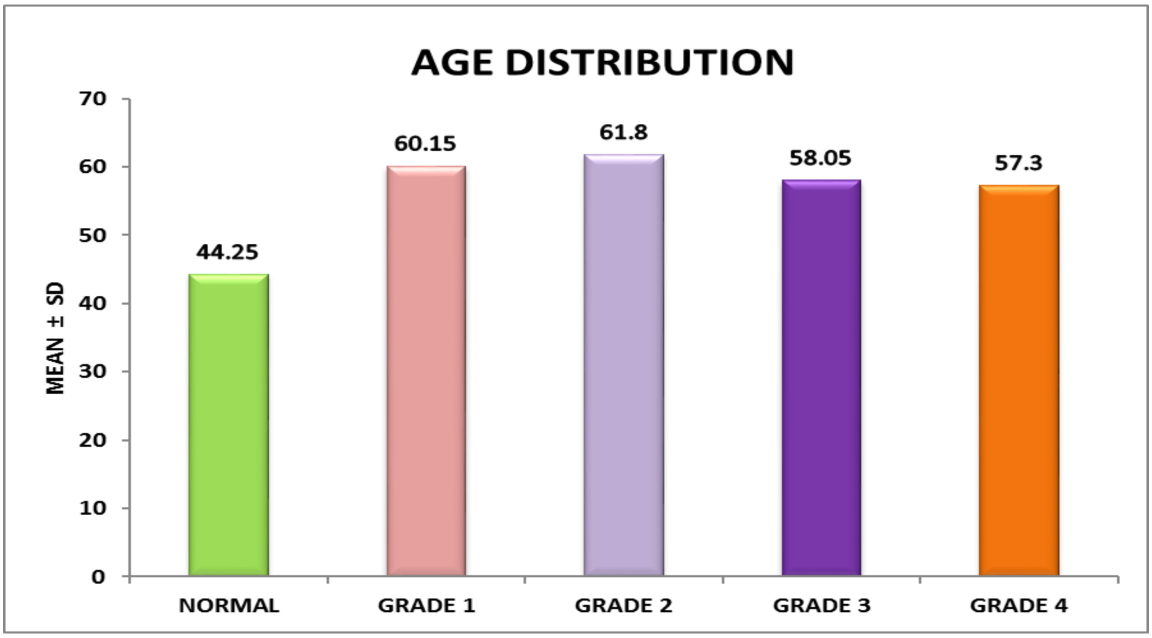


Figure 3 : Distribution of diabetics in various grades of diastolic dysfunction

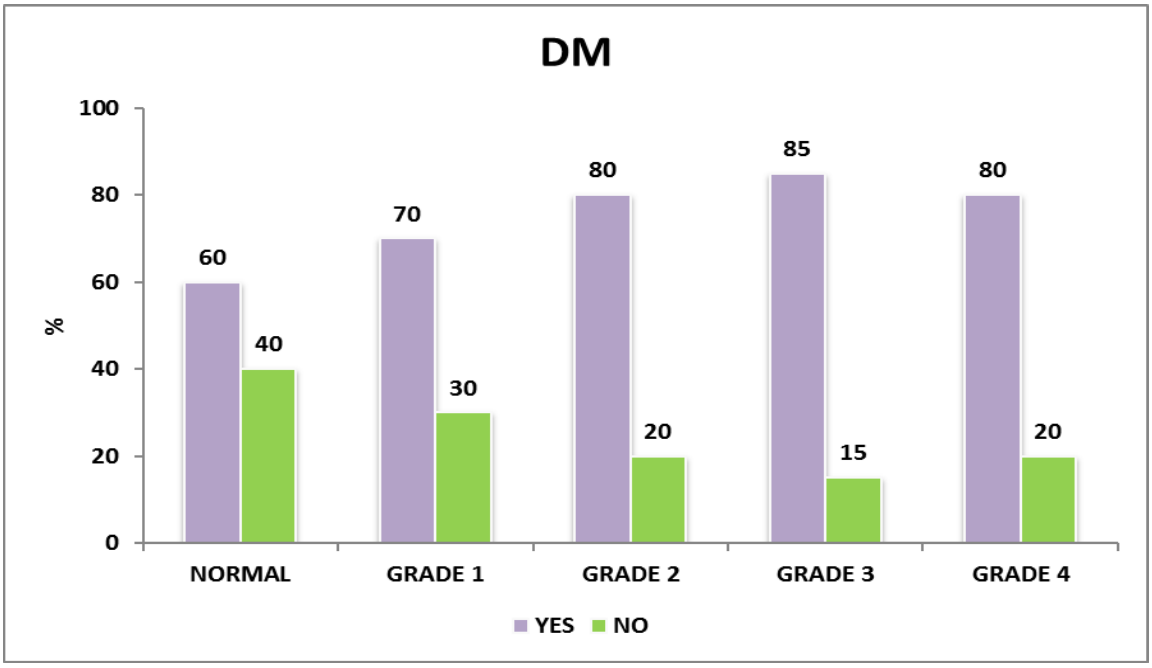
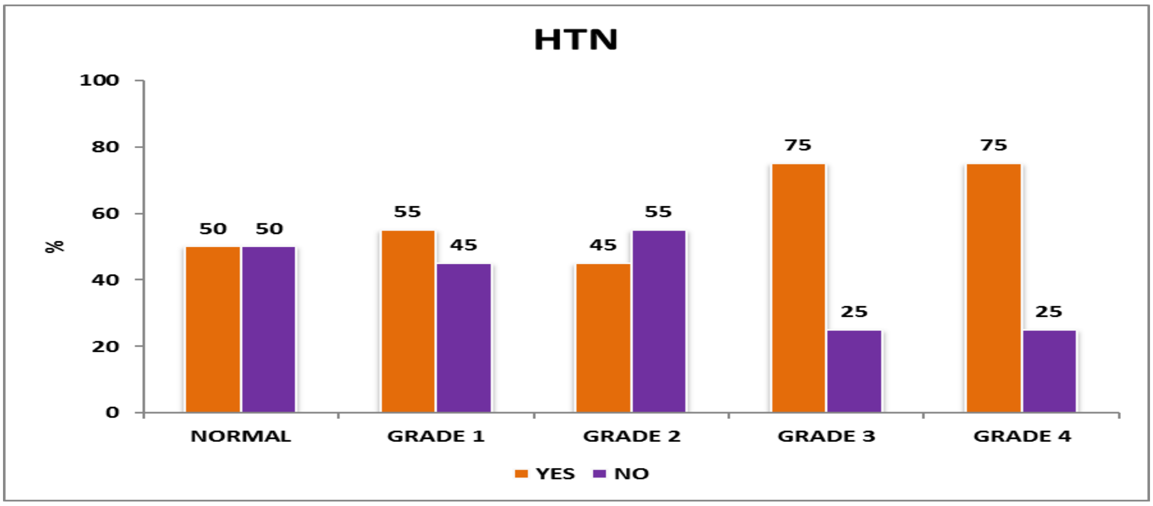


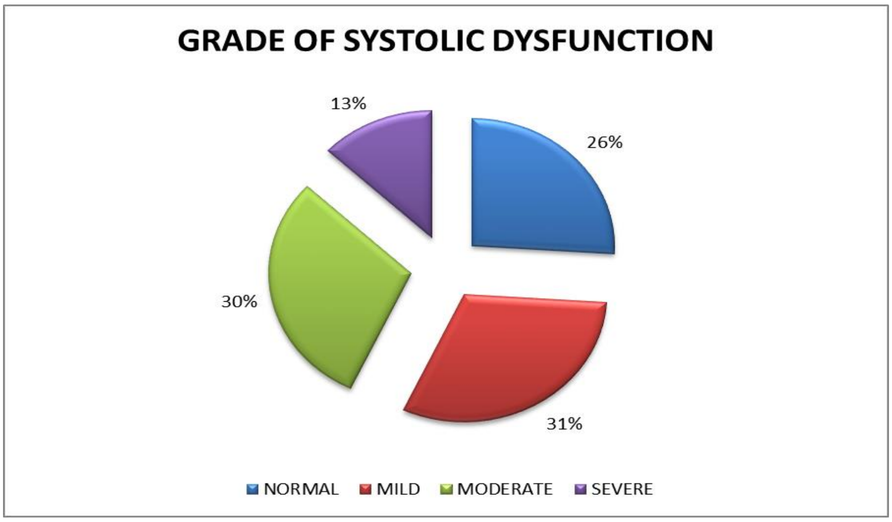
Figure 4 : Distribution of hypertensives in various grades of diastolic dysfunction



Left Ventricular Systolic Dysfunction

Left ventricular systolic dysfunction, determined by the measurement of LVEF as per Simpson's rule algorithm revealed, majority of the patients involved in this study had mild LV systolic dysfunction comprising 31% of the whole study population. Among the study population of 100 patients – 26% had normal LV systolic function; 31% had mild LV systolic function; 13% had moderate LV systolic function; and 30% had severe LV systolic function **(Figure.5).**

