Role of electrocardiographic changes in discriminating acute or chronic right ventricular pressure overload

Elektrokardiyografik değişikliklerin sağ ventrikül basınç yüklenmesini akut veya kronik olarak ayırt etmedeki rolü

Mehmet Mustafa Can, Olcay Özveren[1], Murat Biteker[2], Cihan Şengül[3], Ömer Uz, Zafer Işılak, Ata Kırılmaz

Department of Cardiology, GATA Haydarpaşa Education and Research Hospital, İstanbul-*Turkey*
[1]Department of Cardiology, Faculty of Medicine, Yeditepe University, İstanbul-*Turkey*
[2]Clinic of Cardiology, Haydarpaşa Numune Hospital, İstanbul-*Turkey*
[3]Clinic of Cardiology, Çamlıca Alman Hospital, İstanbul-*Turkey*

ABSTRACT

Objective: Pulmonary embolism (PE) and severe pulmonary stenosis (PS) are two distinct conditions accompanied by increased pressure load of the right ventricle (RV). Despite major advances in our understanding of the mechanisms of RV adaptation to the increased pressure, substantial gaps in our knowledge remain unsettled. One of much less known aspect of pressure overload of RV is its impact on electrocardiographic (ECG) changes. In this study, we aimed to study whether acute and chronic RV overload are accompanied by different ECG patterns.

Methods: Thirty-eight patients with PE underwent ECG monitoring were compared with 20 matched patients with PS in this observational retrospective study. ECG abnormalities suggestive of RV overload were recorded and analyzed in both groups. Logistic regression analysis was used to define the predictors of chronic RV overload.

Results: Among the ECG changes studied, premature atrial contraction (OR-12.2, 95% CI, 1.3-107, p=0.008), right axis deviation (OR-20.4, 95% CI 4.2-98, p<0.001), indeterminate axis (OR-0.11, 95% CI 0.02-0.44, p=0.001 0.11), incomplete right bundle branch block (OR-4.2, 95% CI, 1.1-15.4, p=0.02), late R in aVR (OR-8.4, 95% CI 2.1-33.2, p=0.001), qR in V1 lead (OR-8.3, 95% CI 1.2-74.8, p=0.03) were found to be the independent predictors of chronic RV pressure overload.

Conclusion: Our data indicate that the ECG changes that attributed to the acute RV pressure loading states may be more prevalent in chronic RV overload as compared with acute RV overload. *(Anadolu Kardiyol Derg 2013; 13: 344-9)*

Key words: Right ventricular pressure load, electrocardiography, disease process, regression analysis

ÖZET

Amaç: Sağ ventrikül (SV) fonksiyonu birçok kardiyopulmoner hastalıklarda klinik sonlanım olarak önemli rol oynar. Pulmoner emboli (PE) ve ciddi pulmoner darlık (PD) SV basınç yüklenmesi ile seyreden iki ayrı hastalıktır. SV' nin artmış basınç yüklenmesine karşı verdiği uyum mekanizmaları iyi bilinmesine karşın hala bazı tanımlanmamış boşluklar devam etmektedir. SV basınç yüklenmesinin elektrokardiyografik (EKG) olarak nasıl bir değişiklik yaptığı pek bilinmemektedir. Çalışmamızdaki amaç akut ve kronik basınç yüklenmesini EKG olarak ayrımının yapılabileceğini araştırmaktır.

Yöntemler: Bu retrospektif gözlemsel çalışmamıza 20 PD ve 38 PE tanısı kesinleşmiş hasta dahil edildi. SV yüklenmesini gösterdiği daha önceki çalışmalarda kabul edilmiş EKG kriterleri her iki grupta araştırıldı. Logistic regression analysis was used to define the predictors of chronic RV overload.

Bulgular: Bakılan EKG değişikliklerinden, prematür atriyal atım (OR-12.2, %95 CI, 1.3-107, p=0.008), sağ aks deviasyonu (OR-20.4, %95 CI 4.2-98, p=0.001), indetermine aks (OR-0.11, %95 CI 0.02-0.44, p=0.001), inkomplet sağ dal bloğu (OR-4.2, %95 CI, 1.1-15.4, p=0.02), aVR de geç R dalgası (OR-8.3, %95 CI 1.2-74.8, OR-8.4, %95 CI 2.1-33.2 p=0.001), V1' de qR dalgası (OR-8.3, %95 CI 1.2-74.8 p=0.02) SV kronik basınç yüklenmesini gösteren bağımsız prediktörlerdir.

