Assessment of electrocardiographic left ventricular hypertrophy with coronary computed tomographic angiography

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Assessment of electrocardiographic left ventricular hypertrophy with coronary computed tomographic angiography

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The Master's Thesis
submitted to the Department of Medicine,
the Graduate School of Yonsei University
in partial fulfillment of the requirements for the degree
of Master of Medical Science

Jin-Sun Kim
December 2010

This certifies that the Master's Thesis of
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December 2010

ACKNOWLEDGEMENTS

This page is exclusively designed to note my gratitude and respect for those who helped me to complete my thesis. I am deeply indebted to my supervisor Prof. Hyuk-Jae Chang for his kind help, guidance, support and encouragement throughout my study. This study would not have been possible without his assistance and cooperation. I would like to express my sincere gratitude to my reviewers, Prof. Byung-Wook Choi and Young-Jin Kim who had the patience and fortitude to read my thesis and provided constructive criticism to help me defend it. Their guidance not only improved my thesis but also will benefit my future work. I also sincerely thank my colleague, Jung-Myung Lee, Sung-Jin Hong, Jung-Woo Son and Yong-Sung Seo for their assistance on my work and emotional support. Finally, my husband and daughter are the ones to whom my special gratitude and respect to go. They always play for my side and strengthen my will to achieve my ambition.

Jin-Sun Kim

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ABSTRACT

Assessment of electrocardiographic left ventricular hypertrophy with coronary computed tomographic angiography

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Background/Aims: The prognostic significance of left ventricular hypertrophy (LVH) in electrocardiogram (ECG) has been widely proven. But there are several problems in ECG diagnosis of LVH. The main concern is the low sensitivity of the ECG-LVH criteria and there is too much portion of false positive. Thus, we hypothesize that other measures such as left ventricular (LV) geometry in coronary computed tomographic angiography (CTA) may reveal some anatomical factors (other than LV mass) which correlate with ECG-LVH. The purpose of this study is 1) to compare measures of coronary CTA in ECG-LVH patients with normal ECG control group and 2) to assess the value of measures other than coronary artery in coronary CTA.

Method: Among the 4300 patients who undertaken coronary CTA between March 2003 and April 2009, in the Severance Hospital, Yonsei University College of Medicine, we sorted out 154 patients who met definite criteria for electrical LVH. Additional sorting out was performed for matched control group with normal ECG. LV mass, LV volume, papillary muscle volume and septal distance at 65% RR interval was measured by manual tracing.

Result: LV mass and LV volume at 65% RR interval were significantly larger in ECG LVH group (LV mass, 162.7 ± 38.6 in ECG LVH group vs. 139.2 ± 28.7 in normal ECG group, p <0.001; LV volume, 114.1 ± 30.8 in ECG LVH group vs. 105.0 ± 27.7 in normal ECG group, p = 0.011). Papillary muscle mass was significantly larger in ECG LVH group, but when corrected by LV mass, there was no significant difference between ECG LVH group and normal ECG group (PM mass, 9.0 ± 3.4 vs 7.6 ± 2.7, p < 0.001; PM mass/total LV mass (%), 5.2 ± 1.4 vs. 5.1 ± 1.3, p= 0.734). Mean number of papillary muscle was higher in ECG LVH group, but this finding might be attributable to overall hypertrophic change of myocardium in LVH group (Mean number of papillary muscles : 2.33 ± 0.53 in ECG LVH vs. 2.17 ± 0.44 in normal ECG, p = 0.013)

Conclusion: Left ventricular hypertrophy in ECG might reflect

hypertrophy of left ventricle. But solitary papillary muscle hypertrophy does not seem attributable to ECG LVH. Images of coronary CTA at 65% RR interval look reliable for evaluation of LV mass and papillary muscle mass comparing to end-diastolic CT images.

