ORIGINAL ARTICLE

Pattern of left ventricular hypertrophy seen on transthoracic echo in patients with hypertensive cardiomyopathy when compared with idiopathic hypertrophic cardiomyopathy

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Abstract

Objective: To explore the pattern of left ventricular hypertrophy caused by hypertension and to compare it with idiopathic hypertrophiccardiomyopathy.

Methods: The retrospective study was conducted at the echocardiography lab of Rashid Hospital, Dubai, from January 2009 to January 2010. Cases of 11 patients with significant left ventricular hypertrophy (septum >15mm) due to underlying hypertension were analysed and compared with 11 cases of idiopathic hypertrophic cardiography (septum >15mm) to assess the two groups with similar baseline echocardiographic features. Minitab software was used for statistical analysis.

Results: Although the pattern of hypertrophy in hypertensive patients was more concentric (n=5; 45%), there was also asymmetrical septal hypertrophy in 4 (36%) cases, particularly the elderly with sigmoid shape septum. There was evidence of resting mid-cavity gradient due to reduced left ventricular end-systolic diametre in 4 (36%) cases.

Conclusion: Although the equation between hypertension and left ventricular hypertrophy is more concentric, but it can be associated with left ventricular outflow tract obstruction and significant mid-cavity gradients similar to that seen in idiopathic hypertrophic cardiomyopathy.

Keywords: Hypertrophic cardiomyopathy, Hypertension, Left ventricular hypertrophy, Left ventricular outflow tract.
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Introduction

Hypertension (HTN) is a common disease that affects millions of people worldwide. The echocardiographic findings of left ventricular hypertrophy (LVH) are usually seen and considered benign. It may vary from mild to severe LVH. It may be associated with mild to severe mitral regurgitation with or without impaired systolic function. Hypertrophic cardiomyopathy (HCM) is an idiopathic condition inherited as autosomal dominant disorder. It is defined as asymmetrical septal hypertrophy of > 15mm, systolic motion of anterior mitral leaflet with or without dynamic outflow tract obstruction in the absence of other systemic or cardiac conditions leading to left ventricular hypertrophy. One of the best modalities to see the LVH pattern is 2-D echocardiography which also assesses Doppler measurments and determines the severity of the disease.[1-3] The patients may be associated with symptoms of chest pain, palpitations or shortness of breadth. Hence, complete echocardiographic assesment helps to determine the future course of action for both the groups. Idiopathic HCM is a rare disorder while hypertension is a common disease, but few studies have compared hypertensive heart with HCM heart and the data is limited. This study was planned to fill the gap to whatever extent possible.

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Patients and Methods

The retrospective study was conducted at Rashid Hospital, Dubai, between January 2009 and January 2010. It involved 11 patients each of with hypertension and HCM who were referred to the echo lab of Rashid Hospital, Dubai, for routine transthoracic echocardiography. The diagnosis of HCM was established on the echocardiographic finding of septal wall thickness of > 15mm and septal-to-posterior wall ratio of > 1.4 in the absence of HTN or aortic valve disease causing LVH.

Although a number of patients with systemic HTN were found to have LVH with septal wall thickness greater than 12mm, but we selected a group of 11 patients with significant LVH defined by interventricular septal thickness of $\geq$ 15mm due to underlying HTN so as to have a comparative group with similar baseline septal wall thickness as seen in HCM.

The echocardiographic views were seen on a commercially available echo machine. The parasternal long axis, short axis and m-mode was seen in all patients. The septal wall thickness, posterior wall thickness, left ventricular diastolic and systolic dimensions and septal-

to-posterior wall ratio was checked. The Doppler velocities were checked for colour, pulse wave and continuous wave to assess for diastolic function and mid-cavity or outflow tract obstruction. Hospital records were reviewed to obtain demographic data, symptoms, New York Heart Association (NYHA) functional class, medications and follow-up of the patients. Echocardiographic data, including 2-D, m-mode, colour flow and Doppler findings, were reviewed by a cardiologist.

The confidentiality of the records was maintained as per the hospital policy.

Minitab software was used for statistical analysis. Continous variables were given as mean±SD. The non-parametric Mann-Whitney test was used to assess the distribution between the groups. Correlation between the variables was performed by Kindall's rank correlation test. The results were found at 95% confidence interval and p-value of <0.05 was considered significant.

Results

Demographic features, symptoms and electrocardiogram (ECG) changes were noted (Table-1).