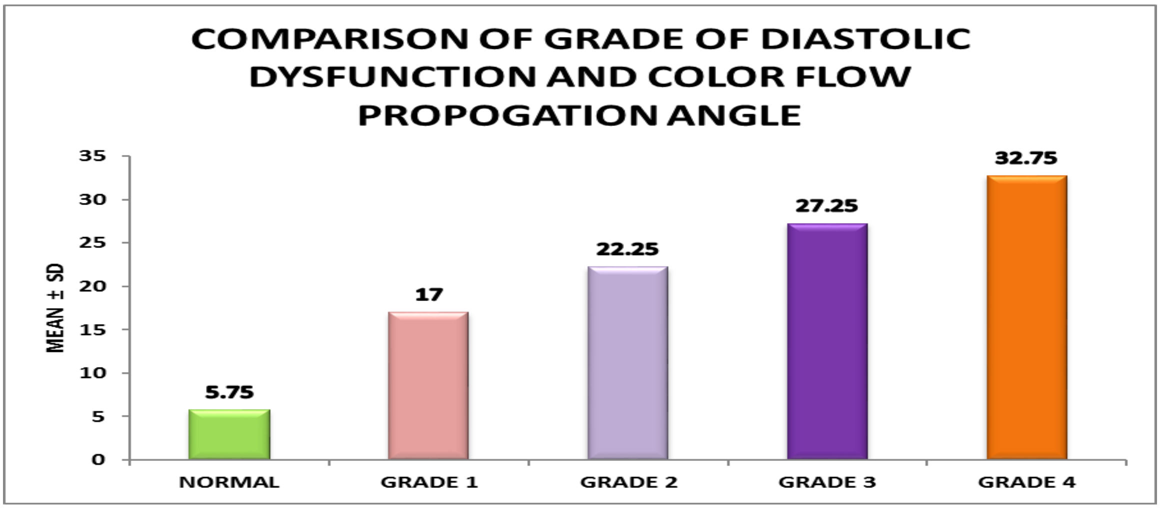
Figure 5 : Distribution of systolic dysfunction in the study population.



Colour Doppler Flow Propagation

Examination of LV colour flow propagation angle values **(Figure 6),** as expected, revealed maximum values noted in both LVDD patient group 4 (27.2 ± 4^{O}) and group 5 (32.7 ± 3^{O}) when compared to patients with normal LV diastolic function (5.75 ± 5^{O}) ($P < 0.001$).

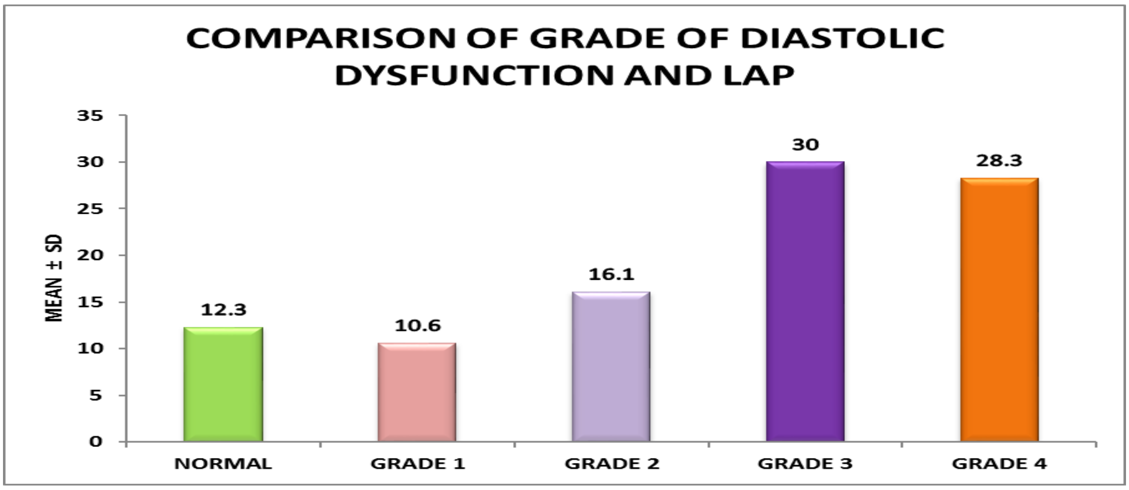
Figure 6 : Comparison of LVDD and Colour Flow Propagation Angle.