Key words: left ventricular hypertrophy, coronary computed tomographic angiography, papillary muscle

Assessment of electrocardiographic left ventricular hypertrophy with coronary computed tomographic angiography

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I. INTRODUCTION

1. Background

The prognostic significance of left ventricular hypertrophy (LVH) in electrocardiogram (ECG) has been widely proven. Several studies have shown that LVH is an important risk factor in patients with hypertension, leading to a fivefold to 10-fold increase in cardiovascular risk (1-5) which is similar to the increase seen in patients with a history of myocardial infarction. The presence of LVH, in addition to hypertension, thus has important implications for assessing risk and managing patients, including decisions on interventions other than antihypertensive treatment, such as lipid lowering treatment and lifestyle modifications (6,7). Accurate and early diagnosis of LVH is therefore an important component of the care of patients with hypertension. Even in general population without hypertension, the ECG-LVH criteria have been

documented to be independent cardiovascular risk factors. An increased prevalence and incidence of LVH on ECG, irrespective cause, is associated with a poor prognosis in very old men and women and regression of ECG LVH in older people, irrespective of cause may confer improvement in risk for cardiovascular disease (8).

But there are several problems in ECG diagnosis of LVH. The main concern is the low sensitivity of the ECG-LVH criteria. Although ECG-LVH criteria are generally considered to be highly specific, the specificity ranges from 53% to 100%. For example, the proportion of the false-positive ECG findings in hypertensive patients in Losartan Intervention for Endpoint Reduction study was 30% (9). Moreover, when true LVH prevalence in less than 10%, more false positive than true positive diagnoses might be obtained(10). Thus it may be unwise to use ECG LVH for screening or epidemiologic purpose.

Another limitation of ECG-LVH diagnosis is derived from the possibility that the QRS voltages might be influenced by a variety of factors other than left ventricular size or mass. These factors include age, gender, race, and body habitus. Their effects may contribute to the limited accuracy of the ECG criteria. Day-to-day variability and variability resulting from variations in the sites of electrode placement also impact QRS voltages and, hence, the diagnostic value of ECG voltage criteria (11).

Nevertheless, the greater convenience and lower cost of the ECG continue to support its widespread use for the diagnosis of ventricular hypertrophy in

clinical practice, epidemiological studies, and clinical trials(12).

But, as previously mentioned, considerable portion of false positive diagnoses is problematic. Because of its prognostic significance, further prices of evaluations are unavoidable in the patients with ECG-LVH. But, in our daily clinical practice, we frequently encounter the final result of further evaluation revealed no specific pathologic condition. In these cases, can we give much meaning to ECG-LVH? And are there other specific causes which induce the false positive results?

Coronary computed tomographic angiography (coronary CTA) is being increasingly used for assessing ischemic heart disease. Although many studies document the potential value of coronary CTA to diagnosing coronary artery disease, little is known about the other measures such as left ventricular (LV) geometry that can be obtained from the same scan data.

2. Purpose of this study

Thus, we hypothesize that other measures such as left ventricular (LV) geometry in coronary CTA may reveal some anatomical factors (other than LV mass) which correlate with ECG-LVH. The purpose of this study is 1) to compare measures of coronary CTA in ECG-LVH patients with normal ECG control group and 2) to assess the value of measures other than coronary artery in coronary CTA.

II. MATERIALS AND METHODS

1. Study population

Four thousands and three hundred patients with suspected coronary artery disease had performed coronary CTA between March 2003 and April 2009, in the Severance Hospital, Yonsei University College of Medicine. Among the 4300 patients, we sorted out 154 patients who met definite criteria for electrical LVH. Additional sorting out was performed for age, sex-matched control group with normal ECG.

2. Electrocardiography

: A standard 12-lead ECG was recorded at 25 mm/s and 1 mV/mm standardization, with a MAC 5000 resting ECG system (GE Medical Systems) within 2 weeks of coronary CTA and interpreted blindly by two investigators.

3. Coronary CTA

64-section CT (Sensation 64, Siemens Medical Solutions, Forchheim, Germany) was used for Coronary CTA. The individual detector collimation was 0.6 mm. The rotation time was 330 ms with an effective temporal resolution of 165 ms. A tube voltage of 120 kV and an effective tube current of 800mA were used. Table feed per tube rotation was 3.3 mm. The scanning time was from 9.0 to 11.0 seconds depending on the scanning range. All scans were performed in a

single breath hold. A 75-mL dose of nonionic contrast media (iopamidol 370 mg/mL; Bracco spa, Milano, Italy) was injected at a rate of 5 mL/s into the antecubital vein. The scan was started on a fixed delay of 5 seconds after the density at the ascending aorta reached 100 Hounsfield units (HU). A 50mL saline chaser was injected at the same rate of 5 mL/s to wash the contrast media from the right ventricle to diminish beam-hardening artifacts. When the mean heart rate was more than 65 beats per minute, 40 to 80mg of propranolol (Pranol DWN, propranolol HCl; Daewoong, Seoul, Korea) was administered to patients by mouth 1 to 2 hours before the examination. If the heart rate did not decrease to lower than 65 beats per minute with the administration of 40 to 80 mg of propranolol, coronary CTA was not performed.

