Electrocardiographic patterns of patients with Hypertrophic cardiomyopathy and their echocardiographic correlation

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Abstract

Background: HCM is an important cause of SCD under 35 yrs and is easily screened by ECG. Prognosis, presentation and severity of hypertrophy have a varied relation with ECG. Extent and prevalence of ECG abnormalities in HCM and their echo correlation is needed to clarify the predictability of the Severity of hypertrophy. ECG categorization of severity would allow easy risk stratification of family members.

Objective: Correlation between the ECG abnormalities and the maximum wall thickness, septal, lateral or apical, and the LVOTO gradients in echo is studied. ECG patterns and pattern of hypertrophy in echo also analysed.

Methods: maximum diastolic thickness of LV at any site more than 13mm and for the apical region more than or equal to 15mm was the criteria for hypertrophy. Measurement of septal thickness in diastole at the thickest part of septum and the other walls in PLAX - average of three M mode values used. Sixty newly diagnosed Hypertrophic cardiomyopathy patients during a 8 month out patient population in our hospital analysed. ECG abnormality and distribution of LVH in echo are analysed. ECG abnormalities noted are LVH criteria (Cornell voltage and Sokolow Lyon), T wave inversion, abnormal Q wave.

Results: LVH in ECG group, 50 patients (83%) had greater maximum septal or other wall thickness than the group with no LVH criteria in ECG 10(17%). Average wall thickness of 20± 4.87 mm v/s 17.8 ± 2.35mm p<0.101 in either group. ECG LVH was significantly associated with atleast moderate, ≥ 20 mm hypertrophy compared to mild hypertrophy only in no LVH in ECG group, p< 0.05. Sensitivity of 93% but low specificity. Presence of abnormal Q wave was not a statistically significant marker of LVH ≥ 20mm. In Apical HCM subset of 18 pts, V4, R wave of ≥ 20 mm and deep T inversion pattern was significantly associated compared to only 9 of 42 patients in non Apical subset p<0.001.

Conclusions: LVH in ECG, predicted greater maximum septal/wall thickness by echo than patients without these ECG changes though p value <0.05 only. V4 R of ≥ 20 mm and deep T inversions in precordial leads pattern was quite specific for Apical HCM. Presence of deep abnormal Q waves, did not significantly correlate with increased septal thickness or LVOTO gradients p<0.205.

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Introduction
Background
Hypertrophic cardiomyopathy (HCM) is an important cause of sudden death under 35 yrs and is a disease which can be easily screened by electrocardiogram (ECG). Nevertheless HCM may present with normal ECG and may be diagnosed by echocardiogram only. The prognosis, presentation and severity of hypertrophy have a varied relation with ECG. Studies(1) have shown poor correlation with ECG. They have not clearly validated that the normal ECG group could probably be a benign variant (2). HCM is a familial disease and normal ECG with echocardiographic diagnosis of HCM are frequent in family members of HCM patients. Pattern of ECG changes and pattern of distribution of hypertrophy have been studied previously. (3)

There could be ethnic variation and rural, urban variation in disease spectrum (3). Published Indian studies are limited for comparison of our population. The studies on pattern of hypertrophy and ECG correlation have been validated with variable results in different studies. A study (4) in Japan with high incidence of apical HCM has shown good relation to ECG pattern in predicting pattern of hypertrophy. The patterns in our population need validation.

Extent and prevalence of ECG abnormalities in HCM and their echocardiographic correlation is needed to further clarify the ECG predictability of the severity of hypertrophy. Symptoms in HCM are related to extent of hypertrophy and also to the left ventricular outflow tract obstruction (LVOTO) gradients and these have a correlation with the incidence of sudden cardiac death (SCD). ECG categorization of severity would allow more easy risk stratification of family members of HCM patients also.

Objectives
This study is to analyse echocardiographic and symptom profile of patients with hypertrophic cardiomyopathy (HCM) in relation to the ECG. Also correlation between the ECG abnormalities and the maximum wall thickness, septal, lateral or apical, and the LVOTO gradients in echocardiogram is studied. Also patterns of hypertrophy and ECG patterns are analysed for specific associations.

Hypothesis
ECG LVH criteria can predict maximum wall thickness in HCM by echocardiogram and the presence of abnormal q and LVH are correlated with LVH and LVOTO gradients.