Left Atrial Pressure

In terms of LAP, the highest values were found in group 5 : 28 ± 12 mmHg and group 4 : 30 ± 10 mm Hg ($P < 0.05$) when compared to patients with normal LV diastolic function (group 1: 12 ± 2 mm Hg) which was statistically found to be significant ($P < 0.05$) **(Figure 7).**

Figure 7 : Comparison of LVDD and LAP.



Discussion

The physiology of normal left ventricular (LV) early-diastolic filling not only is regulated by indices of active relaxation and passive chamber stiffness, but also by principles of fluid dynamics [5]. Even though elastic recoil and myocardial relaxation rate determine LV early-diastolic performance, the integrity of LV synchrony and geometry is essential to maintain the effect of their timely action on early-diastolic LV filling [6]. These factors are not only prime determinants of LV pressure decay during isovolumic relaxation and immediately after mitral valve opening, but they also instigate the generation of a sufficient intra ventricular pressure gradient, which enhances efficient early-diastolic LV filling [6]. Accurate non-invasive echo-Doppler assessments of LV diastolic abnormalities have important therapeutic and prognostic implications. However, at times it remains a potential challenge for many cardiologists, particularly when evaluation of LV relaxation using standard Doppler echocardiographic parameters is hindered by their preload dependency [7].

Both magnitude and vector of the pressure gradient formation are dependent on LV geometry. Specifically, apical to base negative gradient formation is enhanced in small ellipsoidal LV cavities and corresponding velocity of the propagated wave higher than in normal sized ventricles, as blood redistribution by vortex formation and propagation requires that the LV diameter sufficiently exceeds the mitral diameter [8].

In a typical color M-mode tracing while the patient is in normal sinus rhythm, the first wave propagates from the atria to the ventricular apex corresponding to early filling and a second wave follows atrial contraction. The magnitude of these velocities is highest above the valve leaflet tips and decreases as flow approaches the apex, as shown by the change in encoding color. Additionally, in normal ventricles, the spatial position of the maximal velocity is closer to the ventricular apex for the early filling wave than it is for the atrial contraction wave, suggesting that intra ventricular pressure gradients during early filling produce a suction force that accelerates flow beyond the valve orifice [9].

Color M-mode echocardiography provides a spatiotemporal map of early diastolic filling. Conventionally, this has been evaluated by calculating the slope of an isovelocity contour representing the Vp. The flow propagation is characterized by a more rapid initial slope indicating rapid flow propagation that abruptly slows after a deceleration point. In the presence of diastolic dysfunction, the initial slope is reduced and the deceleration point moves progressively closer to the mitral annulus creating an angulation of the flow stream towards the LV free wall. The product of the initial slope and the distance to the deceleration point (Vs) provides a measure of the strength of early diastolic LV suction and may provide a better measure of diastolic function than the conventional Vp. Under normal circumstances, early diastolic filling results from a progressive pressure gradient from the left atrium [10] that extends most of the way to the LV apex. This results in a rapid initial Vp that extends 3.1 ± 0.7 cm from the mitral annulus toward the LV apex. With diastolic dysfunction, the magnitude of the pressure gradient is reduced, and it does not extend as deeply into the left ventricle [11]. Thus, it was observed that with worsening diastolic dysfunction, the initial Vp is reduced and that filling wave decelerates to a lower velocity closer to the mitral annulus leading to more angulation of the flow stream. After termination of the pressure gradient, the terminal Vp is reduced to similar levels in all subjects regardless of diastolic function, hence creating a 'sickling effect' to the flow flame.

The angle of propagation of peak early filling flow was increased in both groups 4 and 5. Although the E/A ratio in group 3 was not different from that in group 1 because of pseudonormalization, the angle of propagation was significantly higher in group 3. In the normal state of left ventricular diastolic filling as seen in group 1, rapid left ventricular relaxation generates a dynamic pressure gradient, initially at the mitral orifice. The intra ventricular minimal pressure does not increase significantly, and the pressure gradient is still maintained in the mid-left ventricle during early diastole. According to the Bernoulli equation, this pressure gradient generates the driving force to fill the blood volume deep into the left ventricle. Thus, the peak left ventricular filling can be rapidly propagated sequentially from the mitral orifice toward the left ventricle.

Generally, in the state of diastolic dysfunction, the trans mitral pressure gradient diminished by the impaired relaxation process results in a decreased early trans mitral flow velocity and greater angulation of the propagated flow stream that increases with grade of diastolic dysfunction, almost drawing a 'sickle' shaped pattern of blood flow towards the LV apex.