For reconstruction of images, retrospective electrocardiographical gating is applied and images are routinely reconstructed at 65% of the R-R interval. When a motion artifact in the obtained data is noted, the optimal phase is selected with the preview function, for reconstruction of the images with the least motion. A slice thickness of 0.75mm and an increment of 0.5mm are used. For postprocessing, Leonardo and Wizard workstations (Siemens Medical Solutions) are used. Multiplanar reformations, maximum intensity projections, and cross-sectional images are obtained for the evaluation of coronary artery. For myocardial and PMT analysis, images are obtained with a minimum of 40 contiguous images with a 2.5- or 3-mm slice thickness starting above the left main coronary artery to the bottom of both ventricles. For determination of

LVS(sum of LV mass and volume) the following sequential steps are taken. First, a single midventricular slice is selected according to the natural cardiac markers that include pericardial fat, epicardial fat in the atrioventricular groove, and the interventricular groove. The midventricular slice is defined as the level containing the coronary sinus slice or the first level below the left atrium. Second, a straight line connecting the anteroposterior juncture of both ventricles is drawn to divide the left and right ventricles, and the area of the left ventricle is then traced. The anteroposterior juncture origin is the interventricular groove, identified by natural markers such as the abrupt dip which represents fat tissue or a high-density circular image which represent the transverse section image of the left anterior descending coronary artery (18). Third, the area determined by this method is then multiplied by the ventricular height which is equal to the value of the thickness of slice multiplied by the number of slices (each 3 mm in thickness) taken to cover the myocardium. The following formula (from a single, midventricular slice) by using the area (A) and span (H) from the noncontrast studies is used (LVS [in mL] = 0.051 x A [in mm2] + 3.92 x H [in mm] - 277), as previously validated by Mao et al. (19). CT LVS is reported in milliliters. Finally, the LVS is measured during diastole as determined by the chosen imaging acquisition window of CT (multidetector CT 65% of the R-R cycle). Minimal interobserver variability for LVS was previously reported (20). For all data sets, one experienced observer manually trace the endocardial and epicardial contours of the LV. All papillary muscles are outlined separately,

excluded from the volume, and included in the mass. Trabeculae are defined as myocardium protruding more than 1.5mm from the circumferential contour of the LV cavity with equivalent signal intensity to the adjacent LV wall (21). The standard 17-segment model is used for segmental analysis.

To confirm the accuracy of LV mass estimation with coronary CTA, we are going to compare LVM estimated at 65% of the R-R cycle with one estimated at end-diastole. For this, we are going to use 8 patient's CT data which are available at multiphase of the cardiac cycles. After verifying the accuracy of coronary CTA at estimating LVM, we are going to use the LVM data estimated in coronary CTA.

4. Methodological reproducibility

All CT tracings are performed by experienced physicians who are blinded to patient clinical history and results of the other methods. In order to assess intraobserver and interobserver reproducibility, repeat tracings are performed in 20 patients (10 ECG-LVH and 10 normal ECG). Repeat tracings are performed at least 30 days following the first tracing by physicians blinded to initial quantitative results.

5. Statistical analysis

Results are expressed as a mean ± SD or n (%). Comparisons of categorical variables are made using the chi-squared test or Fisher's exact test while

Student's t-test is used for comparing continuous variables. If the distributions were skewed, a non-parametric test was used. All analyses are performed using Statistical Analysis Systems (SAS) software (SAS; 9.1.3., SAS Institute, Cary, NC, USA). A p-value <0.05 was considered statistically significant.

