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Coronary Arteriographic Profile in Hypertrophic Cardiomyopathy.

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ABSTRACT

Background: Hypertrophic cardiomyopathy is genetically transmitted primary cardiac disease and an important cause of morbidity and sudden death in young people, including competitive athletes. **Objectives:** The study was designed to compare the CAG findings between normal subject and hypertrophic cardiomyopathy patients who required CAG. **Methods:** HCM was diagnosed by using diagnostic criteria (clinical, electrocardiography and echocardiography) defined by Western Working group. The study was carried out on 60 subjects of which 30 had hypertrophic cardiomyopathy, 30 age and sex control (normal subjects). **Results:** In comparison of control it was observed that HCM cases had significantly larger proximal left anterior descending (3.81+-0.64 vs 2.49+-0.61 P < 0.001), proximal left circumflex (3.29+-0.46 Vs 2.39+-0.60, p < 0.001) and proximal right coronary artery (3.15+-0.47 vs 2.49+-0.42, P < 0.001). Coronary artery stenosis were found in 5 cases of HCM and among them, single vessel disease was present in 3, double vessel disease in I and triple vessel disease in I cases. **Conclusion:** Coronary artery disease (CAD) associated with hypertrophic cadiomyopathy (HCM) is a complex clinical syndrome, difficult to diagnose clinically, that can reliably be recognized by coronary arteriography.

***Keywords*:** Coronary Arteriography, Hypertrophic Cardiomyopathy.

INTRODUCTION

Hypertrophic cardiomyopathy is genetically transmitted primary cardiac disease and an important cause of morbidity and sudden death in young people, including competitive athletes. At present, however, few data exists to estimate the prevalence of this disease in large population. The prevalence of HCM appears to be about 0.2 percent on general population and 0.5 percent in primary medical practice based on identification of the disease phenotype with two-dimensional echocardiography (2D-echo) (Maron et al., 1995)[18].

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Abnormal electrocadiographic (ECG) findings are usual in early stage of HCM and this observation is, therefore, important in its early detection. Echocardiography remains the single most important diagnostic test for HCM. HCM was diagnosed by using the diagnostic criteria (echo, ECG) defined the Western Working Group (McKenna et al., 1997).

Ischemia is suspected to occur frequently in patients with HCM and may result from various mechanisms, for example, decreased coronary flow reserve, disease of small intramuscular arteries, inadequate size of coronary arteries relative to hypertrophied myocardium, diminution of coronary blood flow during systole, coronary artery spasm and coexistent atherosclerotic coronary artery disease (CAD), which can be present in up to a quarter of HCM patients above 45 years of age. Diagnosis of CAD in patients with HCM to difficult to make on clinical grounds, secondary to the high frequency of angina in patients with HCM without CAD.

Pharmacological stress echocardiography is promising but needs to be further studied; stress thallium imaging is best with frequent false positive result. At this time, coronary angiography remains the only reliable test for definitive diagnosis of coexistent CAD in HCM (Harjai et al., 1996)[9].

Kaufmanns et al. (1996)[12] found in their study that coronary artery size increases as left ventricular mass increases in HCM, but size of the coronary arteries is inappropriate with regard to left ventricular hypertrophy. Thus, the stimulus for growth of the coronary arteries is not influenced by

the underlying disease but appears to depend on the degree of left ventricular hypertrophy (Kaufman et al., 1996)[12].

Diagnostic Criteria
The proposed diagnostic criteria for establishing a diagnosis of hypertrophic cardomyopathy are as follows. The diagnosis established by the presence of one major criterion, two minor echocardiographic criteria, or one minor echocardiographic and two minor electrocardiographic criteria (McKenna et al., 1997).

Echocardiography
Major criteria
-Left ventricular wall thickness ≥13 mm in the anterior septum or posterior wall ≥ 15 mm in the posterior septum or free wall.
-Severe systolic anterior motion of the mitral valve (septal-leaflet contact).

