



Relationship between electrocardiographic findings and Cardiac Magnetic Resonance phenotypes in patients with Hypertrophic Cardiomyopathy



Matteo Fronza^a, Claudia Raineri^a, Adele Valentini^b, Emilio Maria Bassi^b, Laura Scelsi^a, Maria Laura Buscemi^a, Annalisa Turco^a, Grazia Castelli^a, Stefano Ghio^{a,*}, Luigi Oltrona Visconti^a

^a Division of Cardiology, Fondazione I.R.C.C.S. Policlinico S Matteo, Pavia, Italy

^b Institute of Radiology, University of Pavia School of Medicine, Pavia, Italy

ARTICLE INFO

Article history:

Received 4 November 2015

Received in revised form 9 January 2016

Accepted 21 February 2016

Available online 2 March 2016

Keywords:

Hypertrophic Cardiomyopathy

Electrocardiography

Cardiac Magnetic Resonance

ABSTRACT

Background: Q waves and negative T waves are common electrocardiographic (ECG) abnormalities in patients with Hypertrophic Cardiomyopathy (HCM). Several studies correlated ECG findings with presence and extent of fibrosis and hypertrophy; however, their significance remains incompletely clarified. Our study aimed to explain the mechanism behind Q and negative T waves by comparing their positions on a 12-lead ECG with phenotypes observed at Late Gadolinium Enhancement (LGE) Cardiac Magnetic Resonance (CMR).

Methods: 12-lead ECG and LGE-CMR were performed in 88 consecutive patients with HCM (42 SD 16 years, 65 males). Using Delta Thickness ratio (DT ratio), and “global” and “parietal” LGE at CMR, the extent and distribution of myocardial hypertrophy and fibrosis were studied in correlation with ECG abnormalities.

Results: Q waves in different leads were not associated with “parietal” LGE score. Lateral Q waves correlated with an increased DT ratio Inferior Septum/Lateral wall ($p = 0.01$). A similar correlation between inferior Q waves and an increased DT Ratio Anterior wall/Inferior wall was of borderline statistical significance ($p = 0.06$). As expected, ECG signs of LV hypertrophy related to a raised Left Ventricular Mass Index (LVMI) ($p < 0.0001$) and mean wall thickness ($p = 0.01$). Depolarization disturbances, including negative T waves in lateral ($p = 0.044$) and anterior ($p = 0.031$) leads correlated with “parietal” LGE scores while QT dispersion ($p = 0.0001$) was associated with “global” LGE score.

Conclusion: In HCM patients, Q waves are generated by asymmetric hypertrophy rather than by myocardial fibrosis, while negative T waves result from local LGE distribution at CMR.

© 2016 The Authors. Published by Elsevier Ireland Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

1. Background

Hypertrophic Cardiomyopathy (HCM) is a genetic disorder with extremely variable morphologic, functional, and clinical features [1]. More than 90% of HCM patients with evidence of the disease at echocardiogram show various abnormalities in the 12-lead ECG [2], including pathological Q waves, signs of LV hypertrophy, and repolarization disturbances such as negative T-wave [3–5]. The pathogenesis of the ECG findings, however, remains unclear. Different studies have correlated ECG abnormalities with different imaging tests, adding important piece of information in what seems to be a pathophysiological puzzle.

Authors have tried to explain the mechanism of these ECG findings using low-definition and two-dimensional imaging investigations, such echocardiography and left ventriculogram, with controversial results [3,4,6]. Late Gadolinium Enhancement Cardiac Magnetic Resonance (LGE-CMR) however, offers high spatial resolution and 3-dimensional tomographic imaging allowing not only for a more accurate morphologic and functional definition of the left ventricle (LV), but also giving information regarding tissue composition and revealing areas of myocardial fibrosis [7–11]. However: Q waves have not been unequivocally correlated yet to a specific phenotype pattern. The hypothesis that an asymmetric distribution of myocardial hypertrophy rather than myocardial fibrosis may generate Q waves has been verified in anterior leads but it has not been verified in other LV walls. In addition, negative T waves have been so far correlated with apical hypertrophy while their association with local distribution of LGE at CMR appears to be relevant and needs to be supported more. The aim of the present study is to verify the hypothesis that in patients with HCM, Q waves are generated by asymmetric distribution of myocardial

* Corresponding author at: Divisione di Cardiologia, Policlinico S.Matteo, Piazza Golgi 1, 27100 Pavia, Italy.

