

## Original Article

# Echocardiographic Profile of Hypertrophic Cardiomyopathy – A Single-Centre, Observational study

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## Abstract:

**Key words:**  
Echocardiography,  
Hypertrophic  
Cardiomyopathy,  
Phenotype,  
Bangladesh.

**Background:** Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disease of left ventricular hypertrophy (LVH). Phenotypic expression varies widely from subclinical hypertrophy to gross asymmetric septal hypertrophy causing left ventricular outflow tract (LVOT) obstruction. On top of genetic and phenotypic heterogeneity, the prevalence of different types of HCM may have geographical, as well as, ethnic variation.

**Methods:** This observational study was carried out during 2010 to 2020 to determine the echocardiographic profile of HCM in Bangladeshi population. All patients undergoing transthoracic echocardiography (TTE) in a private consultation centre of Dhaka, Bangladesh were included. HCM was defined as the presence of a maximal end-diastolic wall thickness of  $\geq 15$  mm anywhere in the left ventricle (LV), in the absence of another cause of hypertrophy in adults. HCM was further classified according to the pattern of myocardial hypertrophy and presence or absence of LVOT, or mid-left ventricular cavity obstruction.

**Results:** Out of 76 cases, non-obstructive HCM was the commonest type (65.8%), followed by HCM causing LVOT obstruction (13.2%), HCM causing mid-LV cavity obstruction (10.5%), and the apical variety (10.5%). Asymmetric septal hypertrophy (ASH) was found in 42.1%, systolic anterior motion (SAM) of anterior mitral leaflet (AML) in 14.5%, mitral regurgitation (MR) in 50%, left ventricular systolic dysfunction in 5.3%, and raised pulmonary artery systolic pressure (PASP) in 15.8% of cases. Maximum LV wall thickness  $\geq 30$  mm was found in 66 out of 76 cases.

**Conclusion:** The study highlights the clinically useful profile of HCM in Bangladeshi population based on conventional echocardiography. Further studies involving clinical, newer echocardiographic modalities and genetic analyses are warranted to discover the additional information in this ethnicity.

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## Introduction:

Hypertrophic cardiomyopathy (HCM) is characterized predominantly by left ventricular hypertrophy (LVH) in the absence of another cardiac, systemic, or metabolic disease capable of producing the magnitude of hypertrophy evident in a given patient and for which a disease-causing sarcomere (or sarcomere-related) variant is

identified, or genetic aetiology remains unresolved.<sup>1</sup> HCM is in fact a genetic disease with an autosomal dominant pattern of inheritance and variable expressivity. Mutation of sarcomere genes result in myofiber disarray and hypertrophy. Pattern of myocardial hypertrophy varies ranging from subtle preclinical hypertrophy to hypertrophy affecting single segment, multiple segments, apical

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segments to diffuse involvement of the left ventricle mimicking concentric hypertrophy. Involvement of right ventricle, i.e., biventricular involvement has also been reported. Besides variation in phenotypic expression, natural history of HCM also varies widely ranging from benign course to chronically symptomatic one to presentation with sudden death. Presence of left ventricular outflow tract (LVOT) or mid LV cavity obstruction and gross myocardial hypertrophy of  $>30$  mm may have prognostic significance.<sup>2-4</sup> Conventionally, HCM is diagnosed and followed up by transthoracic echocardiography (TTE). But now-a-days, other imaging modalities like computed tomographic (CT) scanning, and specially, cardiac magnetic resonance (CMR) are playing increasing role in diagnosis and prognostication of HCM. Besides this, genotyping is contributing to the genetic basis of the disease, and thereby helping confirmation of the diagnosis and genetic counselling. Recognizing the genetic and phenotypic heterogeneity, the diagnostic criteria and classification of HCM are still evolving. Though probably more perfect, these classifications may not be so practical in day-to-day practice. Moreover, necessary technology and expertise may not be available widely. On the other hand, the prevalence of different types of HCM may have geographical, as well as, ethnic variation, e.g., the apical variety of HCM is commoner in Japan than in the western countries.<sup>5-7</sup>

In Bangladesh, the prevalence of different types of HCM is not known. Data regarding other characteristics of HCM are also sparse. So, the present study was planned to find out clinical types of HCM, along with their selected attributes.

