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Original article

Tricuspid annular motion velocity as a differentiation index of hypertrophic cardiomyopathy from hypertensive heart disease

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ABSTRACT

Background: Hypertensive heart disease (HHD) and hypertrophic cardiomyopathy (HCM) are the most frequently encountered entities presenting left ventricular hypertrophy in routine echocardiographic examination, and their differentiation is sometimes difficult. Abnormalities in right ventricular (RV) myocardium have been reported frequently in patients with HCM more than in those with HHD. We therefore hypothesized that tricuspid annular motion (TAM) velocity determined by pulsed tissue Doppler echocardiography can be used to detect RV dysfunction in HCM and discriminate these etiologies.

Methods: TAM velocities were compared among clinically stable patients with 60 HCM and 60 HHD patients as well as 60 age-matched healthy controls. Peak systolic, early diastolic (TAM-e′), and atrial systolic velocities were measured. RV myocardial performance index was measured by tissue Doppler method. To more accurately differentiate HCM from HHD, electrocardiographic findings and brain natriuretic peptide levels, which can both be examined simply and noninvasively, were investigated in addition to echocardiography.

Results: RV wall thickness of the HCM group was greater than the HHD group ($p = 0.092$), while there was no significant difference in RV myocardial performance index between the HCM and HHD groups ($p = 0.606$). TAM-e′ was significantly lower in the HCM group than in HHD and control groups ($p = 0.001$). To differentiate HCM from HHD, TAM-e′ was a powerful predictor as per multivariate logistic regression analysis (hazard ratio, 0.665; $p < 0.001$) of parameters other than those of left ventricular parameters, and the area under the receiver operating characteristic curve (AUC) was 0.686 and the best cut-off value was $\leq$8.0 cm/s (62% sensitivity, 65% specificity). Multivariate logistic analysis revealed that electrocardiographic ST–T changes were the next most effective marker for differentiating HCM after TAM-e′. When TAM-e′ and ST–T changes were combined, the AUC increased to 0.748.

Conclusions: TAM-e′ is a potentially useful index to differentiate HCM from HHD.

Introduction

Hypertrophic cardiomyopathy (HCM) and hypertensive heart disease (HHD) are common presentations of left ventricular (LV) hypertrophy in routine echocardiographic examination [1], and it is sometimes difficult to differentiate between them [2,3]. Approximately 44% patients with HCM present with right ventricular (RV) hypertrophy (RVH) [4]. Although RVH can occur in patients with HHD [5], an autopsy study revealed an increased incidence of abnormalities in the RV myocardium in patients with HCM than in those with HHD [6], indicating a potential difference in RV function.

The following parameters of RV systolic function were commented on in the American Society of Echocardiography guideline: peak systolic velocity of tricuspid annular motion (TAM), RV myocardial performance index (RVMPI), tricuspid annular plane systolic excursion, fractional area change, and RV strain [7]. Among these indices, the TAM velocity by pulsed tissue Doppler echocardiography (TDE) is a simple and reproducible

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technique. TDE is also available on all modern ultrasound equipment [7]. Therefore, we hypothesized that TAM velocity may help in differentiating HCM from HHD.

Methods

Study population

From 12,276 subjects who underwent echocardiography at the Ultrasound Examination Center of Tokushima University Hospital between December 2009 and December 2012, 70 HCM and 60 HHD patients who met the following criteria were randomly selected: (1) sinus rhythm, (2) LV ejection fraction of at least 55%, (3) without more than moderate valvular heart disease, (4) without coronary artery disease, (5) without pulmonary hypertension, and (6) without LV outflow tract obstruction. HCM and HHD were diagnosed as per the American College of Cardiology Foundation/American Heart Association guidelines [8] criteria using family history, past history, echocardiography [9,10], electrocardiography [11], coronary angiography, endomyocardial biopsy [12], magnetic resonance imaging [13], and genetic testing [14]. Coronary artery disease was confirmed by past history or coronary angiography. Pulmonary hypertension was confirmed by systolic pulmonary artery pressure $\geq$40 mmHg. Ten HCM patients concomitant with hypertension were excluded. Thus, 60 patients with HCM were analyzed. LV geometry in HCM was classified by Maron's classification [3].

Patients without abnormal electrocardiographic and echocardiographic findings were randomly extracted from a pool of patients who were referred to our Ultrasound Examination Center between August 2012 and December 2012 for presurgical cardiac evaluation before noncardiac surgery. In order to co-ordinate ages for the purpose of comparison between patients with HCM and HHD, 60 of these patients aged 61 $\pm$ 5 years were used as normal controls. The study protocol was approved by the ethics committee of the Tokushima University Hospital, and informed consent was obtained from all subjects.