Methods
Baseline characteristics - history, clinical features and echocardiographic data are collected on all patients with HCM who initially presented to our institution with a diagnostic echocardiogram. The diagnosis (2,5) of HCM was based on the presence of a hypertrophied, nondilated left ventricle in the absence of other diseases capable of producing the degree of observed hypertrophy. Criteria of hypertrophy in echo was maximum diastolic thickness of left ventricle at any site more than 13mm and for the apical region >15mm (15). The primary imaging modality used for diagnosis in all patients is transthoracic 2-dimensional and Doppler echocardiography performed by a single operator on Phillips HD 11 XE Echocardiographic machine. All patients undergo a 12-lead surface electrocardiogram using BPL CARDIART 6208 ECG machine after a prior consent for participation in the study.

Study design
A Cross sectional analysis

Duration
Data collection done over a period of 8 months to study adequate number of patients to achieve statistical significance.

Inclusion criteria
1) All HCM patients at first diagnosis
2) Echo criteria proven HCM referred from other centres
3) AGE > 18 yrs and <60 years (sigmoid septum and hypertensive HCM common in older age)
Exclusion criteria
1) Rheumatic heart disease or other cause of valvular aortic stenosis, pulmonary stenosis
2) Hypertension at diagnosis or on treatment for same
3) Severe pulmonary hypertension (septal hypertrophy)
4) CAD from history or angiogram proven CAD

Wall thickness
Careful measurement of septal thickness in diastole at the thickest part of septum and also the other walls including the thickest part of left ventricular free wall is done in parasternal long axis (PLAX) from an average of three M mode values taken consecutively. In areas where M mode is difficult frozen diastolic 2D images were used for measurement. In parasternal short-axis view, the left ventricle was divided into 4 regions that identified the anterior and posterior ventricular septum and the anterolateral and posterior LV free wall. The greatest wall thickness in frozen 2D images in diastole, measured at any site in the LV wall was regarded as the maximal thickness. Wall thicknesses were assessed directly by calipers. Severe hypertrophy was defined as wall thickness ≥ 30 mm. Mild hypertrophy ≤ 20mm and moderate 20 to 30 mm

Doppler gradient of LVOTO measured in apical five chamber or subcostal view. Only baseline values to be include in the study. Provocation maneuvers are avoided. Also gradient in upright posture is recorded if good echo window achieved. Continuous wave Doppler sampling at the LVOT is done carefully taking care to achieve maximum parallel alignment. Significant LV outflow obstruction under basal conditions was defined as a peak outflow gradient of more than 30 mm Hg, as estimated by continuous-wave Doppler echocardiography. Diastolic dysfunction is also assessed by Doppler mitral inflow velocities and also by Tissue Doppler early diastolic mitral annulus (Ea) velocity at lateral mitral annulus with Tissue Doppler pulse applied over it in apical 4 chamber view. An Ea of less than 8 cm/sec suggests significant diastolic dysfunction.

Presence of abnormal q waves, LVH in ECG and the extent of septal/ or lateral wall hypertrophy are analysed for correlation. Also pattern of ECG abnormality and distribution of left ventricular hypertrophy in echocardiogram are analysed.

ECG abnormalities noted are LVH criteria, T inversions, abnormal Q waves. Patients with other ECG abnormalities like conduction abnormalities, arrhythmias are excluded.

7) Electrocardiographic criteria:
ECG criteria proposed for the clinical identification of LVH in this study, include
1) the maximum R or S wave height ≥ 30 mm
2) the sum of SV1 (or SV2) and RV5 (or RV6) ≥ 35 mm, an adaptation of the Sokolow-Lyon criteria
3) Cornell voltage criteria
   female R Avl + S V3 ≥ 2.00mV
   male R Avl + S V3 ≥ 2.80mV

The diagnostic criteria for abnormal Q waves and positive and negative T waves were as follows:
(i) Q waves were considered significant if it satisfies any of the below
   - Any Q-wave in leads V2–V3 ≥ 0.02 s or QS complex in leads V2 and V3
   - absence of history of myocardial infarction
   - Q-wave ≥ 0.03 s and > 0.1 mV deep in other leads
(ii) positive or negative T waves: more than 0.2mv in amplitude in precordial leads. In the case of, biphasic T waves, we measure the larger wave.(7)

Statistical analysis
Data compared by chi-square analysis for proportions. A p value less than 0.05 is considered significant. The number of patients deemed necessary to attain statistical significance with a presumed prevalence of LVH in the HCM group was at least 50.

