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Electrocardiogram signs of right ventricular hypertrophy may help identify pulmonary hypertension in patients with dilated cardiomyopathy

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ABSTRACT

Objective: To the authors' knowledge, limited data are available regarding the association between Electrocardiogram (ECG) signs of right ventricular hypertrophy (RVH) and pulmonary hypertension (PH) in patients with dilated cardiomyopathy (DCM). We aimed to assess the accuracy of the recommended ECG criteria of RVH for predicting PH in patients with DCM.

Methods: According to the definition of PH (mPAP ≥ 25 mm Hg), 35 patients with DCM were divided into 2 groups: DCM with PH (n = 22) and DCM without PH (n = 13). Right heart catheterization was performed in all patients. Seventeen parameters of RVH recommended by the AHA/ACCF/HRS for diagnosis of RVH on ECG were determinded.

Results: The following parameters were correlated with mPAP: R_{V1} > 6 mm, S_{V5} > 10 mm, R:S_{V6} < 0.4, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm and P_{II} amplitude. The following parameters were significantly different between DCM patients with and without PH: S in V_5 (S_{V5}) > 10 mm, S in V_6 (S_{V6}) > 3 mm, R:S ratio in V_5 (R:S_{V5}) < 0.75, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm, S > R inI, S > R inII and R:S_{V1} > R:S_{V3}, although results were no longer significant after correcting for multiple comparisons. High specificity (92.3–100%), lowsensitivity (31.8–50%), high positive predictive value, and low negative predictive value of established parameters of RVH were noted for predicting PH in patients with DCM.

Conclusion: Several ECG signs of RVH may be useful for in the diagnosis PH in patients with DCM.

1. Introduction

In the six World Symposium on Pulmonary Hypertension (PH), five groups of disorders that cause PH were identified, PH due to left heart disease(LHD) is the most common form of PH [1,2]. The presence and context of PH due to LHD is a well-established prognostic factor of morbidity or mortality in patients with DCM, and the incidence of cardiac death in patients with DCM with PH was >11-fold that in DCM patients without PH [3,4].

Transthoracic echocardiography is recommended as a screening test in the evaluation of suspected PH, and this will provide essential information regarding concomitant left-sided valvular or ventricular dysfunction, although echocardiography could underestimates the pulmonary artery systolic pressure (PASP) by previous study [5]. Right heart catheterization (RHC) is the gold standard for diagnosis of pulmonary hypertension (PH) and also for differential diagnosis between pre-capillary PH and post-capillary PH, which is essential or therapeutic decisions [1,6,7]. RHC and echocardiography in patients with PH can be technically demanding and often involves significant cost, RHC has been associated with a few complications. Thus, this invasive diagnostic procedure should be performed in expert centers [6]. Simple, non-invasive tools are needed to assist clinicians in the evaluation of patients with possible PH and help clinicians decide whether to proceed with additional further tests. An ECG is a simple diagnostic tool. ECG signs of PH are represented by surrogate parameters of RVH due to right

Abbreviations: BUN, blood urea nitrogen; CO, cardiac output; DBP, diastolic blood pressure; DCM, dilated cardiomyopathy; ECG, electrocardiogram; LAD, left atrial diameter; LHD, left heart disease; LVEDD, left ventricular end diastolic Diameter; LVEF, left ventricular ejection fraction; NPV, negative predictive values; NT pro-BNP, N-terminal fragment pro-brain natriuretic peptide; NYHA, New York Heart Association; mPAP, mean pulmonary artery pressure; PA, pulmonary arterial; PASP, pulmonary artery systolic pressure; PAWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension; PPV, positive predictive values; PVR, pulmonary vascular resistance; RHC, right heart catheterization; RVEDD, right ventricle end diastolic diameter; RVH, right ventricular hypertrophy; SBP, systolic blood pressure; TPG, transpulmonary gradient.

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ventricular pressure overload and the importance of ECG in the diagnosis of PH has already been established reported [8].