Echocardiographic features (Table-2) showed that patients with hypertension had more concentric LVH, while patients with HCM showed more asymmetrical pattern. The left ventricular end-systolic diameter (LVESD) was reduced significantly in both the HTN related LVH and HCM groups (25.1±5.1mm and 25.8±5.1mm, respectively). The interventricular septal thickness was assessed as an independent variable in both the groups, but was not found to be statistically significant to reject the null hypothesis, suggesting that the intraventricular septum (IVS) is significantly thickened in both the groups. When tested for correlation between the variables, significant correlation was found between the ESD and IVS thickness ($p<0.05$) in both the groups, meaning that as the thickness increases, the ESD decreases . There was resting mid-cavity gradient of 30 mmHg in 4 (36%) cases, and left ventricular outflow tract (LVOT) gradient >30 mmHg in 2 (18%) cases of HTN-LVH, while HCM cases showed a resting LVOT gradient of >30mmHg in 3 (27%) cases. Both the groups showed abnormal diastolic function characterised by prolonged deceleration time and reversed early to late diatolic filling ratio (E/A). Mitral regurgitation was mild in both the groups. All the cases in the HCM group were treated medically except for one case that was referred for myomectomy to alleviate LVOT gradient.

Table-1: Patient's characteristics.

	HTN-LVH (n=11)	HCM (n=11)
Age (years):	**59 ± 9.2**	**42 ± 15.2**
Sex:		
Male	8 (73%)	9(82%)
Female	3 (27%)	2(18%)
Ethnic:		
Asian	7 (64%)	9(82%)
Arabs	4 (36%)	2(18%)
Symptoms:		
Asymptomatic	5 (45%)	3(27%)
CP	2 (18%)	3(27%)
SOB	3 (27%)	1(9%)
Palpitations	1 (9%)	4 (36%)
Past History:		
DM	5 (45%)	2(18%)
IHD	1 (9%)	3(27%)
Nil	5 (45%)	6(54%)
ECG Changes:		
LVH	8 (73%)	7(63%)
LVH without strain	3 (27%)	4(36%)

CP: chest pain, SOB: shortness of breath, HTN: hypertension, DM: diabetes mellitus, IHD: ischemic heart disease, LVH: left ventricular hypertrophy.

Table-2: Echocardiographic findings.

	HTN-LVH (n=11)	HCM (n=11)	p-value
2-D findings:			
EDD(mm)	39.1± 6.9	40.2±5.2	NS
ESD(mm)	25.7±5.1	25.8 ± 5.1	NS
IVS (d):	21.2±5.9	21.8 ±7.2	NS
PWT (d):	17.3 ±4.7	13.3± 4.0	NS
S/P Wall Ratio	1.22± 0.31	1.6 ±0.35	NS
LVEF:	56.8%±9.2	58%±7.9	NS
Doppler Findings:			
No gradient	5(45%)	6(54%)	
Resting gradient (mid-cavity) >30mmhg	4 (36%)	2(18%)	
Resting gradient (LVOT) >30mmhg	2(18%).	3(27%)	
Diastolic Dysfunction:			
Grade 1 diastolic dysfunction	9(82%)	9(82%)	
Grade 2 diastolic dysfunction	2 (18%)	2(18%)	
Mitral Regurgitation:			
Mild MR seen	7 (64%)	8(73%)	
No MR seen.	4 (36%)	3(27%)	
Pattern of Hypertrophy:			
Concentric	5(45%)	0	
ASH	4(36%)	7(64%)	
ASH +SAM	2(18%)	3(27%)	
Apico-lateral	0	1(9%)	

EDD: End-diastolic diametre; ESD: End-systolic diameter; IVS: Interventricular septum; PWT: Posterior wall thickness; LVEF: Left ventricular ejection fraction; S/P: Septal/posterior; ASH: Asymmetrical septal hypertrophy; SAM: Systolic anterior motion; LVOT: Left ventricular outflow tract.

Patients with HTN-LVH were older as compared to the patients of HCM (59±9.2 vs 42±15.2 years). The male showed dominance in both hypertensive (n=8; 73% vs n=3; 27%) and HCM group (n=9; 82% vs n=2; 18%) and majority of the patients were Asians. Patients with HTN had more symptoms of shortness of breath (n=3; 27% vs n=1; 9%) compared to those with HCM while the incidence of palpitations was more (n=4; 36% vs n=1; 9%) in HCM than in the HTN-LVH group. The ECG pattern of LVH was seen in both the groups.

Discussion

Hypertension is a common entity that affects many people. Echocardiographic features of HTN include LVH, that can vary from mild to severe LVH. Hypertrophic cardiomyopathy (HCM), on the other hand, is a genetic disorder that leads to asymmetrical septal hypertrophy, abnormal motion of the mitral leaflets and LVOT gradients in the absence of underlying cardiac or systemic disorder.

We compared the echocardiographic features of patients who developed hypertrophy of the myocardium due to underlying systemic hypertension with idiopathic HCM. The demographic data in our patients showed that males were predominantly affected in both the groups and the majority of the patients with HCM (82%) were Asians (n=9; 82%). The Arabs were significantly seen with LVH in the HTN group (n=4; 36%).