### Study Methods:

This was an observational study based on echocardiography to find out the pattern of HCM in Bangladesh. The study was carried out in a private consultation centre of Dhaka City during 2010 to 2020. All patients sent for TTE were included. Echocardiographic examination was carried out by Vivid 7 and Vivid E95 (GE Healthcare, 9900 Innovation Drive, Wauwatosa, WI 53226, USA) following the recommendations of the American Society of Echocardiography (ASE). HCM was defined by echocardiography as presence of a maximal end-diastolic wall

thickness of  $\geq 15$  mm anywhere in the left ventricle, in the absence of another cause of hypertrophy in adults.<sup>1,8</sup>

Once diagnosed, HCM was further classified according to the pattern of myocardial hypertrophy and presence or absence of left ventricular outflow tract (LVOT), or mid-left ventricular cavity obstruction into 4 types: Diffuse non-obstructive, HOCM with LVOT obstruction, HOCM with mid-cavity obstruction, and the apical. Diffuse non-obstructive HCM was defined as basal septal thickness  $\geq 15$  mm, or ratio of septal thickness to thickness

of inferior wall at midventricular level  $>1.5$ , no significantly raised LVOT or mid-cavity gradient. HOCM with LVOT obstruction, and HOCM with mid-cavity obstruction were defined as basal septal thickness  $\geq 15$  mm, or ratio of septal thickness to thickness of inferior wall at midventricular level  $>1.5$ , PLUS LVOT or mid-cavity gradient  $\geq 30$  mmHg. Apical HCM was defined as apical thickness  $\geq 15$  mm, or ratio of apical LV wall thicknesses to basal LV wall thicknesses =  $1.3-1.5$ .<sup>9</sup>

### Results:

The study includes 76 HCM cases studied echocardiographically. Age range of the studied patients was 17 to 75 years, mean  $51.75 \pm 12.50$  years. Out of 76 cases, 61 were male, and 15 were female. Non-obstructive HCM affecting multiple segments was the commonest type constituting 65.8% of cases, followed by HCM causing LVOT obstruction 13.2%. HCM causing mid-LV cavity obstruction and the apical variety comprised of 10.5% each. So, non-obstructive HCM affecting multiple segments and the apical HCM, which is also non-obstructive, together constitutes 76.3% of all cases. Similarly, the HCM causing LVOT and mid-ventricular obstruction together was 23.7%. (Table I). Further analysis of other echocardiographic parameters revealed the distribution of features commonly encountered in HCM by echocardiography. Asymmetric septal hypertrophy (ASH) was found in 42.1%, systolic anterior motion (SAM) of anterior mitral leaflet (AML) in 14.5%, mitral regurgitation (MR) in 50%, LV systolic dysfunction in 5.3%, dilated left atrium (LA) in 75%, and raised pulmonary artery systolic pressure (PASP) in 15.8% of cases. (Table II). Maximum LV

wall thickness  $\geq 30$  mm was found in majority of cases, i.e., 66 out of 76. (Table III).

**Table-I**  
*Clinical pattern of hypertrophic cardiomyopathy (N=76).*

| Type of HCM                          | Frequency | Percentage |
|--------------------------------------|-----------|------------|
| Diffuse, non-obstructive             | 50        | 65.8       |
| HCM with LVOT obstruction            | 10        | 13.2       |
| HCM with mid-ventricular obstruction | 8         | 10.5       |
| Apical                               | 8         | 10.5       |
| Total                                | 76        | 100        |