The underlying pathogenesis of PH due to LHD is not fully understood and is likely to be multifactorial [2,7,9]. The first organ directly affected by LHD is the lung. In response to physical and biological stressors, remodeling of the pulmonary circulation and parenchyma are responsible for contributing to the development of PH. Initially, right ventricular adaptation with hypertrophy and increased contractility compensate for the increase in pulmonary vascular resistance. Ultimately, right ventricular uncoupling to the demands of the pulmonary circulation leads to RV failure [10]. The previous study has suggested that an increase in RV mass in DCM not only associated with a consequence of the myopathic process itself but also burden of pulmonary artery pressure rise [11]. So the RV is the ultimate victim of these vascular processes [3]. Few studies have addressed the predictive value of ECG features of RVH in DCM patients with PH. The aim of our study therefore was to investigate the possibility of using the ECG signs of RVH to detect PH in DCM patients.

2. Methods

2.1. Patients

A total of 35 consecutive patients with DCM between October 2012 and June 2014 were retrospectively enrolled in the study. DCM was defined by the presence of both an LVEF < 50% (using the biplane Simpson's technique) and a dilated LV cavity in the absence of coronary artery stenosis > 50% (as determined by coronary angiography), valvular heart disease, arterial hypertension,and secondary cardiac muscle disease attributable to any known systemic condition [12]. PH due to lung or chronic thromboembolic disease was excluded. Clinical assessment,laboratory examination,echocardiography and coronary angiography were routinely performed. According to the definition of PH (mPAP ≥ 25 mm Hg) [1], 35 patients with DCM were divided into 2 groups: DCM with PH (n = 22) and DCM without PH (n = 13).

2.2. ECG

Standard 12-lead ECGs in the supine position (paper speed 25 mm/s, sensitivity 1 mV = 10 mm) were obtained. The ECGs were analysised by 2 independent observers blinded to the study result. Discrepancies were resolved by consensus. The current guidelines had list 24 ECG criteria for diagnosis of RVH [13]. All ECG criteria were checked in 35 patients, apart from the $R:S_{V1}$, $R:S_{V1} > R:S_{V3}$, $R:S_{V1} > R:S_{V4}$, ventricular activation time, and $R:S_{V5}$ to $R:S_{V1}$, which were checked in 34 patients because no R wave was present in lead V_1 in one patient. Because 10 patients had atrial fibrillation, P_{II} amplitude was checked in the other 25 patients. A retrospective analysis of the ECGs was performed.

2.3. Echocardiography

Patients were imaged in the left lateral decubitus position using a commercially available Philip IE-33 system equipped with a 3.5 MHz transducer. Two-dimensional grey-scale, pulsed, continuous, and color Doppler data were acquired on the same day before right heart catheterization. Left ventricular end diastolic Diameter(LVEDD), right ventricle end diastolic diameter (RVEDD) and left ventricular ejection fraction (LVEF) were determined according to the recommendations [14].

2.4. Right heart catheterization

Right heart catherization had been performed in all patients inserted from a jugular approach by use of a 6F Swan-Ganz catheter (Edwards Life sciences, USA). Cardiac output was estimated using direct Fick principle. PH was defined as mPAP ≥ 25 mm Hg during measurements at rest, without inhalation of nitric oxide and oxygen. Pulmonary arterial hypertension is defined by a mean PAP > 25 mm Hg at rest, by a PAWP<15 mm Hg and by PVR > 3 mm Hg/l/min (Wood units). PH due to left heart disease was defined as mPAP ≥ 25 mm Hg and PAWP > 15 mm Hg. Transpulmonary pressure gradient (TPG) was calculated by subtracting PAWP from mPAP, Pulmonary vascular resistance (PVR) was calculated by dividing TPG by cardiac output, The patient subgroup with no PH was defined as mPAP<25 mm Hg [4,6,7]. World Health Organization (WHO) Groups 1, 3, 4 PAH has been excluded [2].

2.5. Statistical analysis

Statistical analysis was performed with SPSS software (version 13.0, Chicago, Illinois). Continuous variables are expressed as the mean ± SD or as medians and interquartile ranges, normal distribution of variables were analyzed by Kolmogorov- Smirnov test. Independent sample *t*-test were used for comparison of the prevalence of individual RVH parameters between groups with PH and without PH. Statistical differences in categorical variables were evaluated by the chi-square test or Fisher's exact test. The sensitivity, specificity, and positive and negative predictive values (PPV and NPV) of the individual parameters showing statistically significant difference in frequency between groups were calculated. The relationship between ECG parameters of RVH and PH was estimated by Pearson or Spearman correlation tests. Adjusted P values were evaluated by Bonferroni correction. P value < 0.05 was considered statistical significance.

