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ASSESSMENT OF LA CARDIOPATHY IN HFPEF PATIENTS USING ECHOCARDIOGRAPHY AND CMR.

Cardiology

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ABSTRACT

Background: LA dysfunction is associated with the development of HFpEF. Studies on HF have focused on ventricular remodeling, and less emphasis has been placed on atrial structural and functional changes. In this study we characterized HFpEF patients into clinical phenotypes and used newer modality of echocardiography with LA strain analysis and CMR for assessment of LA function in HFpEF patients. Aims: To study prevalence of LA dysfunction in patients of HFpEF. **Methods:** In a single hospital based cross sectional study we studied 64 patients diagnosed with HFpEF. All patients underwent echocardiography with LA strain analysis and CMR. **Results:** Hypertension was the most common phenotype. Echo and CMR based LA dysfunction was seen in 36 (56.25%) and 37(57.81%) patients respectively. LASr (reservoir %), LAScd (conduit%) and LASct (contractile %) was reduced in 57(89.06%), 52(81.25%) and 49(76.56%) patients respectively. LA dysfunction by Echo and CMR was present in majority of patients, in 4th quartile and in 3rd quartile of NT-pro BNP, also patients with reduced LA strain were in 4th quartile of NT-pro BNP and were comparable with severity of HFpEF. LAVI maximum and LAEFI by both Echo and CMR were compared and there was no significant difference in LAVI maximum by Echo and CMR with p value 0.0622. LAEFI by Echo and CMR had significant difference and LAEFI by Echo was over estimated compared to the LAEFI by CMR with p value 0.0003. **Conclusions:** Study highlights that dysfunction of the LA may be considered as an initial mechanism of underlying LA cardiopathy and an indicator of more advanced HFpEF.

KEYWORDS

LA left atria, LAS left atrial strain, LAVI left atrial volume indexed, EF ejection fraction.

INTRODUCTION

HFpEF is LVEF ≥50% corroborated by either elevated natriuretic peptide levels or other evidence of congestion [1]. Common phenotypes, hypertension, Aging, obesity, pulmonary hypertension (PH), and coronary artery disease (CAD) affect HFpEF presentation and progression [2]. LA has a crucial role in LV filling and global heart performance and has a dynamic interaction with ventricular diastole and systole. The performance of both the reservoir and conduit phases is determined by atrial compliance, ventricular relaxation, and trans-mitral pressure gradient. Recent evidence suggests that LA function and remodeling are independently associated with, or may precede, the onset of HF in an asymptomatic healthy population. Up to 45% of patients presenting with new-onset HF symptoms have LA dysfunction as the underlying mechanism, suggesting that LA failure is an early driver in HFpEF and a crucial pathogenic factor [3]. In HFpEF patients, atrial fibrillation and loss of atrial function have been related to worse clinical outcomes, and atrial strain analysis has been used to study LA function [4]. LA function could be impaired in HFpEF, and this impairment could be at least, in part, responsible for the development of clinical symptoms in these patients.

