Original Research Article

Clinical profile of hypertrophic Cardiomyopathy

Rajshekhar I Koujalgi

Associate Professor, Department of Medicine, Bidar Institute of Medical Sciences, Bidar, Karnataka, INDIA.
Email: rajshekharik@gmail.com

Abstract

Background: Hypertrophic cardiomyopathy (HCM) is defined by increased left ventricular (LV) wall thickness (R15 mm in one or more LV myocardial segments), that cannot be explained by abnormal loading conditions. The clinical spectrum of HCM is complex and includes a variety of phenotypes, which leads to different types of manifestations. Although most of the patients are asymptomatic, up to 25% will develop symptoms or risk of arrhythmias and sudden cardiac death (SCD). **Aims and Objectives:** To study the clinical profile of patients of Hypertrophic Cardiomyopathy reporting to a tertiary care centre situated in north Karnataka catering largely to a rural population. **Material and Methods:** The present study was conducted in the Department of Medicine of Bidar Institute of Medical Sciences., Bidar. For the purpose of study total 105 cases of Hypertrophic Cardiomyopathy reporting to the department of Medicine were selected. The diagnosis of HCM was based on 2D Echocardiographic (2D Echo) and ECG documentation. The cases include newly diagnosed and known cases also. After receiving approval from institutional ethical committee, the study was conducted. Informed written consent from all the selected study patients was obtained.Detail history of each patient was recorded on a prestructured proforma. Details regarding age, sex, address and socioeconomic status were noted. Information regarding the presenting complaints, any significant past and family history was also recorded. Complete general and systemic examination was performed in all the patients and the findings were recorded. **Results:** Mean age of patients was 49.79± 14.66years. Majority (73.33%) of the patients were male. The most common presenting complaint was angina followed by dyspnea on exertion. Past history of hypertrophic cardiomyopathy was reported by 41.90% patients. Family history of hypertrophic cardiomyopathy was observed in 9.52%. Prominent JVP 'a' wave was noted in 23.81% patients in the study.S3 heart sound was heard in 6.67% patients where as S4 was heard in 79.05% patients. Left ventricular outflow tract murmur was present in 54.29% patients and mitral regurgitation was observed in 17.14% patients of HCM. On 2D Echo inter ventricular septum thickness and posterior wall thickness in diastole was significantly more in apical HCM as compared to other HCM variants. Left atrium size was also significantly increased in HOCM (38.60±7.94). Mean left ventricular diastolic dysfunction was observed in higher grades in HCM andapical HCM as compared to HOCM and the difference observed was also statistically significant. The mean left ventricular ejection fraction was lowest in HCM and was statistically significant as compared to apical HCM and HOCM. **Conclusion:** Hypertrophic obstructive cardiomyopathy (HOCM) was the commonest cardiomyopathy in the present study. Cardiomyopathy presented with wide variation in clinical features with angina and dyspnea on exertion as the most common presenting complaint. Prominent 'a' wave of JVP and audible fourth heart sound with Left ventricular outflow tract murmur are the common clinical features associated with HCM. 2D Echo examination in HCM was the most important investigation for diagnosis and also for cardiac functional assessment.

Key Words: Hypertrophic Cardiomyopathy, angina, dyspnea, LVOT murmur.

***Address for Correspondence:**
Dr. Rajshekhar I Koujalgi, Associate Professor, Department of Medicine, Bidar Institute of Medical Sciences, Bidar, Karnataka, INDIA.
Email:rajshekharik@gmail.com

Received Date: 08/11/2017 Revised Date: 18/12/2017 Accepted Date: 03/01/2018
DOI:

Access this article online

Quick Response Code:

Website: www.medpulse.in

Accessed Date: 08 January 2018

INTRODUCTION

Although the pathology of HCM was described by two French pathologists in the mid 19th century[1,2] and by a German pathologist in the early 20th century,[3] it remained for the virtually simultaneous reports of Brock *et al*[4] and Teare *et al*[5], some 37 years ago, to bring modern attention to this fascinating entity. Hypertrophic cardiomyopathy (HCM) is the most common genetic heart disease and the leading cause of sudden cardiac death in the young and is also associated with heart failure, disability, and death at any age.[6,7,8]Hypertrophic cardiomyopathy (HCM) is

How to cite this article: Rajshekhar I Koujalgi. Clinical profile of hypertrophic Cardiomyopathy. *MedPulse International Journal of Medicine*. January 2018; 5(1): 07-12. https://www.medpulse.in/Medicine/

defined by increased left ventricular (LV) wall thickness (R15 mm in one or more LV myocardial segments), that cannot be explained by abnormal loading conditions[9].The clinical spectrum of HCMis complex and includes a variety of phenotypes, which leads to different types of manifestations. Although most of the patients are asymptomatic, up to 25% will develop symptoms or risk of arrhythmias and sudden cardiac death (SCD) [10].

MATERIAL AND METHODS

The present study was conducted in the Department of Medicine of Bidar Institute of Medical sciences., Bidar. For the purpose total 105 cases of Hypertrophic Cardiomyopathy reporting to the department of medicine were selected. The diagnosis of HCM was based on 2D echocardiographic and ECG documentation.

Patient selection

Inclusion criteria

Adults: In an adult, HCM defined by a wall thickness ≥15 mm in one or more LV myocardial segments—as measured by imaging technique echocardiography., that is not explained solely by loading conditions.

Children: An LV wall thickness more than two standard deviations greater than the predicted mean (z-score. 2, where a z-score is defined as the number of standard deviations from the population mean).

Exclusion Criteria

1. Genetic and non-genetic disorders presenting with lesser degrees of wall thickening (13–14 mm); with no family history, non-cardiac symptoms and signs, electrocardiogram (ECG) abnormalities, laboratory tests and cardiac imaging.
2. Physiological hypertrophy caused by intense athletic training.
3. In systemic hypertension, coexistence of HCM by LV thickness <25 mm and/or no LVOT obstruction with systolic anterior motion (SAM) and mitral-septal contact.
4. Patients with Isolated basal septal hypertrophy in elderly people.
5. Rheumatic heart disease or other cause of valvular aortic stenosis, pulmonary stenosis.
6. Severe pulmonary hypertension (septal hypertrophy).

The cases include newly diagnosed and known cases also. After receiving approval from institutional ethical committee, the study was conducted. Informed written consent from all the selected study patients was obtained. Detail history of each patient was recorded on a prestructured proforma. Details regarding age, sex, address and socioeconomic status were noted. Information regarding the presenting complaints, any significant past and family history was also recorded. Complete general and systemic examination was performed in all the patients and the findings were recorded.

RESULTS

Table 1: Distribution according to type of cardiomyopathy

Type	No. of patients	Percentage
AHCM	11	10.48
HCM	25	23.81
HOCM	68	64.76
Biventricular HOCM	1	0.95

In the present study of Hypertrophic cardiomyopathy, a total number of 105 patients satisfied the inclusion criteria. It was observed that hypertrophic obstructive cardiomyopathy (64.76%) was the most common type observed in the present study.

Table 2: Age distribution of patients

Age in years	HCM Apical	HCM	HOCM	biventricular HOCM	Total
≤ 17	-	01	03	-	04
18-24	-	--	1	-	01
25-39	-	06	14	-	20
40-64	05	14	43	01	63
≥ 65	06	04	07	-	17
Mean Age	**60.82±10.916*#**	**49.2±15.335**	**48.28±14.457**	**46**	**49.79±14.661**

* Statistically significant difference between HCM apical and HCM, # Statistically significant difference between HCM apical and HOCM, $ Statistically significant difference between HCM and HOCM

It was seen that age group of patients ranged from a minimum of 8 years to a maximum of 72 years. The mean age of patients of HOCM was 48.28±14.457 years, similarly in HCM with diffuse hypertrophy was 49.2±15.335 years, however Apical HCM had higher age group of 60.82±10.916 years.