E-mail addresses: fronza.matteo@gmail.com (M. Fronza), c.raineri@smatteo.pv.it (C. Raineri), a.valentini@smatteo.pv.it (A. Valentini), e.bassi@smatteo.pv.it (E.M. Bassi), l.scelsi@smatteo.pv.it (L. Scelsi), m.buscemi@smatteo.pv.it (M.L. Buscemi), a.turco@smatteo.pv.it (A. Turco), gracecast@gmail.com (G. Castelli), s.ghio@smatteo.pv.it (S. Ghio), l.oltrona-visconti@smatteo.pv.it (L.O. Visconti).

hypertrophy and to demonstrate that negative T waves result from local distribution of fibrosis.

2. Methods

2.1. Population

The initial study population included 137 consecutive patients with Hypertrophic Cardiomyopathy (HCM) referred to our Centre between October 2004 and January 2014. The diagnosis of HCM was based on 2-dimensional echocardiographic evidence of a hypertrophic left ventricle (maximal wall thickness ≥ 15 mm in adult index patient or ≥ 13 mm in adult relatives of a HCM patient) in the absence of another cardiac or systemic disease able to justify the amount of hypertrophy observed (e.g. hypertensive cardiopathy, athlete's heart, storage disease). Using this imaging technique maximal left ventricular wall thickness (LVT max) was initially assessed along with left ventricular outflow tract obstruction (LVOTO), which was measured as instantaneous peak Doppler LV outflow tract pressure gradient ≥ 30 mmHg at rest or during physiological provocation such as Valsalva manoeuvre. Forty-nine patients with a history of surgical myectomy, alcohol ablation, CMR contraindications (e.g. pacemakers, implantable defibrillators or atrial fibrillation), or with a poor quality ECG strip were excluded. Therefore the final study population included 88 patients.

A standard 12-lead ECG was performed before CMR in all patients; it was analysed by ECG analysis software (CALL ECG Version 2.6.5.) and systematically reviewed by a single operator. The third universal definition of myocardial infarction and modified Sokolow–Lyon criteria were used to characterize pathological Q waves [12], and signs of LV hypertrophy [13]. Depolarization disturbances, including T wave inversion, ST depression and QT dispersion, calculated as the difference between minimum and maximum QT duration, were also noted.

Within 6 months from clinical examination and 12-lead ECG, Cardiac Magnetic Resonance images were obtained using a 1.5T scanner (Siemens Symphony, Erlangen, Germany) with a phased array cardiac coil and electrocardiogram gating. Cine magnetic resonance images were acquired to assess ventricular function, using a segmented, balanced, steady-state free precession sequence (b-SSFP) in three long-axis planes and sequential 8 mm short-axis slices from the atrio-ventricular ring to apex. On the latter axis LV endocardial and epicardial borders were manually traced at end diastole and end systole respectively. Other parameters were also measured, including LV mass, end-diastolic (LVEDV), end-systolic (LVESV) volumes divided by body surface area (BSA), as well as ejection fraction (LVEF). All CMR images were analysed by validated software (Argus; Siemens Medical Solutions) and reviewed by experienced radiologist and cardiologist. A 17-segment model for the LV was used to assess extent and distribution pattern of both end-diastolic wall thickness and LGE [14]. In regards with the hypertrophic features of the LV, the short axis was used to measure end-diastolic wall thickness of 16 segments while long axis was used for the apex. All 17 thickness values were marked and then averaged to obtain an absolute value of maximal left ventricular thickness (LVT max) as well as the mean end-diastolic thickness (mean DT). A Delta Thickness ratio (DT ratio) was created to assess LV asymmetric hypertrophy. This was expressed as ratio between the end-diastolic thickness of a hypertrophied wall, including basal, mid and apical segments, and its opposite wall. In particular, a DT ratio between Inferior Septum/ and Lateral wall (DT ratio Sep-lat), anterior/inferior wall (DT ratio ant-inf) and inferior/anterior wall (DT ratio inf-ant) was calculated. Then 0.2 mmol/kg of Gadolinium-DTPA (Gadovist; Bayer Schering Pharma, Berlin, Germany or Magnevist, SheringPharma, Germany) was injected, and 10–15 min later LGE images were obtained with a breath-hold 2-D segmented phase sensitive inversion-recovery sequence (inversion time between 240 and 300 ms). LGE was considered present in areas with signal intensity exceeding 5 standard deviation (SD) compared to normal myocardium signal. The amount of LGE was measured by a