METHODS

In a single hospital based cross sectional study 64 patients Aged ≥ 18 yrs and diagnosed with HFpEF by HFA-PEFF diagnostic algorithm [5] were studied. Patients underwent routine evaluation like ECG, CBC, RFT, NT- pro BNP and were subjected to echocardiography with Speckle-tracking [Equipment- PHILLIPS EPIQ CVx (Hardware version B.1)] and CMR [Equipment- Magneton Avanto Fit 1.5 T (Siemens Inc. Erlangen, Germany)]. **Measurements by echo: (A)** LA volume index: i. Pre Atrial Contraction Volume (LAVpreA): measured at the onset of the P wave on an ECG; ii. Minimal LA volume (LAVmin): measured at the closure of the mitral valve in end-diastole; and iii. Maximal LA volume (LAVmax): measured just before the opening of the mitral valve in end-systole**. (B)** LA EF: (LAVmax-LAVmin/ LAVmax) x100. All volumes were indexed to body surface area (BSA) and expressed in mL/m2. **(C)** LA function was also analysed using strain derived from speckle tracking using dedicated Tomtec software. LA longitudinal deformation was quantified and averaged for six LA segments from the apical 4 chamber view. Strain corresponding to atrial reservoir function [LASr (%)], strain during early diastole reflects atrial conduit function [LAScd (%)] and strain during late diastole reflects atrial contractile function [LASct (%)] were assessed**. Measurements by CMR**: Cine images in 3 planes, Black blood, Flow and Late Gad examination. The sequences included the axial Cine over LA region as well as LV. Retrospective ECG-gated cine imaging of the heart was done in 3 planes, namely short-axis (SA), vertical long axis, and horizontal long axis (4 chamber view). LA volume was measured by the biplane area method on 2CH and 4CH cine images. The LA appendage was included, however, pulmonary veins were excluded. LA Volume = 0.85XA1XA2/L (AVG), A1 and A2 are the areas of LA in the 2CH and 4CH respectively and L corresponds to the average linear measurement in both 4CH and 2CH views. Max LA volume, Min LA volume, Pre-atrial contraction LA volume were calculated. Total LAEF : (LAVmax-LAVmin)/LAVmax was calculated. Bland Altman plot was used for comparing LAVI max and LAEFI by echo and CMR. Patient with HFrEF and HFmEF, severe COPD, valvular heart disease, constrictive pericarditis, high output failure, any contraindication to MRI were excluded.

RESULTS:

All patients had raised NT-pro BNP with mean value of NT-pro BNP being 1687.88 ± 2806.52. Patients were characterized into following clinical phenotypes, 7(10.94%) patients were obese, 12 (18.75%) had atrial fibrillation. Hypertension, diabetes mellitus and smoking was present in 17(26.56%), 13(20.31%), 15(23.44%) patients respectively. Patients with multiple clinical phenotypes were having more LA dysfunction and reduced LA strain values. Based on the LA parameters, LA dysfunction was defined as either increased LA max volume or reduced global LAEF. LA dysfunction by Echo was seen in 36 (56.25%) out of 64 patients. 57(89.06%), 52(81.25%), 49(76.56%) patients had reduced LASr, LAScd and LASct respectively. LA dysfunction by CMR was found in 37(57.81%) patients out of 64 patients (Table1). NT-pro BNP was divided into 4 quartiles with 16 patients in each quartile.

Patients with multiple clinical phenotypes were having higher value of NT-pro BNP. LA dysfunction by Echo was present in 11 (68.75%) patients in 4th quartile of NT-pro BNP. 14 (87.50%), 14 (87.50%), 13 (81.25%) patients had reduced LASr, LAScd, LASct (%) in 4th quartile of NT-pro BNP and was comparable with severity of HFpEF. LA dysfunction by CMR was present in 9 (56.25%) patients in 4th quartile of NT-pro BNP, however majority of patients 12 (75%) were in

3rd quartile of NT-pro BNP and was comparable with severity of HFpEF. LAVI maximum and LAEFI by both Echo and CMR were compared. Mean value of LAVI maximum by both ECHO and CMR was -1.9222 with 95% CI of -3.9456 to 0.101, suggesting that there was no significant difference in the value of LAVI maximum by Echo and CMR with p value 0.0622 (Figure 1). Mean value of LAEFI by both ECHO and CMR was -4.1431 with 95% CI of -6.2900 to -1.9962,1 suggesting that there was significant difference in the value of LAEFI by Echo and CMR and LAEFI by Echo was over estimated compared to the LAEFI by CMR with p value 0.0003 (Figure 2).