Table 1
Comparison of the baseline characteristics between groups with PH and without PH.

	With PH (n = 22)	Without PH (n = 13)	K-S* P value	P value
Age (years)	44.6 ± 11.6	53.6 ± 12.0	0.465	**0.036**
Female, n (%)	4 (18.2)	7 (53.8)		0.057
Disease duration (years)	5.49 ± 4.96	4.54 ± 5.82	0.285	0.613
History				
Atrial fibrillation, n (%)	8(36.4)	2(15.4)		0.259
NYHA class III, n (%)	9(40.9)	9(69.2)		0.164
NYHA class IV, n (%)	13(59.1)	4(31.8)		0.164
Admission vital signs				
SBP (mm Hg)	105.9 ± 16.9	111.0 ± 11.7	0.991	0.352
DBP (mm Hg)	68.6 ± 12.0	68.5 ± 7.47	0.703	0.983
Heart rate, beat/min	82.5 ± 14.7	78.7 ± 16.2	0.711	0.482
Laboratory values at admission				
Creatinine (μmol/L)	95.1 ± 48.0	104.1 ± 31.7	0.392	0.565
BUN (μmol/L)	7.56 ± 3.59	9.11 ± 3.04	0.130	0.221
NT-ProBNP (pmol/mL)	3606.5 ± 1312.5	1928.9 ± 1432.4	0.748	0.004
Echocardiography data				
LVEDD (mm)	71.6 ± 10.7	65.9 ± 7.71	0.493	0.106
LVEF (%)	28.7 ± 7.2	29.6 ± 7.2	0.923	0.725
RVEDD (mm)	28.8 ± 6.10	22.4 ± 3.30	0.987	**0.002**
LAD (mm)	50.7 ± 8.87	42.8 ± 6.47	0.882	**0.009**
Hemodynamic data				
mPAP (mm Hg)	37.9 ± 13.0	16.9 ± 3.35	0.559	**0.000**
PCWP (mm Hg)	23.4 ± 4.87	9.54 ± 4.89	0.804	**0.000**
TPG (mm Hg)	11.4 ± 6.35	6.69 ± 4.7	0.552	**0.023**
PVR ($dyn \cdot s \cdot cm^{-5}$)	335.9 ± 227.1	158.9 ± 84.6	0.189	**0.020**
CO (l/min)	3.39 ± 1.26	4.92 ± 1.22	0.850	**0.003**

*K-S, Kolmogorov-Smirnov test.
Abbreviations: PH, pulmonary hypertension, PA, pulmonary arterial, NYHA, New York Heart Association, SBP, systolic blood pressure, DBP, diastolic blood pressure, BUN, blood urea nitrogen, NT-proBNP, N-terminal fragment pro-brain natriuretic peptide, LVEDD, left ventricular end diastolic Diameter, LVEF, left ventricular ejection fraction, LAD, left atrial diameter, RVEDD, right ventricle end diastolic diameter, mPAP, mean pulmonary artery pressure, PCWP, pulmonary capillary wedge pressure, TPG, transpulmonary gradient, PVR, pulmonary vascular resistance, CO, cardiac output.

3. Results

3.1. Characteristics of the study population

The cohort consisted of 35 patients with DCM of which 11 (31.4%) women and 24 (68.6%) men. 2 patient fulfilled criteria of right bundle branch block. 3 patient had incomplete right bundle branch block. 3 patient fulfilled criteria of left bundle branch block. 6 patients had intraventricular conduction delays. 10 patients had atrial fibrillation. Table 1 summarizes the baseline characteristics of the cohort. The prevalence of individual parameters was compared between groups with PH and without PH. Patients with PH had a younger age, higher NT-ProBNP levels, larger RVEDD, larger LAD (left atrial diameter), higher mPAP, higher PAWP, higher TPG, higher PVR, and lower CO than patients without PH. There were no significant differences between the two groups in sex ratio, disease duration, prevalence of atrial fibrillation, blood pressure, heart rate, creatinine level, blood urea nitrogen level, LVEDD, and LVEF.