III. RESULTS

1. Reliability for coronary CTA measures

To confirm the reliability of LV mass and papillary muscle mass estimation with coronary CTA, we compared LV mass and papillary muscle mass estimated at 65% of the R-R cycle with one estimated at end-diastole. As previously mentioned, eight patient's CT data which are available at multiphase of the cardiac cycles were analyzed. Mean LV mass and mean papillary muscle mass revealed no significant difference between measures at end-diastole and 65% RR interval (Table 1). Intraclass correlation coefficients between parameters at end-diastole and 65% RR interval were 0.976 and 0.988 for LV mass measure and papillary muscle mass measure, respectively (Table 2). Correlations between end-diastolic and 65% RR interval parameters are shown on the interactive graphs presented as Figure 1. and Bland-Altman plot for LV mass and papillary PM mass are presented as Figure 2.

Table 1. Comparison of parameters at end-diastole and 65% RR interval.

N = 8	End-diastolic parameters	RR 65% Parameters	P - value
LVM	150.4 ± 36.8	152.9 ± 40.0	0.248
PM mass	7.1 ± 2.3	7.2 ± 2.4	0.106

LVM, left ventricular mass; PM, papillary muscle

Table 2. Intraclass correlation coefficient between parameters at end-diastole and 65% RR interval.

N = 8	Intraclass correlation coefficient	95% confidence interval
LVM	0.976	0.886 ~ 0.995
PM mass	0.988	0.90~0.998

LVM, left ventricular mass; PM, papillary muscle

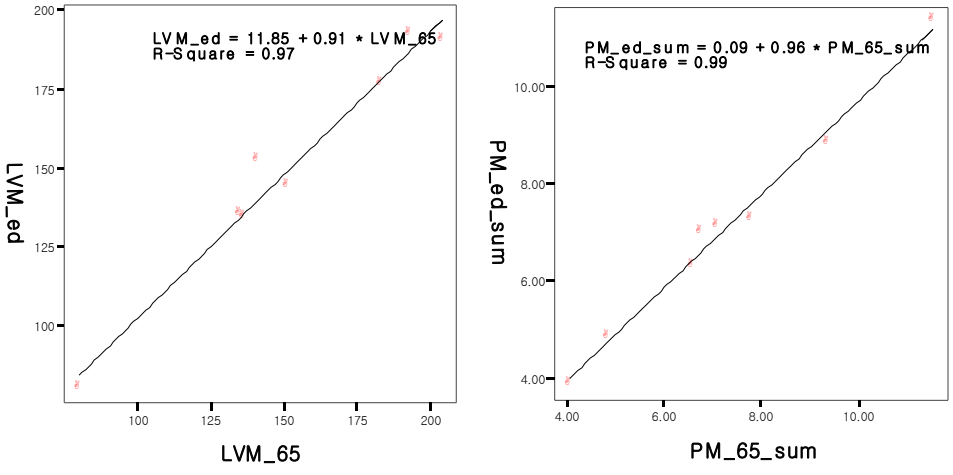

Figure 1. Correlation between end-diastolic left ventricular (LV) mass and 65%

RR interval LV mass (*left*), correlation between end-diastolic papillary muscle (PM) mass and 65% RR interval PM mass (*right*).

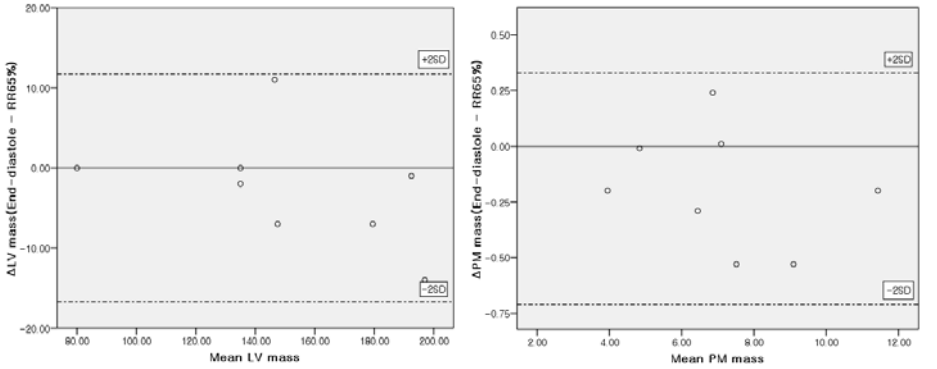

Figure 2. Bland-Altman plot for left ventricle (LV) mass (*left*) and papillary muscle (PM) mass (*right*).