The early diastolic intra ventricular pressure gradient is lost because of rapidly increased left ventricular minimal pressure. Such a damped intra ventricular pressure gradient may lead to slower propagation of early filling flow and greater angulation towards the LV free wall. The state of pseudonormalized trans mitral flow, as seen in group 3, is characterized by concealed relaxation abnormalities because of the increased early trans mitral velocity that is caused by an elevated trans mitral pressure gradient [12]. In the setting of severely reduced left ventricular distensibility, ventricular pressure increases immediately after the filling of a small amount of the blood volume into the left ventricle.

Thus, the large atrio ventricular pressure difference decays rapidly, and the driving force of the ventricular filling may be stalled at the near mitral orifice [12]. Consequently, the filling flow propagation is rapidly attenuated in spite of increased early trans mitral velocity.

In this study it was noted that the flow propagation angle progressively increased as the degree of LVDD worsened. The propagation angle in group 5 was significantly increased when compared with group 1, suggesting that left ventricular filling in patients was increasingly difficult as severity of LVDD advanced.

It is interesting to see the correlation between LVDD and inherent increase in LAP**,** as the intraventricular pressure gradient from base to apex dwindles progressively. The velocity of opening of mitral valve progressively decreases to keep up the grossly deteriorated cardiac output.This observation is further strengthened with the evidence of deteriorating LVEF, doppler derived LV diastolic function and progressive lateralization of color doppler spectral display towards a "sickle" shaped pattern ($p < 0.05$).

The other correlating factor is that ESV showed a linear increase as the doppler derived diastolic function deteriorated, as depicted in **Figure 7.** Which in turn also correlates with lateralization made by colour doppler display pattern consistent with increase in ESV, preventing flow towards apex.

Even though there is a close correlation between ESV, EDV and LVDD the difference is not statistically significant when it comes to comparison of grade 3 and grade 4. But the pattern of color doppler does differ between grade 3 and grade 4, since color doppler imaging is based upon the mean velocity of flow between base to apex which is gradually decreasing.

The color flow makes an increasing angle from the straight line from base to apex as both ESV and EDV keeps increasing. Hence this observation of gross deviation to the lateral wall of LV by the color flow propagation may be termed ominous, indicating very precarious faltering LV function, which in turn determines long term survival, post intervention morbidity, mortality and death due to arrhythmogenicity.

Limitations of the present study includes - first, this was aobservational study; however, the main goal was attained. Second, a small number of patients were included for analysis. However, the main purpose of this study was to determine if there were any difference in flow propagation in patients with various grades of LVDD when compared to patients with normal LV diastolic function and this was demonstrated. Third, even though color M mode is only known to assess parameters in two dimensions and at first glance might be considered inadequate to describe diastolic flows, as these flows occur as vortices and consequently would be better assessed using a three-dimensional mode; the intent of this work was to evaluate the influence of diastolic dysfunction on intraventricular flow propagation. Future studies may determine the relationship that might exist between flow propagation angle, LV cavity size and residual volume in diastole in LVDD. Fourth, lack of invasive hemodynamic data might be considered as a limitation. However, the main study goal was met. It can also be argued that we lack sequential echocardiographic studies on LVDD patients to validate the utility of flow propagation angle findings over time. Finally, some might argue that intra observer as well as inter observer variability were not considered, we do acknowledge the potential limitation of using color M mode to study diastolic flows.

The main goal of this study was to bring forth utility value of various modalities of assessing diastolic function. Even though there has been a plethora of information about the sinister effect of progressive increase in ESV, EDV on doppler derived diastolic filling pattern, so far there has been no systematic study of the correlation between LV volume changes and associated peculiar filling pattern of LV as exhibited by M Mode derived colour doppler imaging in LVDD.

In short color doppler imaging by eye balling can be considered as a ready reckoner for the magnitude of LV function deterioration which in turn ultimately determines prognosis, long term survival and quality of life with or without intervention.

Conclusion

The present study was concentrated on color doppler flow spectral display from base to apex, which showed increase in lateralization as grade of heart failure increased, resulting in a “sickle” pattern of flow propagation. This study endorses the idea that careful examination of color flow quality gives an estimate of progressive deterioration in LV filling and stroke volume. In the present study it was concluded that routine observation of color doppler filling pattern by M Mode might add useful information in certain grey areas of decision making as in for surgery and other interventions. Wider the deviation of color flow pattern more the seriousness of ventricular dysfunction.

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