3.2. Prevalence of RVH in two groups according to ECG criteria

The ECG signs of right ventricular hypertrophy in DCM with PH was clearly evident (Fig. 1).The comparsion of the prevalence of RVH between the groups with or without PH was given in Table 2. The following parameters were significantly more common in the DCM with PH group than in the DCM without PH group: $S_{V5} > 10$ mm, $S_{V6} > 3$ mm,

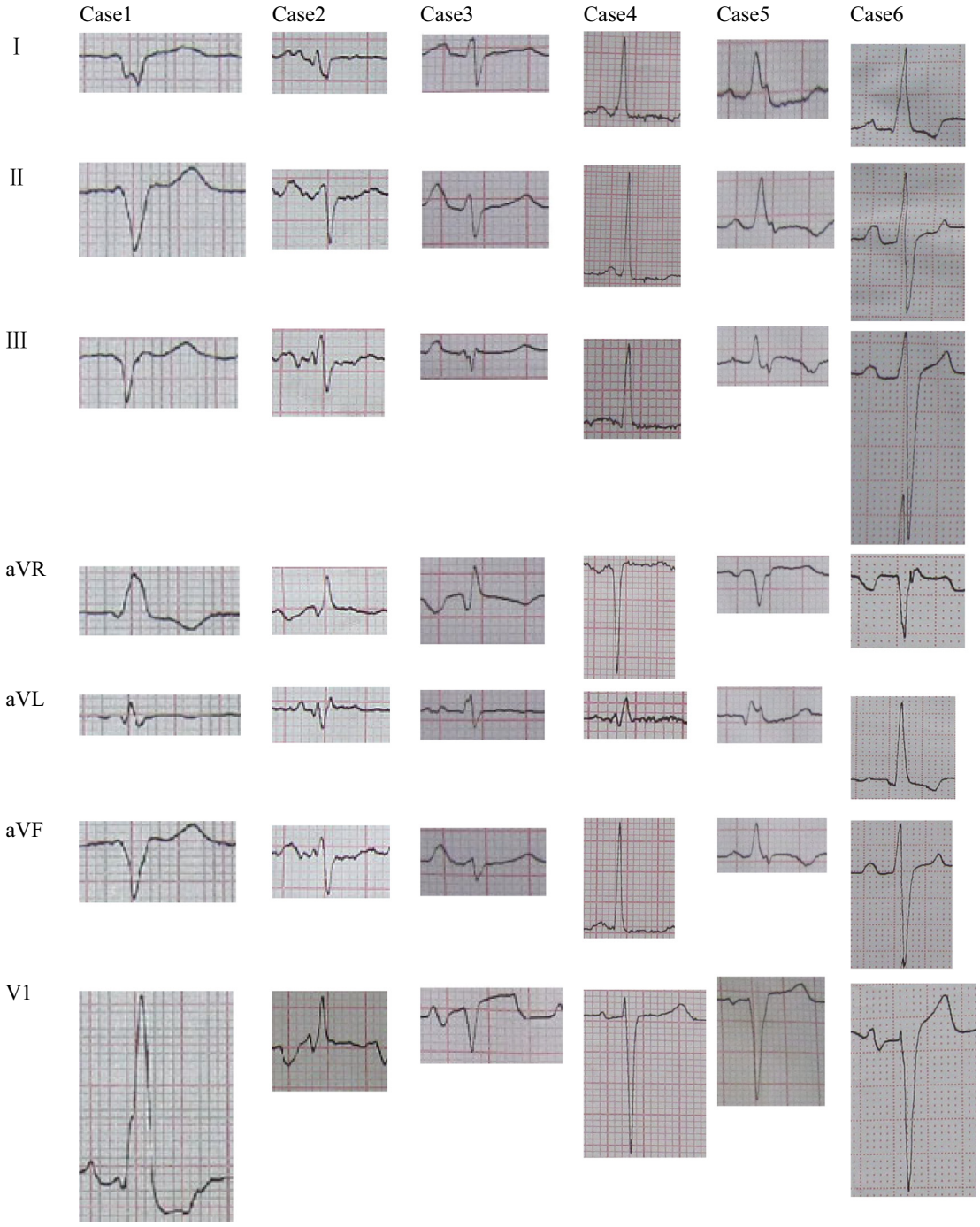

Fig. 1. Electrocardiogram signs of right ventricular hypertrophy in 3 case(1–3) of DCM with PH and 3 case(4–6) of DCM without PH.