HCM, hypertrophic cardiomyopathy; LVOT, left ventricular outflow tract

**Table-II**  
*Echocardiographic findings in hypertrophic cardiomyopathy (N=76).*

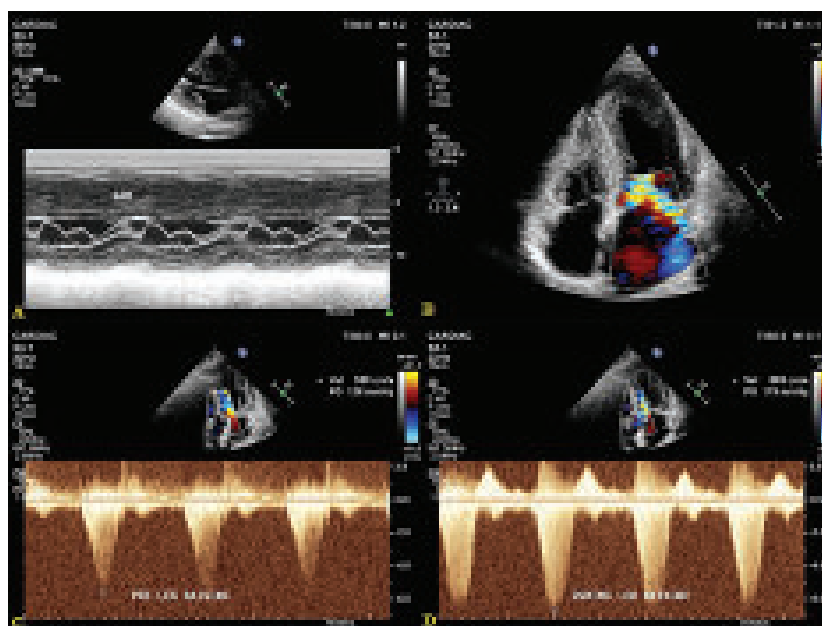
| Echocardiographic parameter | Frequency | Percentage |
|-----------------------------|-----------|------------|
| ASH                         | 32        | 42.1       |
| SAM of AML                  | 11        | 14.5       |
| MR                          | 38        | 50         |
| LV systolic dysfunction     | 4         | 5.3        |
| Dilated LA                  | 57        | 75         |
| Raised PASP                 | 12        | 15.8       |

ASH, asymmetric septal hypertrophy; SAM, systolic anterior motion; AML, anterior mitral leaflet; MR, mitral regurgitation; LV, left ventricle; LA, left atrium; PASP, pulmonary artery systolic pressure

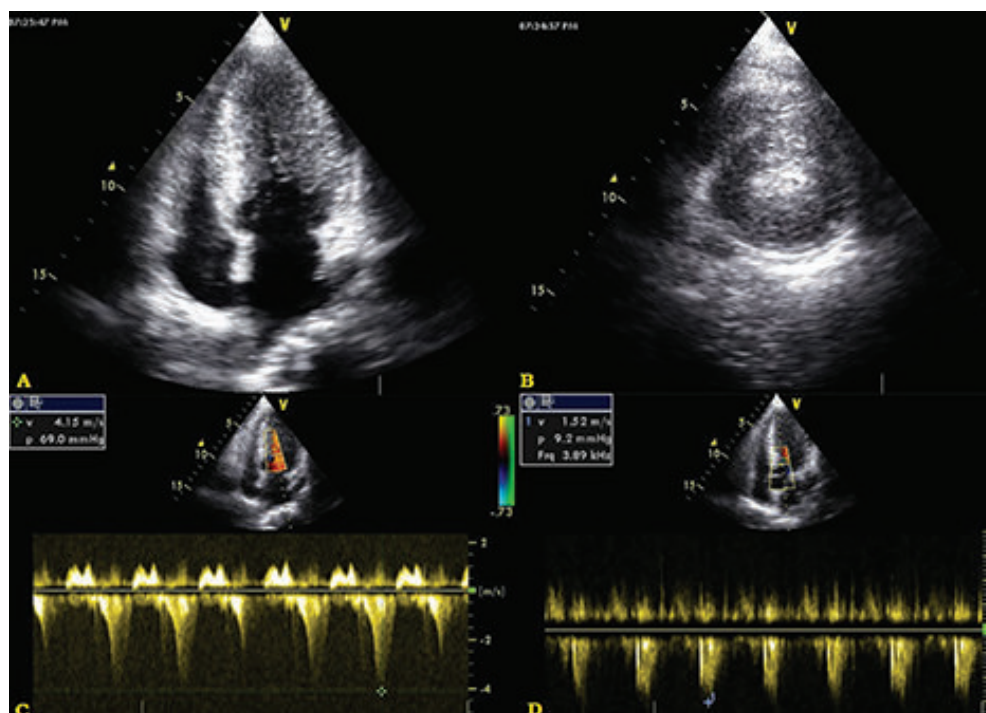
**Table-III**  
*Distribution of the HCM cases by maximum LV wall thickness (N=76).*

| Type of HCM                          | Maximum LV wall thickness <30 mm | Maximum LV wall thickness $\geq 30$ mm | Total |
|--------------------------------------|----------------------------------|--|-------|
| Diffuse, non-obstructive             | 7                                | 43                                     | 50    |
| HCM with LVOT obstruction            | 1                                | 9                                      | 10    |
| HCM with mid-ventricular obstruction | 0                                | 8                                      | 8     |
| Apical                               | 2                                | 6                                      | 8     |
| Total                                | 10                               | 66                                     | 76    |