Minor criteria
-left ventricular wall thickness 12 mm in the anterior septum or posterior wall, or of 14 mm in the posterior septum or free wall.
-Moderate systolic anterior motion of the mitral valve (no leaflet-septal contact).Redundant mitral valve leaflets.

Electrocardiography
Major criteria
-LVH plus repolarization changes (Romhilt and Estes).
-T wave inversion in leads I and aVL (≥ 3 mm), with QRS-T wave axis difference ≥ 30 degree, V3- V6 (≥ 3 mm) or II and III and aVF (≥ 5 mm)
-Abnormal Q (> 40 ms of > 25 percent R wave) in at least two leads from II, III, aVF (in absence of left anterior hemiblock), V1- V4 or I, aVL, V5- V6.

Minor criteria
-Complete bundle branch block or (minor) intraventricular conduction defect (in LV leads).
-Minor repolarization changes in left ventricular leads.
-Deep S V2 (> 25 mm). Unexplained chest pain, dysponea of syncope.

MATERIALS AND METHODS

This prospective study was carried out in the Department of Cardiology, Bangabandhu Sheikh Mujib Medical University (BSMMU) and Combined Military Hospital (CMH), Dhaka, during the period of April 2000 to November 2001. This cross-sectional prospective study was carried out on 60 subjects, of which 30 had hypertrophic cardomyopathy and 30 age and sex matched control. Informed contents were obtained from each of the study patient.

Inclusion criteria
Control (n=30)
1. Subjects presenting with chest pain, equivocal-negative exercise studies.
2. Who had no valvular or congenital heart disease of left ventricular hypertrophy.
3. Who had normal coronary arteriography.
4. Those who underwent coronary arteriography.

Case (n=30)
1. Patients presenting with unexplained chest pain, dysponea or syncope.
2. Hypertrophic cardiomyopathy diagnosed using diagnostic criteria (clinical, echocardiography, electrocardography) defined by Western Working Group (McKenna et al., 1997).
3. Patients who underwent coronary arteriography.

Exclusion criteria
Case (n=30)
1. Thickening of left ventricular wall associated with hypertension.
2. Patients with congenital heart disease.
3. Patient with valvular heart disease.
4. Known patients of coronary artery disease.
5. Patients unwilling to participate in the study.

Hypertrophic cardiomyopathy (HCM)
HCM was diagnosed by using diagnostic criteria (clinical, electrocardiography and echocardiography) defined by Western Working group (McKenna et al., 1997).

Electrocardiographic study
In all patients standard 12-lead electrosensors were recorded on the date of clinical evaluation, by using limb leads, augmented unipolar leads and unipolar chest leads from V1- V6 at a paper speed of 25 mm/ sec. The electrocadiograms were studied carefully with special reference to the points shown in the ECG diagnostic criteria defined by Western Working Group (McKenna et al., 1997).

Echocardiographic study
Two-dimensional, M-mode echocardiography with Doppler ultrasound examination were performed with ALOKA Series-5000, System-5 Gevingmed. Transducer of 3.5 MHz were used for echocardiographic studies. The echocardiograms were studied carefully with special reference to the points shown in the echo diagnostic criteria defined by Western Working Group (McKenna et al., 1997).

Procedure for coronary arteriography
Coronary arteriography and where needed left ventriculography were done in all patients by

standard Judkin's technique through femoral approach by modified Seldinger technique. All antianginal (vasoactive) medications were discontinued for 24 hours. Routine premedication consisted of oral diazepam (10 mg), with avoidance of nitroglycerine (> 2 hours). A nonionic contrast material (iopamiro-370) was used for coronary arteriography to minimize hyperemic reactions with transient changes in coronary blood flow (Hess et al., 1980). The prerequisites for CAG were followed according to hospital protocol (Deligonal et al 1995)[6 or 7].