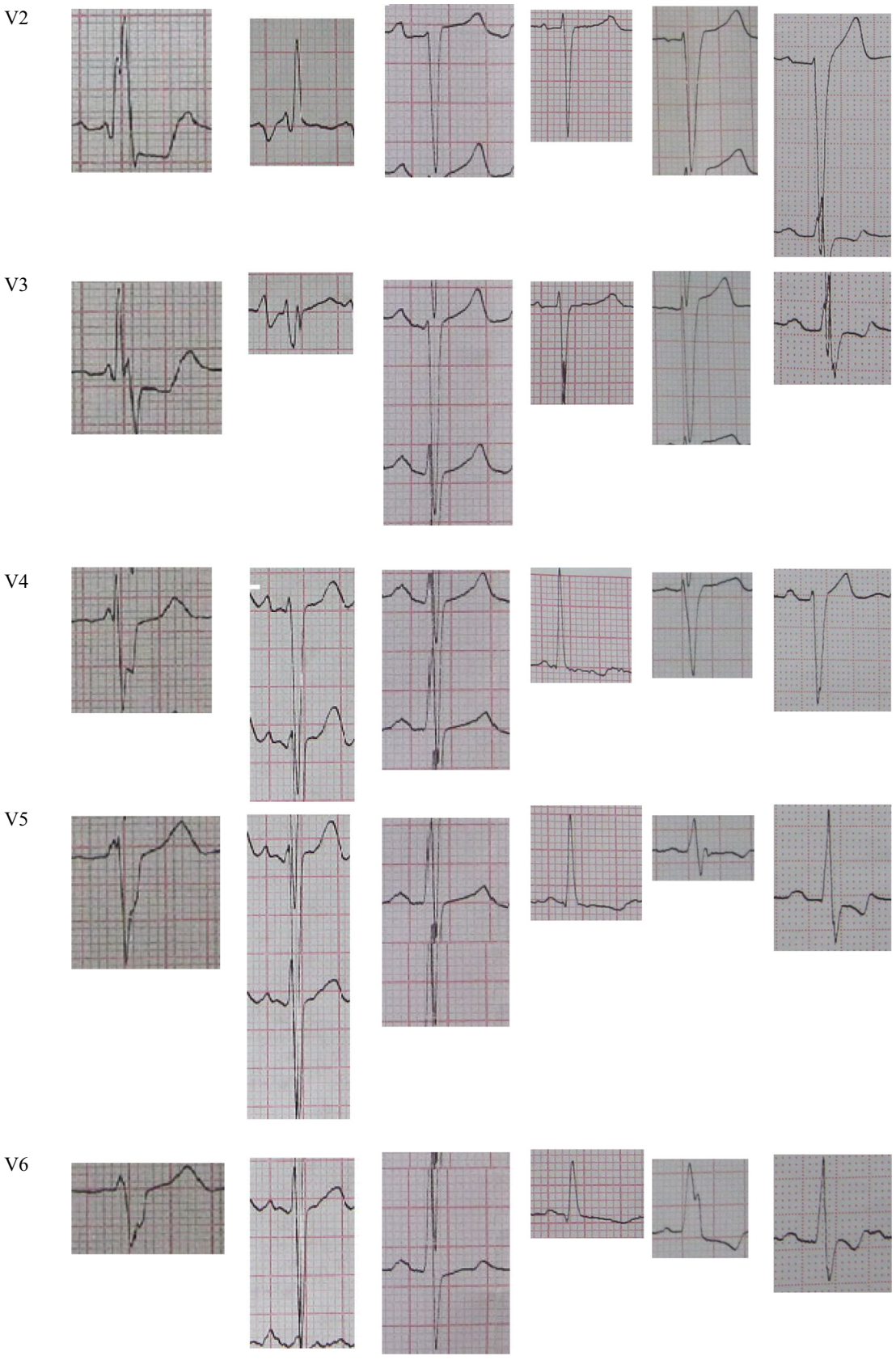

Fig. 1 (*continued*).