2. Baseline characteristics

Among 154 pairs of ECG LVH and normal ECG subjects, 20 pairs were dropped out due to insufficient or poor CT images for LV mass or papillary muscle mass measure. The baseline characteristics of the rest of the pairs were shown on Table 3. Because two groups were matched for age, sex and BMI, these parameters were similar between two groups. Other clinical characteristics, such as hypertension, diabetes mellitus, smoking did not reveal significant differences. Neither laboratory parameters nor presence of coronary artery disease on coronary CTA were significantly different between two groups.

Table 3. Baseline characteristics

	Electrical LVH (N= 134)	Normal ECG (N=134)	p-value
Age	59.4 ± 10.3	58.7 ± 10.2	0.555
Sex	109 (81.3%)	109 (81.3%)	1.0
Height(cm)	166 ± 8	166 ± 7	0.548
Weight(kg)	68.2 ± 10.7	69.0 ± 9.7	0.495
BMI	24.8 ± 2.7	24.8 ± 3.5	0.996
Hypertension	78 (58.2%)	79 (59.0%)	0.992
SBP(mmHg)	128 ± 14	124 ± 17	0.060
DBP(mmHg)	79 ± 11	78 ± 10	0.199
Diabetes mellitus	23 (17.2%)	34 (25.4%)	0.221
Smoking	30 (22.4%)	24 (17.9%)	0.478
Hemoglobin(g/dL)	14.3±1.7	14.4 ± 1.6	0.405
Glucose(mg/dL)	106 ± 26	108 ± 30	0.542
BUN(mg/dL)	15.5 ± 5.2	15.3 ± 4.2	0.741
Creatinine(mg/dL)	1.09 ± 1.22	1.02 ± 0.3	0.510
Total cholesterol(mg/dL)	187 ± 32	188 ± 41	0.831
Triglyceride(mg/dL)	144 ± 80	164 ± 119	0.139
HDL-cholesterol(mg/dL)	49 ± 12	47 ± 12	0.149
LDL-cholesterol(mg/dL)	117 ± 29	119 ± 35	0.554
Result of CTCA			0.777
Normal or minimal	76 (56.7%)	73 (55.3%)	
CAD	25/33(18.7/24.6%)	31/28 (23.5/21.2%)	0.969
1-vessel disease	29 (21.6%)	31 (23.5%)	
2-vessel disease	12 (9.0%)	13 (9.8%)	
3-vessel disease	9 (6.7%)	8 (6.1%)	

ECG, electrocardiogram; LVH, left ventricular hypertrophy; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; BUN, blood

urea nitrogen; HDL, high-density lipoprotein; LDL, low-density lipoprotein; CTCA, coronary computed tomographic angiography; CAD, coronary artery disease.

3. Measures of coronary CTA

Measures of coronary CTA are demonstrated in Table 4. LV mass and LV volume at 65% RR interval were significantly larger in ECG LVH group (LV mass, 162.7 ± 38.6 in ECG LVH group vs. 139.2 ± 28.7 in normal ECG group, p <0.001; LV volume, 114.1 ± 30.8 in ECG LVH group vs. 105.0 ± 27.7 in normal ECG group, p = 0.011). Papillary muscle mass was significantly larger in ECG LVH group, but when corrected by LV mass [PM mass/total LV mass (%)], there was no significant difference between ECG LVH group and normal ECG group (PM mass, 9.0 ± 3.4 vs 7.6 ± 2.7, p < 0.001; PM mass/total LV mass (%), 5.2 ± 1.4 vs. 5.1 ± 1.3, p= 0.734).

Mean number of papillary muscle was higher in ECG LVH group (Mean number of papillary muscles : 2.33 ± 0.53 in ECG LVH vs. 2.17 ± 0.44 in normal ECG, p = 0.013) (Table 5). But this finding might be attributable to overall hypertrophic change of myocardium in ECG LVH group. Minor papillary muscle variation might be considered as accessory papillary muscle if the mass is quite sizable. The composition of various number of papillary muscle is different between ECG LVH group and normal ECG group (Figure 3).