Quantitative coronary arteriography

Quantitative evaluation of coronary arteriograms was performed with a semiautomatic computer system (Bucim et al., 1990)[5]. For each vessel segment, two to three end-diastolic measurements in different projections were carried out and averaged to correct for biologic variations in coronary artery dimensions (Suter et al., 1992)[30]. Multiangled standard views including anteroposterior (AP), left anterior oblique (LAO), LAO cranial, LAO caudal (spider) and straight left lateral for left coronary system; and right anterior oblique (RAO), LAO and RAO cranial and LAO cranial for right coronary artery were recorded for analysis.

Proximal coronary diameters of the three major coronary vessels (left anterior. left circumflex and right coronary artery) were measured in all patients by using an automatic edge detection programme. We identified vessel edges. Absolute coronary diameters were calculated by the performance of an identical quantitative programme or the angiographic catheter of known dimensions (Cordis 7 Fr, 2.33 mm) (Spears et al., 1983). Proximal coronary diameters of he left anterior descending and left circumflex arteries were defined as the vessel segment immediately beyond the bifurcation of the left main coronary artery over a length of ~ 1 cm. The computed traced this segment automatically and calculated the mean diameter over this segment. The proximal diameter of the right coronary artery was defined as the vessel segment 1-2 cm distal to the coronary ostium. A vessel segment was analyzed over a length of ~ 1 cm and the mean diameter was calculated as for the left coronary artery (Brown et al., 1997)[May be 4].

Definitions:

Normal coronary artery: Angiographically at the epicardial coronary arteries should be clearly visible and there should be no stenosis even non-significant and no irregularities of ectasis.

Diseased (obstructed) coronary artery:

Angiographically more than 50 percent narrowing of the luminal diameter of any visible coronary artery should be taken as diseased. Angiographically less than 50 percent narrowing of luminal diameter of any visible coronary artery taken as non-significant CAD. Left main coronary artery stenosis should be taken when there is stenosis of left main coronary artery. Single- vessel disease (SVD) should be taken when there is stenosis either left anterior descending (LAD) or left circumflex (LCx) or right coronary artery (RCA). Double-vessel disease (DVD) should be taken when there are stenosis of any two of three (LAD, LCx, RCA) vessels. Triple-vessel disease (TVD) should be taken when there are stenosis of all three vessels (LAD, LCx and RCA) (Deligonal et al., 1995)[6 or 7].

RESULTS

This prospective study was carried out at BSMMU and CMH, Dhaka, during the period of April, 2000 to November, 2001. A total number of 60 subjects were equally divided into control (normal coronary arteriography) and case (HCM diagnosed by criteria defined by Western Working Group, McKenna et al., 1997). All 60 subjects of this study underwent coronary arteriography.

Table 1: Characteristics of the study subjects.

Parameters	Control (n=30)	Case (n=30)
Age (years) (mean± SD)	44.35 ± 15.14	45.00± 15.38
Sex (No. /%)		
Male	27(90.0)	27(90.0)
Female	3(10.0)	3(10.0)

Table 2: Distribution of risk factors.

Risk factors	Control (n=30)		HCM with normal coronary artery (n=25)		HCM with abnormal coronary artery (n=5)	
	No.	(%)	No.	(%)	No.	(%)
Smoking	12	(40.00)	7	(28.00)	4	(80.00)
Current	10		5		4	
Past	2		2		0	
Diabetes mellitus	2	(6.67)	2	(8.00)	4	(80.00)
Dyslipidaemia	5	(16.67)	4	(16.00)	5	(100.00)
Family history of coronary artery disease (CAD)	5	(16.67)	2	(8.00)	2	(40.00)

Table 3: Comparison of risk factors between control and HCM cases with normal coronary artery.

Risk factors	Control (n=30)		HCM with normal coronary artery (n=25)		P value[a]
	No.	(%)	No.	(%)	No.
Smoking	12	(40.00)	7	(28.00)	NS
Diabetes mellitus	2	(6.67)	2	(8.00)	NS
Dyslipidaemia	5	(16.67)	4	(16.00)	NS
Family history of coronary artery disease (CAD)	5	(16.67)	2	(8.00)	NS

Table 4: Distribution of ECG parameters of the study subjects.