Table 3: Sex distribution of patients

	Male	Female	Total	P value
Number of HCM	77 (73.3%)	28 (26.7%)	105	0.353 (NS)
Mean Age	49.0±15.303	51.96±12.732	49.79±14.661	

It was observed that, out of 105 patients 77 (73.3%) were male with mean age of 49.0±15.303 years and 28 (26.7%) were female with slightly higher mean age of 51.96±12.732 years. But the difference in sexwise age distribution not statistically significant.

Table 4: Distribution of patients according to presenting complaints

Complaints	No. of patients	%
Angina	70	66.67%
Dyspnea on exertion	66	62.86%
Palpitation	19	18.10%
Syncope	15	14.29%
Fatigue	13	12.38%
Other	5	4.76%
Asymptomatic	1	0.95%

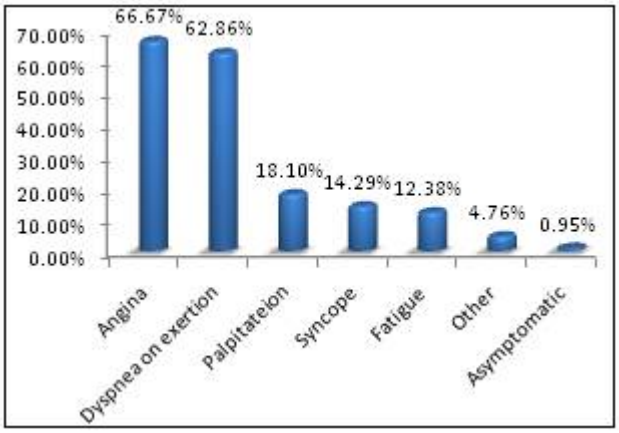

Figure 1: Distribution of patients according to presenting complaints

The most common presenting complaint was angina followed by dyspnea on exertion. Palpitation and Syncope was reported by 18.10% and 14.29% patientsrespectively. Only 1 patient had resuscitated cardiac arrest and one patient was asymptomatic. Most of the patients with Angina, Dyspnea, Palpitations had NYHA class II severity.

Table 5: Distribution of patients according to past and familyhistory

	History	No. of patients	Percentage
Past h/o	HOCM	44	41.90%
	Arrhythmia	10	9.52%
	Heart failure	1	0.95%
	HTN	23	21.90%
	DM	16	15.24%
	smokers	21	20%
	Athlete	1	0.95%
Family H/O	hypertrophic cardio myopathy	10	9.52%
	Sudden cardiac death	6	5.71%

Past history of hypertrophic cardiomyopathy was reported by 41.90% patients. 21.90% patients were known hypertensive whereas 15.24% were diabetic in the present study. Knowncases of arrhythmia were 9.52% patients.Family history of hypertrophic cardiomyopathy was observed in 9.52% whereas history of sudden cardiac death in family was given by 5.71% patients.

Table 6: Distribution of patients according to examination findings

Variable	No. of patients	Percentage
Pulse rate (mean ± SD)	84.83± 17.85	
SBP (mean ± SD)	124.71 ± 19.01	
DBP (mean ± SD)	78.37 ± 8.67	
Prominent 'a'wave of JVP	25	23.81%
S3	7	6.67%
S4	83	79.05%
LVOT	57	54.29%
MR	18	17.14%

The mean pulse rate in the study patients was 84.83± 17.85 per min. Systolic blood pressure(SBP) was 124.71 ± 19.01 mm of Hg whereas Diastolic blood pressure(DBP) was 78.37 ± 8.67 mm of Hg. Prominent JVP'a' wave was noted in 23.81% patients in the study. S3 heart sound was heard in 6.67% patients where as S4 was heard in 79.05% patients.Left ventricular outflow tract murmur was present in 54.29% patients and mitral regurgitation was observed in 17.14% patients of HCM.