Table 4. Parameters of coronary computed tomographic angiography

	Electrical LVH (N = 134)	Normal ECG (N= 134)	p- value
LV mass(g)	162.7 ± 38.6	139.2 ± 28.7	<0.001
LV volume(g)	114.1 ± 30.8	105.0 ± 27.7	0.011
PM mass(g)	9.0 ± 3.4	7.6 ± 2.7	< 0.001
PM mass/total LV mass (%)	5.2 ± 1.4	5.1 ± 1.3	0.734
Total N of PM	2.33 ± 0.53	2.17 ± 0.44	0.013
Minimal distance of anterolateral PM to septum(cm)	1.80 ± 0.53	1.76 ± 0.45	0.499

LV, left ventricle; PM, papillary muscle mass

Table 5. Difference in number of papillary muscle

	Number of papillary muscle		
	2	3	4
ECG LVH	93 (70.5%)	35 (26.5%)	4 (3.0%)
Normal ECG	113 (84.3%)	17 (12.7%)	4 (3.0%)

ECG, electrocardiogram; LVH, left ventricular hypertrophy

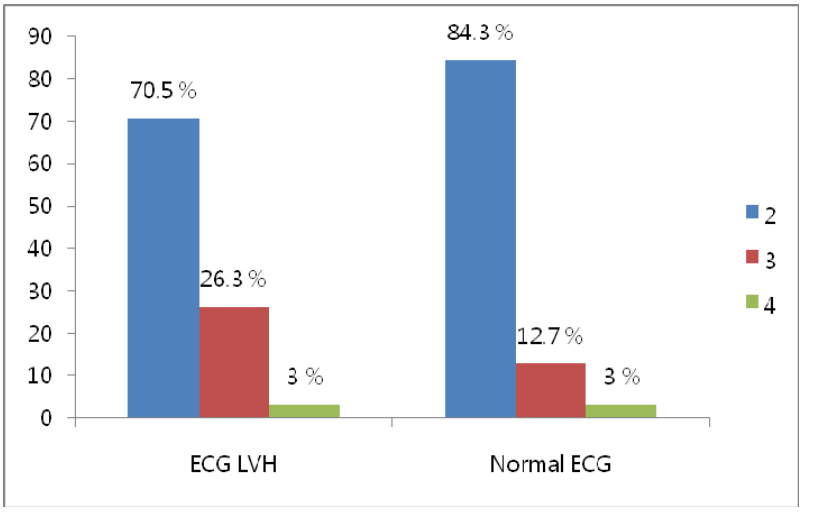

Figure 3. Number of papillary muscle identified by coronary computed tomographic angiography in 134 patients with ECG LVH and 134 normal ECG subjects.

IV. DISCUSSION

ECG-LVH represents electrical field of myocardium rather than anatomical LV mass (LVM). In other words, LVM is one of the characteristics of LVH but not the only one (12). Thus, it is the complex interplay of anatomical and electrophysiologic factors that affect the resultant QRS pattern because both affect the way the depolarization propagates though the ventricles. The ECG findings must be considered in relation with LVM and vice versa but not as a surrogate for LVM estimation because the information on electrophysiologic properties is complementary to the anatomical characteristics. Nevertheless,

there are many studies which demonstrate the relation between ECG-LVH and LVM. Anatomically, LVH is characterized by an increase in muscle mass, and mass is a function of both left ventricular chamber size and left ventricular wall thickness. So this study is sought to focus on the anatomical factor measures in coronary CTA.

As previous mentioned, the QRS voltages in ECG might be influenced by a various factors such as age, gender, race, hypertensive status and body habitus. In general population, the prevalence and incidence of LVH, determined by ECG or EchoCG, increases progressively by age (13, 14). In the case of obesity, it has been shown to be independently associated with LVH (15) and known as a factor which decreases the sensitivity of the ECG for detection of LVH (16). In consideration of gender, Rautaharju et al. (17) showed that breast tissue appears to have a practically negligible effect on ECG amplitudes in women. In any case, to remove the possible effect of age, gender and hypertensive status, we performed age, gender and hypertension status matched coupling between ECG-LVH group and normal ECG control group.