$R:S_{V5} < 0.75$, $R_{V1} + S_{V5\ or\ V6} > 10.5$ mm, S > R inI, S > R inII, $R:S_{V1} > R:S_{V3}$. There were no significant differences in the prevalence of the following parameters between the two groups: $R_{V1} > 6$ mm, $R:S_{V1} > 1$, $R_{aVR} > 4$ mm, $S_{V1} < 2$ mm, $R_{V5,6} < 3$ mm, $R:S_{V6} < 0.4$, $R:S_{V5}$ to $R:S_{V1}$ ratio < 0.04, $(R_I + S_{III})$-$(S_I + R_{III}) < 15$ mm, Max.$R_{V1,\ V2}$ + max. $S_{I,\ aVL}$-$S_{V1} > 6$ mm, R peak $_{V1}$ (QRS duration<0.12 s), RSR_{V1}, QR_{V1}, S > R

Table 2
The prevalence comparison of RVH between two groups.

	With PH (n = 22)	Without PH (n = 13)	P value	Adjusted P value*
R in V_1(R_{V1}) > 6 mm, n (%)	5(22.7)	0(0)	0.134	1.00
R:S ratio in V1($R:S_{V1}$) > 1, n (%)	6(27.3)	0(0)	0.081	1.00
S in V_5(S_{v5}) > 10 mm, n (%)	9(40.9)	0(0)	**0.013**	0.312
S in V_6(S_{v6}) >3 mm, n (%)	11(50.0)	1(7.8)	**0.013**	0.312
R in aVR(R_{aVR}) > 4 mm, n (%)	5(22.7)	0(0)	0.134	1.00
S in V1(S_{V1}) < 2 mm, n (%)	4(18.2)	0(0)	0.274	1.00
R in V_5 or V_6($R_{v5,6}$) < 3 mm, n (%)	3(13.6)	0(0)	0.279	1.00
R:S ratio in V_5 ($R:S_{v5}$) < 0.75, n (%)	7(31.8)	0(0)	**0.031**	0.744
R:S ratio in V_6($R:S_{v6}$) < 0.4, n (%)	1(4.5)	0(0)	1.000	1.00
R:S in V_5 to R:S in V_1 ratio < 0.04, n (%)	3(13.6)	0(0)	0.279	1.00
($R_I + S_{III}$)-($S_I + R_{III}$) < 15 mm, n (%)	17(77.3)	7(53.8)	0.258	1.00
Max. R in V1 or V_2 + max. S inIor aVL-S in V1 > 6 mm, n (%)	6(27.3)	1(7.8)	0.220	0.312
R in V1 + S in V_5 or V_6 > 10.5 mm, n (%)	9(40.9)	0(0)	**0.013**	1.00
R peak V1(QRS duration < 0.12 s), n (%)	12(54.5)	4(30.8)	0.293	0.312
$QR_{V1,}$ n (%)	3(13.6)	0(0)	0.279	1.00
RSR_{V1} present (>0.12 s), n (%)	1(4.5)	0(0)	1.000	1.00
S > R inI, n (%)	10(45.4)	0(0)	**0.005**	**0.12**
S > R inII, n (%)	11(50.0)	1(7.8)	**0.013**	**0.312**
S > R in III, n (%)	13(59.1)	10(76.9)	0.463	1.00
$S_I$and $Q_{III,}$ n (%)	3(13.6)	1(7.8)	1.000	1.00
$R:S_{V1}$ > $R:S_{V3,}$ n (%)	10(45.4)	1(7.8)	**0.027**	0.648
$R:S_{V1}$ > $R:S_{V4,}$ n (%)	6(27.3)	0(0)	0.081	1.00
Negative T-wave V_1 through $V_{3,}$ n (%)	2(9.1)	1(7.8)	1.000	1.00
P_{II} amplitude, n (%)	1(4.5)	0(0)	1.000	1.00

*Adjusted by Bonferroni correction.

in III, S_I and $Q_{III,}$ $R:S_{V1}$ > $R:S_{V4}$, Negative T-wave V1~V3, P_{II} amplitude, although results were no longer significant after correcting for multiple comparisons.

3.3. Relationship between ECG criteria of RVH and mPAP

The relationship between ECG criteria of RVH and mPAP and RV diameter were shown in Table 3. R_{V1}, Sv_5, $R:Sv_6$, R_{V1} + $S_{V5\ or\ V6}$ and P_{II} amplitude were correlated with mPAP.

3.4. Predictive values of ECG signs of RVH in diagnosing PH in patients with DCM

Sensitivity, specificity, positive and negative predictive values of ECG signs of RVH in diagnosing PH in patients with DCM were shown in Table 4. S_{V5} > 10 mm, S_{V6} > 3 mm, $R:S_{V5}$ < 0.75, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm, S > R inI, S > R inII and $R:S_{V1}$ > $R:S_{V3}$ had a low sensitivity (31.8–50%), but a high specificity (92.3–100%) in identifing DCM patients with PH.