Sonuç: PE için kabul edilen EKG kriterleri PD grubunda daha sık saptandı. Daha da ötesi istatiksel olarak anlamı bulunan EKG kriterlerin basınç yüklenmesinin zamanlaması hakkında sınırlı tanısal değeri olduğu saptandı. *(Anadolu Kardiyol Derg 2013; 13: 344-9)*

Anahtar kelimeler: Sağ ventrikül basınç yüklenmesi, elektrokardiyografi, hastalık süreci, regresyon analizi

Address for Correspondence/Yazışma Adresi: Dr. Mehmet Mustafa Can, Bağcılar Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği İstanbul-*Türkiye* Phone: +90 212 440 40 00 Fax: +90 212 440 42 42 E-mail: mehmetmustafacan@yahoo.com

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Introduction

Right ventricular (RV) function plays a central role in the clinical outcome in a wide variety of cardiopulmonary disorders. Acute pulmonary embolism (PE) and severe pulmonary stenosis (PS) are two distinct conditions accompanied by increased pressure load of the RV. RV adaptation to these diseases is complex and depends on many factors. The most important factors appear to be the type and severity of myocardial injury or stress, the time course of the disease (acute or chronic), and the time of onset of the disease process (newborn, pediatric, or adult years) (1). In acute pressure-overload states such as PE, an adult with a previously normal RV is incapable of acutely generating a mean pulmonary artery pressure 40 mm Hg, and RV failure occurs early in the presence of a significant embolic burden (2). PS is the example of chronic pressure load that is well tolerated by the RV which usually adapts well to pulmonary valve stenosis even when severe, with symptoms being unusual in children and adolescents.

Despite major advances in our understanding of the mechanisms of RV adaptation to the increased pressure, substantial gaps in our knowledge remain unsettled. Several indices including hemodynamic changes, imaging and, finally, laboratory biomarkers are well established, and routinely used for assessing the severity of the disease (3-6). In contrast, much less known aspect of pressure overload of RV is its impact on electrocardiographic (ECG) changes. ECG findings consistent with pulmonary hypertension and RV ischemia and strain, including complete and incomplete right bundle branch block (RBBB), ST-segment changes and T-wave inversions, the S1Q3T3 pattern, and nonsinus rhythm, had been observed more frequently in patients with RV pressure load (7-9).

Although previous studies showed that these ECG changes may provide suggestive or supportive evidence of RV overload by demonstrating RV hypertrophy and strain, and right atrial dilation, there is no available data to explain the disease process whether it is chronic or not.

Thus, the objective of the index study was to evaluate whether acute and chronic RV overload are accompanied by different ECG patterns.

Methods

Study design

An observational retrospective study.

Patient population

The echocardiographic data and cardiac catheterization data were used to identify all of the patients diagnosed with major PE and severe PS. The inclusion criteria of the study groups were:

1) Acute PE consisted of the following findings signifying RV dysfunction and/or pulmonary hypertension (PH) caused by PE:

a) echocardiographic findings indicating RV pressure overload and/or PH (RV dilation, paradoxical septal wall motion, loss of inspiratory collapse of the inferior vena cava, or tricuspid regurgitation jet velocity >2.8 m/sec or <2.5 m/sec in the absence of inspiratory collapse of the inferior vena cava) without evidence of mitral valve disease or left ventricular dysfunction

b) The study group considered for evaluation those patients who presented with acute symptoms onset, i.e those who were admitted to the hospital within 48 h after suffering symptoms related to major PE, and who had a complete 12- lead ECG recording.

Subjects with high-risk acute PE (hemodynamically unstable patients), chronic pulmonary disease with regular ongoing medication, history of previous APE, ongoing anticoagulation therapy, end-stage cancer (estimated life expectancy less than seven months) were excluded from the study.

2) Severe PS: The diagnosis of valvular PS with a normal aortic root was diagnosed in all patients with echocardiography and confirmed by cardiac catheterization. Patients with added anomalies were excluded. The severity of PS was defined as RV pressure above 50 mm Hg.

Twenty patients (12 males; mean age 32±8 years) with severe PS and 38 patients with acute PE (24 males; mean age 31±6 years) were included in this study.

As part of the diagnostic procedure on admission pulmonary angiography was performed in all patients with PE, confirming the presence of pulmonary thromboembolic obstruction of the major pulmonary arteries. On the other hand, all patients with severe PS underwent catheterization of the heart revealed the presence of the severity of the PS. A complete echocardiographic examination was available in all patients and revealed the presence of the RV pressure load and/or pulmonary hypertension.

The retrospective study has been approved by the Local Institutional Review Board and an informed written consent was obtained from each study patient before examinations.

Catheterization techniques

The hemodynamic catheterization study was performed with the aid of monitored intravenous sedation or general anesthesia. Pressure measurements were made with fluid-filled catheters and gradients were determined by the non-simultaneous pullback technique.