Table 7: Distribution of various ECHO findings in cardiomyopathy patients

HCM type	Apical HCM	Diffuse HCM	HOCM	Biventricular HOCM	Total N
IVSd mean	1.38 ± 0.10*#	1.712 ± 0.22$	1.945 ± 0.39	1.8	
PWd mean	1.04±0.102*#	1.27±0.144	1.34±0.305	1.6	
LA Size mean	26.54±8.44#	25.48±7.89$	38.60±7.94	36	34.19 ± 9.956
Mean LV diastolic dysfunction	1.55 ± 0.82#	1.92 ± 0.4$	1.03 ± 0.24	1	
Mean LVOT Gradient(mmHg)	-	0.8±4	58.25 ± 25.48	62	-
RVOT Gradient(mmHg)	-	-	-	35	-
Atrial Arrythmia	3	6	4	-	13
Ventricular Arrythmia	-	2	4	-	06
AV block	-	1	2	-	03
non-sustained ventricular tachycardia, and	0	0	1	0	01
Mean LVEF%	**57.64±6.58**	**57.16±6.52$**	**59.97±2.93**	**56**	**59.02±4.63**

*Statistically significant difference between HCM apical and HCM, # Statistically significant difference between HCM apical and HOCM, $ Statistically significant difference between HCM and HOCM

On 2D Echo inter ventricular septum thickness and posterior wall thickness in diastole was significantly more in apical HCM as compared to HCM. Left atrium size was also significantly increased in HOCM (38.60±7.94). Mean left ventricular diastolic dysfunction was observed in higher grades in HCM and apical HCM as compared to HOCM and the difference observed was also statistically significant. The mean left ventricular ejection fraction was lowest in HCM and was statistically significant as compared to apical HCM and HOCM.

DISCUSSION

The present study was conducted in the Department of Medicine of Bidar Institute of Medical Sciences., Bidar. For the purpose of study total 105 cases of hypertrophic cardiomyopathy were selected. The cases include new as well as old cases. In the present study patients ranged from 8 years to 72 years of age. Mean age of patients was 49.79±14.66years. Majority (73.33%) of the patients were male and were belonging to lower socioeconomic class.The most common presenting complaint was angina (66.67%) followed by dyspnea (62.86%) on exertion. Palpitation and syncope was reported by 18.10% and 14.29% patients respectively. According to Braunwald E *et al*[6], Wigle ED *et al*[8] and Maron BJ *et al*[11]patients with obstructive HCM typically complain of dyspnea, angina, and presyncope and/or syncope on exertion. The severity of symptoms on upright exertion does not necessarily correlate with the magnitude of the obstructive pressure gradient measured in the supine position, which is understandable, particularly when the lability of the obstruction is taken into account. Patients with nonobstructive HCM present with these symptoms less frequently, and usually the symptoms are milder.[12]The clinical course of HCM is variable. Many patients are asymptomatic or mildly symptomatic. The symptoms of HCM include dyspnea due to stiffening and decreased blood filling of the ventricles, angina i.e. exertional chest pain due to reduced or restricted blood flow to the coronary arteries, palpitations due to the aforementioned ischemia, as well as disruption of the electrical system running through the abnormal heart muscle, light headedness, fatigue, fainting or syncope and sudden cardiac death. As mentioned, dyspnea is largely due to increased stiffness of the left ventricle, which impairs filling of the ventricles, but also leads to elevated diastolic pressure in the left ventricle and left atrium, causing back pressure and interstitial congestion in the lungs. Symptoms are not closely related to the presence or severity of an outflow tract gradient.[13]According to Elliott PM*et al*[14] although presyncope and syncope on exertion are common in obstructive HCM, it is extremely important to recognise that these symptoms may also result from atrial and ventricular arrhythmias at rest or on exertion, or from failure of blood pressure to rise normally on exertion, even in non-obstructive HCM. Thus, a history of palpitations, particularly rapid heart action when associated with presyncope/syncope, is an integral part of history taking.Family history of hypertrophic cardiomyopathy was observed in 9.52% whereas history of sudden cardiac death in family was given by 5.71% patients.Recently, the results of