Table 3
The relationship between ECG criteria of RVH and mPAP and RV.

ECG criteria	mPAP		RV	
	r	P	r	P
R in V_1	0.520	0.001	0.164	0.394
$R:S_{V1}$	0.007	0.968	0.481	**0.008**
S_{v5}	0.356	0.036	0.455	**0.013**
S_{v6}	0.328	0.055	0.499	**0.006**
R_{aVR}	0.124	0.477	0.465	**0.011**
S_{V1}	−0.126	0.471	−0.610	**0.000**
R_{v5}	0.140	0.424	−0.291	0.126
R_{v6}	0.092	0.600	−0.406	**0.029**
$R:S_{v5}$	−0.140	0.424	−0.024	0.900
$R:S_{v6}$	−0.346	0.042	−0.108	0.578
$R:S_{V5}$ to $R:S_{V1}$	0.052	0.768	0.060	0.759
($R_I + S_{III}$)-($S_I + R_{III}$)	−0.324	0.058	−0.379	**0.042**
$MaxR_{V1,V2}$ + $maxS_{I,aVL}$-S_{V1}	0.325	0.061	0.542	**0.003**
R_{V1} + $S_{V5,V6}$	0.553	0.001	0.408	**0.028**
R peak $_{V1}$	0.135	0.440	0.091	0.638
P_{II} amplitude	0.494	0.014	0.139	0.549
QRS duration (ms)	−0.142	0.416	−0.234	0.222
Frontal plane QRS axis	0.146	0.402	0.500	**0.006**

Table 4
Predictive values of ECG signs of RVH in diagnosing PH in patients with DCM.

ECG criteria	sensitivity	specificity	PPV	NPV
S in V5(S_{v5}) > 10 mm	40.9%	100.0%	100%	50%
S in V6(S_{v6}) > 3 mm	50.0%	92.3%	91.7%	52.2%
R:S ratio in V5($R:S_{v5}$) < 0.75	31.8%	100%	100%	46.4%
R in $_{V1}$ + S in V_5 or V_6 > 10.5 mm	40.9%	100.0%	100.0%	50%
S > R inI	45.5%	100%	100.0%	52.0%
S > R inII	50.0%	92.3%	91.7%	52.2%
$R:S_{V1}$ > $R:S_{V3}$	45.5%	92.3%	90.9%	50%

4. Discussion

In the present study, we first evaluated the value of ECG of RVH to detect PH in DCM patients. The chief findings of the present study were that: 1) Group with PH had a younger age, higher NT-ProBNP levels, larger RVEDD, larger LAD, higher mPAP, higher PAWP, higher TPG, higher PVR, and lower CO than Group without PH; 2).

The ECG parameters S_{V5} > 10 mm, S_{V6} > 3 mm, $R:S_{V5}$ < 0.75, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm, S > R inI, S > R inII, $R:S_{V1}$ > $R:S_{V3}$ were different between DCM patients with and without PH. 3) R_{V1} > 6 mm, Sv_5 > 10 mm, $R:Sv_6$ < 0.4, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm, P_{II} amplitude were correlated with mPAP; 4) S_{V5} > 10 mm, S_{V6} > 3 mm, $R:S_{V5}$ < 0.75, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm, S > R inI, S > R inII and $R:S_{V1}$ > $R:S_{V3}$ may be useful for in the diagnosis PH in patients with DCM.

Previous studies indicated that the presence of PH in DCM patients was associated with poor prognosis [15,16]. PH, assessed using echocardiography in DCM patients was associated with a history of right heart failure and NYHA class [15]. PH causes elevated RV wall stress and leads to RV hypertrophy as a consequence of RV remodeling. The importance of the right ventricle in patients who have LV systolic dysfunction due to DCM had been recognized [15]. Our results showed that DCM patients with PH had larger RVEDD than those without PH. Although the echocardiography was the most commonly used in assessed PH, the predicted value of ECG in diagnosing PH due to left heart disease assessed by echocardiography was reported first by Al-Naamani et al., with the conclusion that a low positive predictive values and negative predictive values in not only the ECG parameters of RVH based on lead V1, but also lead V5 or V6 [8]. In the present study, the ECG criteria of RVH based on lead V_5 or V_6 (including Sv_5 > 10 mm, Sv_6 > 3 mm, $R:S_{V5}$ < 0.75, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm) had higher prevalence in patients DCM with PH than without PH. Poor R-wave progression was seen in 16 patients (46%), similar with Wilensky et al. study [17] as the R_{V1} > 6 mm was found only in 5 patients (14%). We failed to detect the predicting value of ECG parameters of lead V_1, including R_{V1} > 6 mm, $R:S_{V1}$ > 1, S_{V1} < 2 mm, R peak $_{V1}$ (QRS duration<0.12 s), RSR_{V1} and QR_{V1} in diagnosing PH in patients with DCM.