In spite of matching trial, we failed to figure out other anatomical factor that might contribute to ECG LVH except LV mass. Results of previous studies about solitary papillary muscle hypertrophy do not coincide with our results. This might be due to relatively small portion of solitary papillary in general population.

V. CONCLUSION

Left ventricular hypertrophy in ECG might reflect hypertrophy of left ventricle. But solitary papillary muscle hypertrophy does not seem attributable to ECG LVH. Images of coronary CTA at 65% RR interval look reliable for evaluation of LV mass and papillary muscle mass comparing to end-diastolic CT images.

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ABSTRACT(IN KOREAN)

관동맥 혈관 컴퓨터 단층촬영을 통한 심전도상의 좌심실 비대의 평가

< 지도교수 장혁재 >

연세대학교 대학원 의학과

김진선

서론: 심전도상에서 나타나는 좌심실 비대와 임상적 예후와의 연관 관계는 이미 널리 밝혀진 바 있다. 그러나 심전도의 좌심질 비대 진단 기준은 낮은 민감도를 나타내므로, 위양성 결과가 상당수에서 나타나게 된다. 본 연구에서는 관동맥 혈관 컴퓨터 단층촬영을 통하여 좌심실의 질량 외에도 다양한 해부학적 요인들이 심전도상의 좌심실 비대와 연관될 가능성을 확인해 보고자 하였으며, 이러한 요인들이 임상에서 나타나는 위양성 결과에 기여할 것으로 가정하였다.

대상 및 방법: 2003년 3월부터 2009년 4월까지 연세대학교

세브란스 병원을 내원하여 관동맥 혈관 컴퓨터 단층촬영을 시행한 4,300 명의 환자 중, 심전도상의 좌심실 비대 진단 기준을 충족한 154명의 환자를 가려내었다. 이 154명의 환자들과 나이, 성별, 체질량 지수가 유사한 154명의 정상 심전도 환자를 짝짓기하여 추려낸 뒤, 심전도상의 좌심실 비대군과 정상 심전도군사이의 좌심실 질량, 좌심질내 부피, 유두근 질량 및 중격까지의 거리를 관동맥 혈관 컴퓨터 단층촬영을 이용하여 측정하고 비교해 보았다.

결과: 65% RR 간격에서 측정한 좌심실 질량 및 좌심실내 부피는 심전도상의 좌심실 비대군에서 유의하게 큰 값을 보였다 (좌심실 질량, 심전도상의 좌심실 비대군: 162.7 ± 38.6, 정상 심전도군: 139.2 ± 28.7 , p <0.001; 좌심실내 부피, 심전도상의 좌심실 비대군: 114.1 ± 30.8, 정상 심전도군: 105.0 ± 27.7, p = 0.011). 유두근 질량은 심전도상의 좌심실 비대군에서 유의하게 크게 나타났으나, 좌심실 질량에 대한 비율로 보정하였을 때는 유의한 차이를 보이지 않았다 (유두근 질량, 심전도상의 좌심실 비대군: 9.0 ± 3.4, 정상 심전도군: 7.6 ± 2.7, p < 0.001; 유두근 질량/총 좌심실 질량 (%), 5.2 ± 1.4 대 5.1 ± 1.3, p= 0.734). 유두근의 평균 개수는 심전도상의 좌심실 비대군에서 유의하게 많은 것으로 나타났으나, 이러한 결과는 전반적인 심근의 비후에 의해, 가벼운 변이가 더하여 셈해졌을 가능성이 있을 것으로 사료된다 (유두

근의 평균 개수, 심전도상의 좌심실 비대군: 2.33 ± 0.53, 정상 심전도군: 2.17 ± 0.44, p = 0.013).

결론: 심전도상에서 나타나는 좌심실 비대는 실제 좌심실의 비후를 반영하는 것으로 보이며, 유두근의 단독 비후만으로는 심전도에서 나타나는 좌심실 비대의 원인이 되지 못하는 것으로 나타났다. 65% RR 간격에서 얻은 관동맥 혈관 컴퓨터 단층촬영의 영상은 이완기말 컴퓨터 단층촬영에 견줄 때, 좌심실 질량과 유두근의 질량 평가에 신뢰할 만한 검사 결과를 보여주었다.

핵심되는 말: 좌심실 비대, 관동맥 혈관 컴퓨터 단층촬영, 유두근