molecular genetic studies have resulted in a quantum leap in our basic knowledge and understanding of the mendelian dominant inheritance of HCM and have far-reaching prognostic and clinical implications.[15,16,17] HCM is now described as a heterogeneous disease of the sarcomere,[15,18] in that at least 34 missense mutations have been described in the β-myosin heavy chain gene (chromosome 14q11-q12)[19,20], 7 mutations have been described in cardiac troponin-T (chromosome 1),[18] and 2 mutations in α-tropomyosin (chromosome 15q2).[15,18] Another locus has been found on chromosome 11p13-q13,[21] and familial HCM with Wolff-Parkinson-White syndrome maps to a locus on chromosome 7q3.[17] The hypertrophy in HCM may be compensatory in response to the abnormalities induced by these mutations. This belief is supported by the upregulation of genes commonly observed in compensatory hypertrophy, ie, atrial and brain natriuretic peptides and angiotensin-converting enzyme.[22,23] These molecular genetic studies are already having important clinical implications in that some mutations carry a benign prognosis,[18] whereas others, possibly interacting with angiotensin-converting enzyme genotypes,[24] have increased penetrance, early onset of manifestations, and a bad prognosis,[18,25] thus explaining the malignant family history noted by some authors.[12,26]The mean pulse rate in the study patients was 84.83± 17.85 per min. systolic blood pressure was 124.71 ± 19.01 mm of Hg whereas diastolic blood pressure was 78.37 ± 8.67 mm of Hg. Prominent JVP'a' wave was noted in 23.81% patients in the study. Fourth heart sound was heard in 79.05% patients. Left ventricular outflow tract murmur was present in 54.29% patients and mitral regurgitation was observed in 17.14% patients of HCM. Prominent 'a' wave in the jugular venous pulse indicates RV involvement in HCMmay be detected by and rarely by a right-sided fourth heart sound, reflecting RV diastolic dysfunction, and by a systolic ejection murmur along the left sternal border, reflecting subpulmonic or midventricular obstruction to RV outflow.[8,27]On 2D Echo interventricular septum thickness and posterior wall thickness in diastole was significantly more in apical HCM as compared to HCM.Left atrium size was also significantly increased in HOCM (38.60±7.94). Mean left ventricular diastolic dysfunction was observed in higher grades in HCM and apical HCM as compared to HOCM and the difference observed was also statistically significant. The mean left ventricular ejection fraction was lowest in HCM and was statistically significant as compared to apical HCM and HOCM.Transthoracic Echo/Doppler examination in HCM is undoubtedly the most important form of laboratory investigation. These combined techniques can determine the location and extent of hypertrophy, systolicand diastolic function, the presence and degree of systolic anterior motion of mitral valve, the severity of the subaortic and/or midventricular obstruction, the direction and degree of mitral regurgitation, the presence of additional mitral valve abnormalities and LA size. Transesophageal echo/Doppler studies are valuable in defining additional mitral valve abnormalities and the level of outflow obstruction and are used intraoperatively in planning, guiding, and assessing the results of surgical intervention.[28-31]

CONCLUSION

Thus from the above results and discussion we conclude thatHypertrophic obstructive cardiomyopathy was the commonest cardiomyopathy in the present study. Hypertrophic Cardiomyopathy presented with wide variation in clinical features with angina and dyspnea on exertion as the most common presenting complaint. Prominent 'a' wave of JVP and audible fourth heart sound with Left ventricular outflow tract murmur are the common clinical features associated with HCM. 2 D echo examination in HCM was the most important investigation for diagnosis and also for cardiac functional assessment.