semi-quantitative method, using a visual score post-processed by software CMR42 (Circle Cardiovascular Imaging, Calgary, Alberta, Canada) and blindly assessed by 2 different and expert operators. Each segment was scored out of three (1 if LGE involved $<25\%$, 2 if between 25% and 50%, and 3 if more than 50% of the wall thickness) and a “parietal” and “global” LGE scores were calculated as the sum of the visual scores of basal, mid and apical segments of a given wall and the whole ventricle.

Data were analysed using the software STATA 13.1 (STATA Corporation College Station, Tx, USA). Continuous variables were expressed as median and interquartile range and analysed with the Mann–Whitney U test; categorical variables were expressed as a percentage and compared by Fisher's exact test. The association between continuous variables was analysed using Pearson's coefficient R or Spearman's coefficient R, depending on the distribution, with their associated CI at 95%. A two-tailed *p* value of <0.05 was considered statistically significant. All participants provided written informed consents.

3. Results

Clinical characteristics and risk factors are detailed in Table 1. The study population consisted of 88 patients, 65 males (74%) and 23 females (26%), aged between 8 and 76 years old (mean age 42 SD 16 years). Fifty-seven patients (65%) were in New York Heart Association functional class I (NYHA I), 23 patients (26%) in NYHA II, 8 patients (9%) in NYHA III. Only 14 patients showed signs of subvalvular obstruction with an intraventricular gradient >30 mmHg at continuous-Doppler placed on the LVTOT during the echocardiogram study. Thirty-one patients had a familial history of HCM (FHHCM), while 8 patients (9%) was found to have a LVT max >30 mm at USS.

Only 2 patients (2%) had a normal ECG, while the other 86 (98%) showed different ECG abnormalities. The presence of negative T waves on ECG was by far the most common ECG abnormality detected (89%), while signs of LV hypertrophy and pathological Q waves [12] in different leads were found in 51% and 38% of cases respectively. Other electrocardiographic abnormalities are listed in detail in Table 2.

Morpho-functional data were obtained from a systematic review of CMR images (Table 3). In detail, the mean wall thickness was 11.2 SD 2.9 mm, the mean value for the maximum thickness was 23 SD 7.5 mm, and the LV Mass Index (LVMI) was 94.2 SD 42.7 g/m². Seventy-eight patients (88%) had features of asymmetric LV hypertrophy either localised in the posterior septum, anterior or posterior wall, with 13 patients (15%) had a LVT max >30 mm at CMR. Seventy-five patients (86%) showed different levels and patterns of LGE after paramagnetic medium of contrast injection, with a mean “global” LGE score above 10 (10.4 SD 9.9). Twenty-nine patients (33%) had parietal LGE score >1 in antero-lateral wall (6th, 12th and 16th segments) while

Table 1
Clinical features and risk factors.

Clinical features and risk factors	N (%)
Age (years)	42 \pm 16
Male	65 (74%)
Functional class NYHA I–II	80 (91%)
Family history of SCD	31 (25%)
Cardiac arrest or SVT	3 (3%)
NSVT	25 (28%)
Loss of consciousness	1 (1%)
Abnormal BP response during exercise	3 (3%)
Familial history of HCM	9 (10%)
High-risk genetic mutation	2 (2%)
LVT max >30 mm	8 (11%)
LVOTO	14 (16%)

SVT sustained ventricular tachycardia, SCD: Sudden Cardiac Death secondary to HCM, NSVT: nonsustained ventricular tachycardia at 24-hour tape, BP: Blood Pressure, HCM Hypertrophic Cardiomyopathy, LVT max >30 mm: Left Ventricular Thickness >30 mm at cardiac USS.

Download English Version:

<https://daneshyari.com/en/article/2926923>

Download Persian Version:

<https://daneshyari.com/article/2926923>

[Daneshyari.com](https://daneshyari.com)