Parameters	Control (n=30)		Case (HCM) (n=30)	
	No.	(%)	No.	(%)
Major criteria				
LVH plus repolarization changes (Romhilt and Estes)	0		26	(86.67)
T wave inversion in leads I and aVL (≥ 3 mm), with QRS-T wave axis difference ≥ 30 degree, V3- V6 (≥ 3 mm) or II and III and aVF (≥ 5 mm)	5	(16.67)	25	(83.33)
Q (> 40 ms of > 25% R wave) in at least two leads from II, III, aVF (in absence of left anterior hemiblock), V1- V4 or I, aVL, V5- V6	1	(3.33)	5	(16.67)
Minor criteria				
Complete bundle branch block or (minor) Intraventricular conduction defect (in LV leads)	5	(16.67)	3	(10.00)
Minor repolarization in left ventricular leads	15	(50.00)	4	(13.33)
Deep S V2 (> 25 mm)	0		3	(10.00)

Table 5: Distribution of echo parameters of study subjects.

Parameters	Control (n=30)		Case (HCM) (n=30)	
	No.	(%)	No.	(%)
Major criteria				
LVH ventricular wall thickness ≥ 13 mm in the anterior septum or posterior wall, or ≥ 15 mm in the posterior septum or free wall	0		29	(96.67)
Severe systolic anterior motion of mitral valve (septal-leaflet contact)	0		6	(20.00)
Minor criteria				
Left ventricular wall thickness of 12 mm in the anterior septum or posterior wall, or of 14 mm in the posterior septum or free wall	1	(3.33)	1	(3.33)
Moderate systolic anterior motion of mitral valve (no septal-leaflet contact)	0		18	(60.00)
Redundant mitral valve leaflets	0		0	
Others:				
Intraventricular septum/left ventricular posterior wall ratio	1.04		1.64	
Mild-systolic closure of aortic valve	0		3	(10.00)
Diastolic dysfunction by Doppler echo	3	(10.00)	24	(80.00)
Ejection fraction % (mean ± SD)	58.0±5.0		72.0±6.5	

Table 6: Haemodynamic and left ventricular angiographic data.

Parameters	Control (n=12) (Mean ± SD)	Case (n=14) (Mean ± SD)	P value[a]
LVSP (mmHg)	110.0±20.0	110.0±20.0	<0.05*
LVEDP (mmHg)	8.0±3.0	21.0±8.00	<0.01*
EF (%)	60.0±4.0	70.0±8.0	<0.05*
MR	0	3	
RWMA	0	2	

Table 7: Qualitative coronary angiographic data.

Parameters	Control (n=30)		Case (HCM) (n=30)	
	No.	(%)	No.	(%)
Origin of coronary artery	Normal		Normal	
Dominant vessel				
Right	26	(86.67)	23	(76.67)

Left	3	(10.00)	4	(13.33)
Codominan	1	(3.33)	3	(10.00)
Myocardial bridging	0		1	(3.33)
Coronary artery stenosis	0		5	(16.67)
Severity of CAD among 5 cases:				
Single-vessel disease (SVD)			3	(60.00)
Double-vessel disease (DVD)			1	(20.00)
Triple-vessel disease (TVD)			1	(20.00)

Table 8: Comparison quantitative coronary arteriographic data between control and case.

Variables	Control (n=30) (Mean ± SD)	Case (HCM) (n=30) (Mean ± SD)	P value[a]
Coronary arteries dimension (mm)			
Proximal LAD	2.49±0.61	3.81±0.64	<0.001*
Proximal LCx	2.39±0.60	3.29±0.46	<0.001*
Proximal RCA	2.49±0.42	3.15±0.47	<0.001*
LAD/ LCx ratio	1.05±0.09	1.16±0.14	<0.001*
IVS/ LAD ratio	3.16±0.36	4.75±0.73	<0.001*
IVS thickness (mm)	7.70±1.37	17.97±3.12	<0.001*

DISCUSSION

Coronary arteriography remains the only reliable test to know the coronary arteiographic profile in HCM.