Results
Of the 60 patients, 40(66%) were in functional class II NYHA, 10(17%) were in class III and 10(17%) asymptomatic. Syncope occurred in 6(10%). Mean age of the total group of patients was 49 years (range 24-60 years). Male to female ratio was 2:1

LVH by ECG criteria positive in 50(83%) and negative in 10(17%)

Patients with abnormal electrocardiograms 52 (87%) had greater maximum septal or other wall thickness than patients with normal electrocardiograms 8(13%). But this did not reach statistical significance p>0.101 (TABLE 1). Electrocardiographic LVH was associated with atleast moderate ≥ 20 mm hypertrophy by echo as compared to mild hypertrophy only in the no LVH in ECG group of patients p<0.05, sensitivity 95% and specificity of 25% only for ECG LVH criteria in predicting
significant hypertrophy in HCM.

In Apical HCM subset of 18 patients, V4 R wave >20 mm and deep T inversion in precordial leads was seen in 16 patients as compared to only 9 out of 42 in the non Apical subset (p<0.001). Pattern was 78% specific for apical subtype sensitivity 89% (Table 3). Presence of abnormal Q wave was not a statistically significant marker of LVH of ≥20 mm p<0.205 (Table 2).

The most common variety of HCM in our study was ASH and apical variety probably because of referral bias of these groups in view of easy screening of both.

Most of the study group had a tissue E < 8 cm/s of septal mitral annulus suggestive of diastolic dysfunction, 50 of the total 60 patients.

Discussion

The prevalence of abnormal electrocardiograms in patients with hypertrophic cardiomyopathy has been reported previously as 10%, 28%, 18%, and

<table>
<thead>
<tr>
<th>ECG LVH Criteria</th>
<th>Echo max wall thickness&gt;20mm</th>
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<tr>
<td>+</td>
<td>27</td>
<td>23</td>
</tr>
<tr>
<td>-</td>
<td>2</td>
<td>8</td>
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P<0.05

Table 2  P<0.205

<table>
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<tr>
<th>ECHO septum thickness</th>
<th>Abnormal Q</th>
<th>NOQ</th>
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<tr>
<td>≥20mm</td>
<td>15 (51.7%)</td>
<td>14 (48.39%)</td>
</tr>
<tr>
<td>&lt;20mm</td>
<td>11 (35.5%)</td>
<td>20 (64.5%)</td>
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P < 0.001  Table 3

almost 100%. Our results report nearly 90% incidence. Our study indicates that normal electrocardiograms are rare in patients with HCM, 10% only. We also observe that abnormal ECG, especially LVH criteria were associated with more septal thickening though it was not statistically significant. Abnormal q waves though common are not a predictor of significant hypertrophy nor LVOT gradient.

In a previous study by Maron et al, most extreme phenotypic expressions with massive degrees of LV hypertrophy (11) unique to HCM showed increased precordial or standard lead voltages in only 50% of the cases.

Findings substantiate that although surface ECG potentials bear some specific correlation to left ventricular mass in HCM electrocardiography is not a strong predictor of the true magnitude of LV hypertrophy as assessed by 2D echo which was suggested by Maron et al as well. In contrast, scalar electrocardiography does have clinical utility by raising the suspicion of undiagnosed patients with HCM within large athlete populations, military recruits etc (13, 14).

Symptom status was NYHA I or II in most patients and only limited number of patients have worse clinical status. Syncope was less common, only 10% which corresponds to that reported in previous studies. Population in our study was older than average age group for sudden death in HCM which suggests that they could represent a subset of survivors in a larger group of patients including those at higher risk of SCD. The lesser events in our study population could be explained so. Only one patient had a family history of SCD and none had history of revived cardiac arrest or ventricular tachycardia.

Regarding the patterns of hypertrophy, the least common was the septum and lateral wall hypertrophy unlike other studies, which could be explained by our subset being that of referred patients rather than community screening study. Most common variants were asymmetric septal and apical hypertrophy.

Conclusions

Patients with the electrocardiographic findings of LVH, had greater maximum septal or any wall thickness by echocardiography than patients without these ECG changes though p value <0.05 only. V4, R > 20 mm and deep T inversions pattern was quite specific for Apical HCM. Presence of prominent abnormal Q waves, did not significantly correlate with increased septal thickness or LVOTO gradients p<0.205.