Echocardiography

Echocardiographic examination was performed by high-end machine (Vivid E9, GE Healthcare, Horten, Norway; iE33, Philips Healthcare, Andover, MA, USA; Aplio 80, Toshiba Medical, Tochigi, Japan; ProSound α-10, Hitachi-Aloka Medical, Tokyo, Japan). Echocardiographic examinations were performed by six experienced sonographers who were blinded to the present study protocol but unblinded to the clinical information. LV wall thickness of interventricular septal wall and posterior wall were measured from short-axis view. LV mass index was measured by M-mode echocardiography in HHD and controls and by truncated-ellipsoid method in HCM [9]. M-mode echocardiography was used to obtain LV end-diastolic and end-systolic diameters and left atrial (LA) diameter. LV and LA volumes were calculated using the modified Simpson's rule from apical two- and four-chamber views. Pulsed Doppler indices were measured in the apical four-chamber view. Systolic pulmonary artery pressure (sPAP) was estimated by sum of peak tricuspid regurgitation pressure gradient measured with continuous-wave Doppler echocardiography and mean right atrial pressure. Mean right atrial pressure was estimated by inferior vena cava size and collapsibility as per previous guideline [7]. RV free wall thickness was measured at end-diastole by M-mode echocardiography from left parasternal window. From recordings of transmitral flow velocity pattern, the peak early (E) and late (A) diastolic flow velocities, and E-wave deceleration time were measured. From pulmonary venous flow velocity pattern, peak systolic (PVS), peak diastolic (PVD), and atrial reversal (PVA) flow velocities were obtained.

After conventional echocardiographic examination, TDE was performed from an apical four-chamber view to obtain mitral annular motion (MAM) velocity of the lateral side and TAM velocity of the RV free wall side. Peak systolic MAM velocity (MAM-s′), peak early diastolic MAM velocity (MAM-e′), and peak atrial systolic MAM velocity (MAM-a′) were recorded and measured [15]. TAM velocities were measured and assessed in accordance with the guideline for the echocardiographic assessment of the right heart in adults [7], and peak systolic (TAM-s′), early diastolic (TAM-e′), and atrial systolic (TAM-a′) velocities were measured. In addition, RVMPI was measured by TDE [7].

Electrograph and biochemical data

To more accurately differentiate HCM from HHD, electrocardiographic findings and brain natriuretic peptide (BNP) levels, which can both be examined simply and noninvasively, were investigated in addition to echocardiography [11,16]. Immediately before echocardiographic examination, patients had their blood drawn and underwent 12-lead electrocardiography. BNP levels were measured after quickly centrifuging collected blood. For the 12-lead electrocardiography, seven criteria (Table 1) were investigated to differentiate HCM and HHD [11,17]: (1) P wave abnormalities, (2) abnormal Q waves, (3) increased QRS voltage, (4) increased QRS duration, (5) ST–T changes, (6) conduction abnormalities, and (7) RVH.

Statistical analysis

Data analysis was performed using the SPSS software (version 19.0.0.2; IBM Corporation, Armonk, NY, USA). Data are presented as mean $\pm$ standard deviation (SD) for continuous variables and as percentages for categorical variables. Intraclass correlation coefficients and paired Student's *t*-test were used to assess reproducibility of TAM-e′. Comparisons among more than three groups were made by one-way analysis of variance, followed by Tukey's honestly significant difference test. Comparisons of BNP levels between HCM and HHD groups were made by the unpaired Student's *t*-test or Mann–Whitney test. Furthermore, categorical variables were compared using the χ^2 test. To identify predictors that could accurately differentiate between HCM and HHD and identify HCM more conclusively than abnormal echocardiographic LV geometry, we performed receiver operating characteristic (ROC) curve and logistic regression analysis for parameters other than those of echocardiographic LV geometry. Multiple regression analysis was used to identify the determinant of the diagnosis and was performed

Table 1
Criteria for electrocardiographic abnormalities.

P-wave abnormalities
Negative portion of the P wave in lead V1 $\geq$0.1 mV in depth and $\geq$0.04 s in duration, or peaked P wave in leads II and III or V1 $\geq$0.25 mV in amplitude.
Abnormal Q waves
$\geq$0.04 s in duration or $\geq$25% of the height of the ensuing R wave or a QS pattern in two or more leads.
Increased QRS voltage
Amplitude of R or S wave in limb leads $\geq$2 mV, S wave in lead V1 or V2 $\geq$3 mV, or R wave in lead V5 or V6 $\geq$3 mV.
Increased QRS duration
QRS duration >0.1 s.
ST–T changes
ST-segment depression or T-wave flattening or inversion in two or more leads.
Conduction abnormalities
Right or left bundle branch block, or first-degree atrioventricular block.
Right ventricular hypertrophy (RVH)
If any of the following three requirements were met: (1) R/S ration in lead V5 or V6 <1, (2) right axis deviation >90°, or (3) R/S ratio >1 and negative T wave in lead V1.