The mean frontal QRS axis of >100° had a highly predictive value of RVH and moderate correlation with mPAP [18]. Lau et al. also concluded the QRS axis were significantly correlated with mPAP [19]. The frontal plane QRS axis was also correlation with RV(r = 0.500, P = 0.006), but no correlation with mPAP in our study. 7 DCM patients with PH had the mean frontal QRS axis of>110°. S > R inIand S > R inIIwere the sign of right axis deviation, this may be due to right ventricular hypertrophy/dilation, had a low sensitivity, but a high specificity in predicted the presence of PH in patients DCM.

Why do Sv_5 > 10 mm, Sv_6 > 3 mm, $R:S_{V5}$ < 0.75, R_{V1} + $S_{V5\ or\ V6}$ > 10.5 mm, S > R inI, S > R inII and poor R-wave progression have higher prevalence in our patients with PH than without PH? It is speculated that RVH due to PH leads to right ventricular dilatation with associated right atrial hypertension, hypertrophy or dilatation, the eventual

outcome is a picture of biventricular and biatrial enlargement in patients with ischaemic or non-ischaemic cardiomyopathy [20]. For unknown reasons, ventricular dilation causes the QRS vector to shift towards the transverse plane and away from the frontal plane, resulting in differential effects on QRS voltages in the chest and the limb leads described by Goldberger [21]. So-called Goldberger's triad consists of: 1). High precordial QRS voltages, defined as (S_{V1} or S_{V2}) + (R_{V5} or R_{V6}) ≥3.5 mV; 2). Relatively low limb lead QRS voltages, defined as total QRS amplitude (i.e. R + S) ≤ 0.8 mV in each of the limb leads; and 3). Poor R wave progression in the precordial leads V1 to V_3 or V_4 [22,23].

4.1. Limitation

The present study has several limitations. This was a retrospective study in a single center with a relatively small sample size. Our findings should be considered preliminary, and should be verified by larger population sample defined according to these specific criteria. Moreover, it should be noted that only a few of the recommended ECG criteria proved to be useful in the diagnosis of RVH in previous study [24], and most of the ECG criteria for RVH have high positive and low negative predictive value which means that a significant proportion of patients with RVH will be underdiagnosed using the ECG criteria [18]. So that, it may lead to low sensitivity of ECG signs of RVH in diagnosing PH in patients with DCM. Finally, in the present study, the ECG analysis included only assessment of RV hypertrophy criteria, biventricular hypertrophy was not assessed using cardiac magnetic resonance imaging and may influence some of our findings.

In summary, DCM patients with PH had worse clinical and hemodynamic parameters than those without PH. R_{V1} > 6 mm, Sv_5 > 10 mm, $R{:}Sv_6$ < 0.4, $R_{V1} + S_{V5\ or\ V6}$ > 10.5 mm were correlated with mPAP. The recommended ECG criteria based on the S wave amplitude in ECG lead V5 (S_{V5} > 10 mm, $R{:}S_{V5}$ < 0.75, $R_{V1} + S_{V5\ or\ V6}$ > 10.5 mm), S wave amplitude in V_6, the ratio of R wave amplitude to S inI, the ratio of R wave amplitude to S inII and $R{:}S_{V1}$ > $R{:}S_{V3}$ were useful for predicting were useful for in the diagnosis PH in patients with DCM.

Authors' contributions

CC and JL carried out the patient enrollment, data collection. ZL, XH, XY, OX and LW participated in the data collection and performed the statistical analyses. CC,JL and XL conceived of the study, and participated in its design and coordination and helped to draft the manuscript. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

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