Both the groups showed septal wall thickness with median of 21.8±7.2mm in case of HCM and 21.2± 5.9mm in case of HTN -LVH. However, the posterior wall thickness was significantly more in the hypertensive group with a mean of 17.3 compared to 13.3mm. The other parameter that was found to be of importance in the hypertensive group was LVESD that showed that greater the septal wall thickness, the minimum will be the LVESD that can lead to intracavitary gradient. Studies have looked into the LV mass and its relation with maximal wall thickness, but found that maximal wall thickness did not correlate well with the LV mass in patients with HCM.[1]

The LVH pattern in the HCM group was more asymmetrical with 64% (n=7) of cases having asymmetrical septal hypertrophy. It was consistent with earlier studies reporting it to be 55-65%. However, some of them have even reported it to be 95%, particularly the upper septal hypertrophy.[2,3] In our case we didn't find concentric LVH. However, it could be seen in 5% of the cases as reported by one study.[3] In our study the apico-lateral hypertrophy was seen in 9% (n=1) of the cases in the HCM group. However, there was no case with only apico-lateral hypertrophy in the HTN group.

The LVH in the hypertensive group was more concentric (n=5; 45%) as is usually seen in the echocardiography lab. However, asymmetrical septal hypertrophy was seen in 4 (36%) cases and asymmetrical septal hypertrophy with systolic anterior motion (SAM) in 2 (18%) cases. The pathophysiology of this can be understood by a study that assessed myocardial fibrosis in the hypertensive heart and hypertrophic heart and showed that the percentage area of fibrosis was more in the septal rather than free wall in the HCM heart, but was equal in the septum and free wall in the hypertensive heart.[4] Other studies have also looked into the myocardial fiber diameter and its regional distribution in the hypertensive heart, and hypertrophic heart but did not find any significant difference between the two conditions due to change in the myocardial fiber diameter.[5]

The Doppler findings of both the groups showed abnormal diastolic function mainly as impaired relaxation with prolonged deceleration times and reversed E/A ratio. The patients with HTN-LVH showed more mid-cavity resting gradients (36% vs. 18%) and this can be attributed to the reduced LVESD due to increased septal wall thickness seen in this group.

The LVOT obstruction has been reported as predictor of worse outcome in patients with HCM.[6] Echocardiography is the best modality to assess the resting and provocable gradients. Resting gradients are seen in 25% of the cases and provocable gradients can be seen in more than half of the cases.[7] The LVOT resting gradient of > 30mmHg was similar as compared to the previous studies in the HCM group and was found in 3 (27%) of the cases while mid - cavity resting gradient of >30mmhg was found in 2 (18%) the cases.

The HTN-LVH group also showed an LVOT resting gradient of >30mmhg in 2 (18%) cases which may be due to the sigmoid shape septum seen in hypertensive patients and elderly women.[8] That it is also a bad prognostic marker in the hypertensive group is difficult to say due to other associated conditions like diabetes and ischaemic heart disease seen in this group.

The left ventricular systolic (LVS) function was slightly decreased (56.8% vs. 58%) in the HTN-LVH group compared to the HCM group and may be related to the natural progression of disease process rather than wall thickness. A study assessed different hypertrophic regions and systolic function in HCM (obstructive and non-obstructive) and HTN heart and although systolic function was markedly impaired in regions of moderate to severe hypertrophy in all groups, but did not seem to be caused by differences in wall thickness, but by the degree of

myocardial disease process.[9] The LVS function has been assessed by various techniques such as Simpson's rule, Teichholz's formula and mitral ring motion by tissue Doppler, but no single method is superior to the other. Hence, one method should be used in conjunction with the other to avoid over- or under-estimation of the systolic function.[10,11]

Patients in both the groups suffered from symptoms of chest pain and shortness of breadth. However, increased shortness of breath (27% vs. 9%) was seen in the HTN group which may be due to a combination of diastolic dysfunction and impaired systolic function. The incidence of palpitations was more (36% vs. 9%) in HCM patients and 1 (9%) case developed chronic atrial fibrillation and was advised myomectomy with pacemaker. One study has reported the incidence of atrial fibrillation in HCM patients to be 15.4%, with 6% having sustained, while 9.4% had paroxysmal atrial fibrillation. Hence, the HCM group was more prone to developing atrial arrhythmia.[12] In the hypertensive group, there was no significant arrhythmia seen except in 1 (9%) case that developed paroxysmal atrial fibrillation. The arrhythmia is associated with increased risk of stroke and has detrimental prognostic value in both the groups.

The primary limitation of the current study was its small sample size, but since it was an echocardiographic assessment of the two groups, it helped us to look for two different conditions that can have similar echocardiographic features. The second limitation is the retrospective analysis of the cases. Larger studies may be considered to see both the groups which are different in etiologies but can give rise to echocardiographically significant findings.

Conclusion

The pattern of left ventricular hypertrophy was more concentric in the hypertensive group compared to idiopathic HCM. The reduced LVESD was the most important marker to look for significant mid-cavity resting gradient. Hypertensive patients referred to echocardiography lab with significant LVH should be assessed for intra-cavitary gradients.

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