REFERENCES

1. Liouville H. Rétrécissementcardiaque sous aortique. Gaz Med Paris.1869; 24:161-163.
2. Hallopeau L. Rétrécissementventriculo-aortique. Gaz Med Paris.1869; 24:683-684.
3. Brock RC. Functional obstruction of the left ventricle. Guys Hosp Rep.1957; 106:221-238.
4. Schmincke A. UeberlinksseitigemuskuloseConusstenosen. Dtsch Med Wochenschr. 1907; 33:2082.
5. Teare RD. Asymmetrical hypertrophy of the heart in young adults. Br Heart J. 1958; 20:1-8.
6. Braunwald E, Lambrew C, Rockoff SD, Ross J Jr, Morrow AG. Idiopathic hypertrophic subaortic stenosis, I: description of the disease based upon an analysis of 64 patients. Circulation. 1964; 29(suppl IV): IV-3-IV-119.
7. Goodwin JF. The frontiers of cardiomyopathy. Br Heart J. 1982; 48:1-18.
8. Wigle ED, Sasson Z, Henderson MA, Ruddy TD, Fulop J, Rakowski H, Williams WG. Hypertrophic cardiomyopathy: the importance of the site and the extent of hypertrophy: a review. ProgCardiovasc Dis. 1985; 28:1-83.
9. Authors/Task Force members, Elliott PM, Anastasakis A, Borger MA, Borggrefe M, Cecchi F, Charron P, Hagege AA, Lafont A, Limongelli G *et al*. ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). European Heart Journal. 2014; 35:2733–2779.