This cross-sectional prospective study was carried out on 60 subjects, of which 30 had hypertrophic cardiomyopathy and age 30 and sex matched control. In both the groups, 90 percent were male and 10 percent female. Mean (±SD) age were 45.00±15.38 and 44.35±15.14 years, respectively, in HCM cases and control.

Twelve (40%) of control, 7 (28%) of HCM with normal coronary artery and 4 (80%) of HCM with abnormal coronary artery were smokers. Diabetes mellitus was found in 2 (6.67%) control, 2 (8%) HCM cases with normal coronary artery and 4 (80%) HCM cases with abnormal coronary artery. Dyslipidaemia was present in 5 (16.67%) control, 4 (16%) HCM cases with normal coronary artery and 5 (100%) HCM cases with abnormal coronary artery. Family history of CAD was found in 5 (16.67%) control, 2 (8%) HCM cases with normal coronary artery and 2 (40%) HCM cases with abnormal coronary artery. Comparison of risk factors between control and HCM cases with normal coronary artery was statistically no significant. Diabetes mellitus and dyslipidaemia were more common in HCM cases with abnormal coronary artery than control (P < 0.01 and P< 0.001. respectively). Smoking and family history of CAD were not statistically significant when compared between the two groups. In HCM with abnormal coronary artery age, smoking, diabetes mellitus and dyslipidaemia were significant higher than HCM with normal coronary artery (P < 0.05, P< 0.05, P< 0.01 and P <0.001, respectively). There was no significant difference for positive family history of CAD between the two groups.

HCM cases had significantly higher left ventricular systolic pressure (130.00±32.0 vs 110.00±20.00 mmHg, P< 0.05), higher left ventricular end-diastolic pressure (21.0±8.0 vs 8.0±3.0 mmHg, p< 0.01) and more ejection fraction (70.0± 8.0 vs 60.0±4.0, P<0.05) then control. Mitral regurgitation was found in 3 regional wall motion abnormalities in 2 HCM cases.

Origin of coronary artery both in control and HCM cases were normal. Twenty-six (86.67%) right dominant, 3 (10%) left dominant and I (3.33%) co-dominant coronary vessels were among control group. Twenty-three (76.67%) right dominant, 4 (13.30%) left dominant and 3 (10%) co-dominant coronary vessels were found Hem cases. One (3.33%) of HCM cases had myocardial bridging. Coronary artery stenosis were found in 5 (16.67%) cases of HCM and among them, single vessel disease was present in 3 (60%), double vessel disease in I (20%) and triple vessel disease in I (20%) cases.

HCM cases had significantly larger coronary artery dimension than control group and among the coronary arteries, proximal LAD (3.81±0.64 vs 2.49±0.61 mm. P < 0.001), proximal LCx (3.29±0.46 vs 2.39±0.60 mm, P < 0.001) and proximal RCA (3.15±0.47 vs 2.49±0.42 mm, P < 0.001). LAD/ LCx ratio was significantly higher in HCM cases than control (1.16±0.14 vs 1.05±0.09, P < 0.001). HCM cases had significantly higher IVS/LAD ratio (4.75±0.73 vs 3.16±0.36, P< 0.001) and IVS thickness (17.97±3.12 vs 7.70±1.37, P < 0.001) than control.

CONCLUSION

Coronary artery disease (CAD) associated with hypertrophic cadiomyopathy (HCM) is a complex clinical syndrome, difficult to diagnose clinically, that can reliably be recognized by coronary arteriography. Lesion of coronary artery plays an important role in the progression from hypertrophic

cadiomyopathy to dilated cardiomyopathy. Increased coronary artery dimensions were observed in HCM but when analyzed with respect to regional ventricular thickness, these subjects demonstrated relative inadequate enlargement in coronary artery dimension.

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