Doppler data and calculations

Echocardiographic examinations were performed using commercially available cardiac ultrasound systems and standard examination techniques. The Doppler signals analyzed included continuous wave Doppler maximum pulmonary valve velocity, pulse wave Doppler subvalvular pulmonary velocity, and continuous wave Doppler tricuspid valve regurgitation (TR) velocity. Instantaneous pressure gradients were calculated by using the simplified Bernoulli equation: maximum instantaneous

gradient [maximum Doppler gradient 4(V2)^2]. Maximum Doppler gradient corrected for proximal velocity (corrected maximum gradient) was calculated using the expanded equation 4 (V2^2 - V1^2). The RV-right atrial (RA) gradient was calculated as 4 times the square of the TR velocity [4(TR velocity)^2]. In the preceding equations V2: maximum pulmonary valve velocity (m/sec), V1: subvalvular pulmonary velocity (m/sec), and RV-RA gradient systolic gradient between RA and RV (mm Hg).

Doppler time interval was measured from the tricuspid valve inflow and right ventricular outflow Doppler tracings, as described by Tei et al. and others (10,11).

The interval 'a' from cessation to onset tricuspid valve inflow is equal to the sum of isovolumetric contraction time (ICT), ejection time, and isovolumetric relaxation time (IRT). Ejection time 'b' is derived from the duration of ventricular outflow Doppler velocity profile. The sum of ICT and IRT was obtained by subtracting 'b' from 'a'. The MPI was calculated as (a-b)/b. Isovolumetric relaxation time was measured by subtracting the interval 'd' between the R wave and cessation of ventricular outflow from the interval 'c' between the R wave and the onset of tricuspid valve inflow. Isovolumetric contraction time was calculated by subtracting isovolumetric relaxation time from (a-b). Five consecutive beats were measured and averaged for each measurement. The RV diastolic anterior wall thickness was measured from subcostal imaging and The RV base-to-apex shortening during systole was measured as the systolic displacement of the lateral portion of the tricuspid annular plane systolic excursion (TAPSE). TAPSE was recorded on the M-mode format under 2-dimensional echocardiographic guidance. Data were averaged over 3 beats (5 beats in cases of atrial fibrillation) (12).

Electrocardiography data

Standard 12-lead ECGs (D1 to D3, aVR, aVL, aVF, V1 to V6) were obtained within a median 24 hours from onset of diagnosis. Two investigators independently analyzed ECGs. The following ECG abnormalities were regarded as suggestive of RV overload (13, 14).

1) sinus tachycardia (>100 beats/min) or bradycardia (<60 beats/min); 2) atrial arrhythmias; 3) atrioventricular conduction abnormalities; 4) abnormal axis pattern (S1Q3T3 pattern, i.e. S waves in lead I and Q waves in lead III with T-wave inversion in III); 5) shift in the transition zone (R5S) to V5 or further leftward; 6) complete or incomplete RBBB; 7) peripheral low voltage (in the limb leads); 8) pseudoinfarction pattern (prominent Q waves) in leads III and aVF; 9) ST segment elevation 0.1 mV over the right (V2–V3) or the left (V4–V6) precordial leads; 10) ST segment depression ≥0.05 mV over the right or the left precordial leads; and 11) T-wave inversion over the right or the left precordial leads. Two characteristic ECG´s tracings of both PE ve PS can be seen on Fig. 1 and Fig. 2, respectively.

Statistical analysis

Statistical Package for the Social Sciences (SPSS version 11.0, SPSS Inc., Chicago, IL, USA) was used for statistical analyses. Mean±standard deviation was used for continuous variants, while percentages were used for categorical variants. Unpaired t-test was used to test the difference between the continuous variants which showed normal distribution between patient and control groups. Normal distribution was tested with Shapiro-Wilk test. Chi-square test was used to test the categorical variants. Logistic regression analysis was performed to determine the independent predictors of chronic pressure overload. A p value of <0.05 was considered significant for all tests..

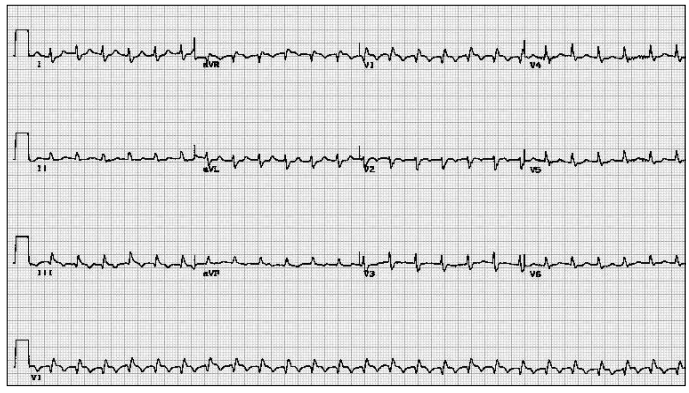

Figure 1. Sinus tachycardia and S1Q3T3 pattern in a patient with acute pulmonary embolism