10. Maron BJ, Casey SA, Poliac LC, Gohman TE, Almquist AK andAeppli DM. Clinical course of hypertrophic cardiomyopathy in a regional United States cohort. Journal of the American Medical Association. 1999; 281 :650–655.
11. Moon JC, Fisher NG, McKenna WJ and Pennell DJ. Detection of apical hypertrophic cardiomyopathy by cardiovascular magnetic resonance in patients with non-diagnostic echocardiography. Heart. 2004; 90:645–649.
12. E. Douglas Wigle, MD; Harry Rakowski, MD; Brian P. Kimball, MD; William G. Williams, MD. Hypertrophic Cardiomyopathy Clinical Spectrum and Treatment. Circulation..1995; 92: 1680-1692
13. Bonow R, Braunwald E, Zipes DP, Libby P. "The Cardiomyopathies".Braunwald's heart disease: a textbook of cardiovascular medicine (7th ed.). Philadelphia: WB Saunders. 2005.
14. Elliott PM, GimenoBlanes JR, Mahon NG, *et al*. Relation between severity of left-ventricular hypertrophy andprognosis in patients with hypertrophic cardiomyopathy.Lancet 2000; 357:420–4.
15. Thierfelder L, MacRae C, Watkins H, McKenna W, Vosberg HP, Seidman JG, Seidman C. Familial hypertrophic cardiomyopathy is a disease of the sarcomere. Circulation. 1994;90(suppl I):I-519. Abstract.
16. Watkins H, McKenna WJ, Thierfelder L, Suk HJ, Anan R, O'Donoghue A, Spirito P, Matsumori A, Moravec CS, Seidman JG, Seidman CE. Mutations in the genes for cardiac troponin T and α-tropomyosin in hypertrophic cardiomyopathy. N Engl J Med. 1995; 332:1058-1064.
17. MacRae C, Ghaisas N, McGarry K, McKenna W, Seidman JG, Seidman CE. Familial hypertrophic cardiomyopathy with Wolff-Parkinson-White syndrome maps to a locus on chromosome 7q3. Circulation. 1994;90(suppl I):I-25
18. Thierfelder L, Watkins H, MacRae C, Lamas R, McKenna W, Vosberg HP, Seidman JG, Seidman CE. α-Tropomyosin and cardiac troponin T mutations cause familial hypertrophic cardiomyopathy: a disease of the sarcomere. Cell.1994; 77:701-712.
19. Fananapazir L, Epstein ND. Genotype-phenotype correlations in hypertrophic cardiomyopathy: insights provided by comparisons of kindreds with distinct and identical β-myosin heavy chain gene mutations. Circulation.1994; 89:22-32.
20. Roberts R. Molecular genetics: therapy or terror? Circulation. 1994; 89:499-502. Editorial.
21. Carrier L, Hengstenberg C, Beckmann JS, Guicheny P, Dufour C, Bercovici J, Dausse E, Berebbi-Bertrand I, Wisnewsky C, Pulvenis D, Fetler L, Vignal A, Weissenbach J, Hillaire D, Feingold J, Bourhour JB, Hagege A, Desnos M, Isnard R, Dubourg O, Komajda M, Schwartz K. Mapping of a novel gene for familial hypertrophic cardiomyopathy to chromosome 11. Nat Genet. 1993; 4:311-314.
22. Takemura G, Fujiwara H, Mukoyama M, Saito Y, Nakao K, Kawamura A, Ishida M, Kida M, Uegaito T, Tanaka M, Matsumori A, Fujiwara T, Imura H, Kawai C. Expression and distribution of atrial natriuretic peptide in human hypertrophic ventricle of hypertensive hearts and hearts with hypertrophic cardiomyopathy. Circulation. 1991; 83:181-190.
23. Derchi G, Bellone P, Chiarella F, Randazzo M, Zino V, Vecchio C. Plasma levels of atrial natriuretic peptide in hypertrophic cardiomyopathy. Am J Cardiol. 1992; 70:1502-1504.
24. Marian AJ, Yu QT, Workman R, Greve G, Roberts R. Angiotensin-converting enzyme polymorphism in hypertrophic cardiomyopathy and sudden cardiac death. Lancet. 1933; 342:1085-1086.
25. Watkins H, Rosenzweig A, Hwang DS, Levi T, McKenna W, Seidman CE, Seidman JG. Characteristics and prognostic implications of myosin missense mutations in familial hypertrophic cardiomyopathy. N Engl J Med.1992; 326:1108-1114.
26. Maron BJ, Lipson LC, Roberts WG, Epstein SE. 'Malignant' hypertrophic cardiomyopathy: identification of a subgroup of families with unusually frequent premature death. Am J Cardiol. 1978; 41:1133-1140.
27. Wigle ED, Heimbecker RO, Gunton RW. Idiopathic ventricular septal hypertrophy causing muscular subaortic stenosis. Circulation. 1962;26:325-340
28. Pollick C, Morgan CD, Gilbert BW, Rakowski H, Wigle ED. Muscular subaortic stenosis: the temporal relationship between systolic anterior motion of the anterior mitral leaflet and pressure gradient. Circulation. 1982; 66:1087-1093.
29. Maron BJ, Gottdiener JS, Epstein SE. Patterns and significance of distribution of left ventricular hypertrophy in hypertrophic cardiomyopathy: a wide angle, two dimensional echocardiographic study of 125 patients. Am J Cardiol.1981; 48:418-428.
30. Yock PG, Hatle L, Popp RL. Patterns and timing of Doppler-detected intracavity and aortic flow in hypertrophic cardiomyopathy. J Am Coll Cardiol.1986; 8:1047-1058.
31. Grigg LE, Wigle ED, Williams WG, Daniel LB, Rakowski H. Transesophageal Doppler echocardiography in obstructive hypertrophic cardiomyopathy: clarification of pathophysiology and importance in intraoperative decision making. J Am CollCardiol. 1992; 20:42-52.

Source of Support: None Declared
Conflict of Interest: None Declared