Table 1

Echo and CMR findings	Frequency	Percentage (%)
Echo Findings		
LA function		
Normal	28	43.75%
Any one abnormal (Reduced LAEF or increased LAVI max)	28	43.75%
Both abnormal (Reduced LAEF and increased LAVI max)	8	12.50%
LA dysfunction	36	56.25%
LASr (reservoir strain %)		
Normal	7	10.94%
Abnormal (reduced)	57	89.06%
LAScd (conduit strain %)		
Normal	12	18.75%
Abnormal (reduced)	52	81.25%
LASct (contractile strain %)		
Normal	15	23.44%
Abnormal (reduced)	49	76.56%
CMR Findings		
LA function		
Normal	27	42.19%
Any one abnormal (Reduced LAEF or increased LAVI max)	32	50.00%
Both abnormal(Reduced LAEF and increased LAVI max)	5	7.81%
LA dysfunction	37	57.81%

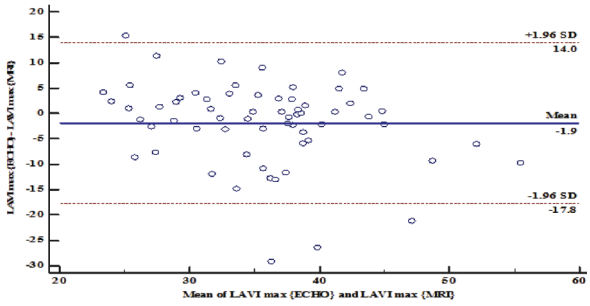

Figure 1:-Bland Altman plot comparing ECHO LAVI maximum and CMR LAVI maximum.

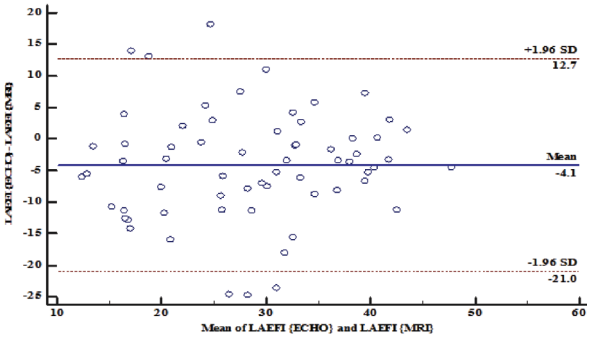

Figure 2:- Bland Altman plot comparing ECHO LAEFI and CMR LAEFI

DISCUSSION

Characterization of various clinical phenotypes were studied. Hypertension was the most common clinical phenotype. LA dysfunction by Echo, was seen in 36 (56.25%) out of 64 patients. Out of these 8 (12.50%) patients had both reduced LAEF and increased LAVI maximum and 28 (43.75%) patients were having reduced LAEF or increased LAVI maximum. This subset of study patients highlights that impaired LA reservoir function (measured by LAEF) does not necessarily associate with LA volume enlargement as also demonstrated by Bao L et al, 2022. It may occur independently of LA enlargement. Previously LA size was utilised, now the role of LA function as a marker is increasingly being evaluated, both independently and in combination with LA size. Strain analysis has also been utilised for evaluation of LA function. Impairments in LA strain has shown to be the most robust imaging markers distinguishing HFpEF from noncardiac causes of dyspnea. In this study, LASr, LAScd and LASct was reduced in most of the patients, 57(89.06%), 52(81.25%), 49(76.56%) respectively. CMR based LA dysfunction was found in 37(57.81%) patients. however, any one of the value of LAEF or LAVI maximum was abnormal in 32(50.00%) of patients and in only 5 (7.81%) patients both value of LAEF and LAVI maximum were abnormal out of 64 patients. This subset of study patients highlights that impaired LA reservoir function (measured by LAEF) does not necessarily associate with LA volume enlargement as also demonstrated by Santos et al 2019. It may occur independently of LA enlargement. Also study showed that NT-pro BNP was comparable with severity of HFpEF.

CONCLUSION:

LA has an important role in modulating LV filling. Most studies in HF have focused on ventricular remodeling, and much less emphasis has been placed on atrial structural and functional changes. This study highlights that dysfunction of the left atrium may be considered as an initial mechanism of underlying LA cardiopathy and an indicator of more advanced HFpEF.

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