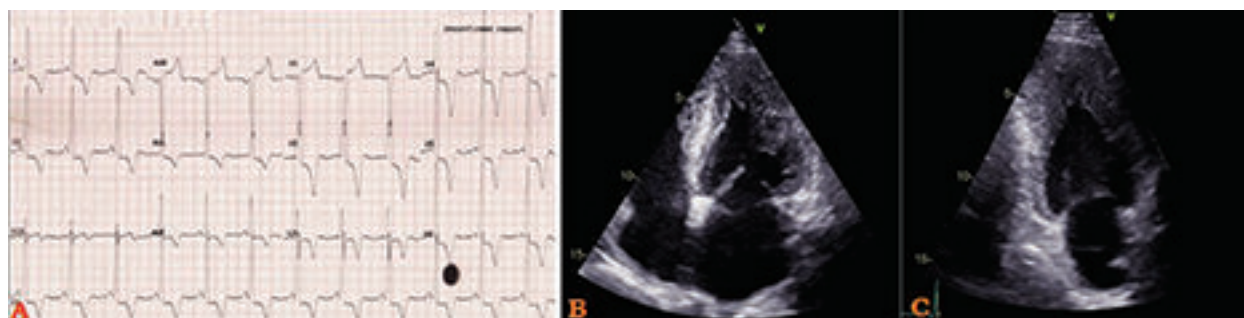
HCM, hypertrophic cardiomyopathy; LV, left ventricular; LVOT, left ventricular outflow tract



**Fig.-1:** Hypertrophic obstructive cardiomyopathy. Panel A, 2D-guided M-mode echocardiography showing asymmetric septal hypertrophy and systolic anterior motion (SAM) of anterior mitral leaflet (AML). Panel B, 2D-guided colour Doppler imaging showing moderate eccentric mitral regurgitation caused by SAM of AML. Panel C and D, 2D- and colour Doppler-guided spectral Doppler imaging showing raised left ventricular outflow tract gradient of 126 mmHg at rest which increases to 179 mmHg during leg raising.



**Fig.-2:** Hypertrophic cardiomyopathy, mid-ventricular type. Panel A and B, 2D echocardiography apical long-axis view and apical short-axis view at the level of mid-ventricle respectively showing gross hypertrophy involving the mid segments of left ventricle almost obliterating the cavity in systole. Panel C, 2D- and colour Doppler-guided spectral Doppler imaging showing raised mid left ventricular cavity peak gradient of 69 mmHg at rest. Panel D, 2D- and colour Doppler-guided spectral Doppler imaging showing normal left ventricular outflow tract peak gradient of 9.2 mmHg at rest.



**Fig.-3:** Hypertrophic cardiomyopathy, apical variety. Panel A, electrocardiography showing deeply inverted T waves in precordial leads. Panel B and C, 2D echocardiography apical 4-and 2-chamber view respectively showing gross hypertrophy involving the apical segments, as well as, the apex of left ventricle.

### Discussion:

HCM is a disease of heterogeneity in terms of clinical presentation, natural history, morphology, and the genetic substrates that characterize inherited hypertrophic cardiomyopathy.<sup>10</sup> Also, the classification of HCM is still in evolution. In the simplest form, only 2 phenotypes of HCM were identified: obstructive type (70%) and non-obstructive type (30%).<sup>11,12</sup>

HCM is popularly classified into 4 types: type I: hypertrophy involving the basal septum; type II: hypertrophy involving the whole septum; type III: hypertrophy involving septum, anterior, and anterolateral walls; type IV: LV apical hypertrophy.<sup>13</sup> In between two, Wigle ED et al. classified HCM into three functional phenotypes: subaortic obstructive, midventricular obstructive and cavity oblitative.<sup>14</sup> On the basis of septal

contour and location and extent of hypertrophy, more complex classification has been proposed: reverse curvature, sigmoidal septum, neutral contour, apical form, mid-ventricular form.<sup>15</sup> In 2006, the American Heart Association Working Group suggested that HCM should be defined genetically and not morphologically.<sup>16</sup> Subsequently, the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases recommended a morphological classification, the key point of this latter approach is that clinical evaluation of patients more often starts with the finding of a hypertrophied heart rather than a genetic mutation.<sup>17</sup>

In the present study, almost 3/4<sup>th</sup> were non-obstructive HCM (classical and apical together), while only 1/4<sup>th</sup> was obstructive type (LVOT and mid-cavity together). In the literature, presence of LVOT obstruction, either at rest or with provocation, varies widely from 25% to 75% of HCM cases.<sup>18,19</sup> The lower prevalence of obstruction seen in this study may be due to the fact that, LVOT gradient was measured at rest only. In fact, most patients with HCM do not exhibit significant resting LVOT obstruction.<sup>19</sup> On the other hand, midventricular obstructive HCM was found in 10.5% of cases. This figure is much higher than that of the reported prevalence of 1%.<sup>2</sup> In this study, the proportion of apical HCM was 10.5%. Among the different types of HCM, apical variety probably shows the highest ethnic variation in prevalence, accounting for up to 25% in Asian populations and 1% to 10% in non Asians.<sup>7,20,21</sup> Recognizing the limitations of smaller sample size of the study, it can be concluded that, apical HCM is commoner in Bangladeshis. A study involving 34 Bangladeshi adults with HCM was carried out in the Bangabandhu Sheikh Mujib Medical University classified HCM into 3 types: asymmetric septal 56%, concentric 29%, apical 15%.<sup>22</sup> The study does not provide any information regarding the obstructive and non-obstructive types of HCM.

ASH was found in less than half of the HCM cases. ASH is defined by a septal-to-posterior diastolic wall thickness ratio  $\geq 1.3$  (or  $\geq 1.5$  in hypertensive patients).<sup>23</sup> Presence of ASH is not diagnostic of HCM; sigmoid septum may commonly be confused with ASH. ASH may occur with or without LVOT obstruction. SAM of AML was found in 14.5% of

HCM patients which is lower than the reported prevalence of 30% to 60%.<sup>24</sup> Like ASH, presence of SAM is not pathognomonic for HCM. SAM of AML may also occur in LV hypertrophy due to other causes, hyperdynamic states and hypovolaemia. Mitral regurgitation (MR) was noted in half of the HCM cases. In HCM, MR may result from LVOT obstruction or primary mitral valve leaflet abnormalities. MR associated with LVOT obstruction is typically mid-to-late systolic and eccentric with posteriorly or laterally directed jet. Three-fourths of the studied patients had dilated LA, a common association with LVH and LV diastolic dysfunction. Positive association has been described between left atrial size and sudden cardiac death.<sup>25,26</sup> In 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy, left atrial diameter has been considered for risk stratification in HCM.<sup>2</sup> Dilated LA also increases the risk of atrial fibrillation. Raised PASP and LV systolic dysfunction were found in 15.8% and 5.3% of cases.

In this study, out of 76 cases of HCM, 66 had maximum LV wall thickness  $\geq 30$  mm. The severity and extent of LVH measured by transthoracic echocardiography are associated with the risk of sudden cardiac death in patients with HCM. Accordingly, extreme LV wall thicknesses of  $\geq 30$  mm has been used for risk stratification.<sup>2-4</sup>

The study has got some important limitations. This was an echocardiography-based study, lacking relevant clinical information. Only, conventional echocardiography was done – parameters related to strain imaging, 3-dimensional echocardiography and stress echocardiography are lacking. LVOT gradient was measured at rest in general, so, some cases of obstructive HCM might be classified as non-obstructive type. Left ventricular contrast echocardiography was not used, hence, some cases of apical HCM might be missed. Genetic testing might detect subclinical HCM.

### Conclusion:

HCM is a heterogenous type of cardiomyopathy. It appears to show significant ethnic and geographical diversity. The study highlights the clinically useful profile of HCM in Bangladeshi population based on conventional echocardiography. Further studies involving clinical, newer echocardiographic modalities and genetic

analyses are warranted to discover the additional information in this ethnicity.

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### Conflict of Interest - None.

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