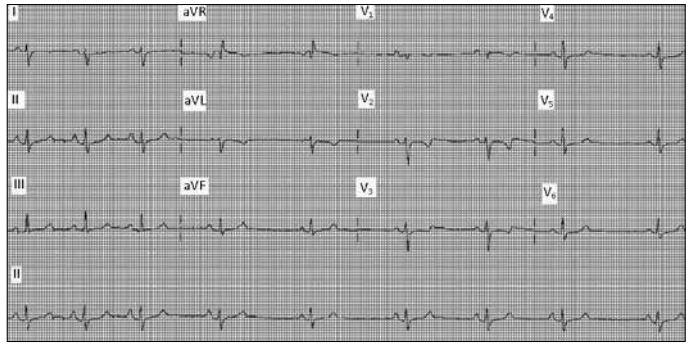

Figure 2. The electrocardiogram reflects tall and narrow P waves in lead II and at I and V_1

Results

Clinical characteristics of patients groups

Baseline demographic and clinical characteristics in PS and PE patients are presented in the Table 1. While age and gender matched well between PS and PE patients, patients with PS have higher systolic and diastolic and pulmonary artery pressures, TAPSE, MPI, RV 2D and free wall thickness in comparison to PE cohort. Number of the patients with dilated RV (RV appearing larger than the left ventricle from apical and/or subcostal view, or RV end-diastolic parameter >30 mm from the parasternal view) were similar in the PE group (acute RV pressure overloading group) and PS group (chronic RV pressure overloading group).

Electrocardiography

The differences in ECG indices between PS and PE cohort are outlined in the Table 2. Among the ECG changes studied,

Table 1. Baseline clinical characteristics of study participants

Variables	PS	PE	*p
Age, years	32±8	31±6	NS
Gender, n (%)			
Men	12 (60)	24 (63)	NS
Women	8 (40)	14 (37)	NS
Heart rate, bpm	68±8	98±12	<0.05
Systolic blood pressure, mmHg	120±8	92±8	<0.05
Diastolic blood pressure, mmHg	68±6	55±5	<0.05
RV systolic pressure, mmHg	110±4	70±6	<0.05
RV end-diastolic diameter >30 mm, n (%)	16 (80)	30 (78)	NS
Leftward interventricular septum shift, n (%)	15 (75)	29 (76)	NS
RV 2D free wall thickness subcostal (4-chamber view), cm	0.8±0.1	0.4±0.1	<0.05
TAPSE, mm	22±2	11±3	<0.05
RV MPI	0.42±0.08	0.56±006	<0.05

Data are presented as mean SD and number (percentage)
*Unpaired t-test and Chi-square test
MPI - myocardial performance index, PE - pulmonary embolism, PS - pulmonary stenosis, RV - right ventricle, TAPSE - tricuspid annular plane systolic excursion, 2D - two dimensional

premature atrial contraction (p=0.008), right axis deviation (p<0.001), indeterminate axis (p=0.001), incomplete right bundle brunch block (p=0.02), late R in aVR (p=0.001), qR in V1 (p=0.02), and P pulmonale (p=0.03) were significantly more common in patients with PS than in those with major PE. Premature atrial contraction (OR-12.2, 95% CI, 1.3-107, p=0.008), right axis deviation (OR-20.4, 95% CI 4.2-98, p<0.001), indeterminate axis (OR-0.11, 95% CI 0.02-0.44, p=0.001 0.11), incomplete right bundle branch block (OR-4.2, 95% CI, 1.1-15.4, p=0.02), late R in aVR (OR-8.4, 95% CI 2.1-33.2, p=0.001), qR in V1 lead (OR-8.3, 95% CI 1.2-74.8, p=0.03) were found to be the independent predictors of chronic RV pressure overload.

Discussion

The present study shows the ECG changes that attributed to the acute RV pressure loading states (Premature atrial contraction, right axis deviation, indeterminate axis, incomplete RBBB, late R in aVR, qR in V1) may be more prevalent in patients with chronic pressure loading states. RV pressure overload from acutely increased pulmonary arterial pressures has been implicated in the development of ECG abnormalities in patients with PE (15-18). Indeed, a variety of ECG changes have been suggested to have diagnostic value in patients with RV dysfunction but the findings are insensitive and non-specific (19). The most common findings are sinus tachycardia and nonspecific ST segment and T wave changes (18). Changes that strongly suggest acute pressure overload indicate strain on the right side of the heart; these changes include T wave inversion in precordial leads V1 through V4, transient RBBB, new right or left deviation of the QRS axis, sudden onset of atrial fibrillation or other atrial arrhythmias, and ECG signs of RV hypertrophy or right atrial enlargement. This pattern of right sided heart strain is usually accompanied by T wave inversion in the precordial leads. Although a number of studies have previously examined the prevalence of the associated ECG abnormalities in patients with acute RV pressure overload due to acutely increased pulmonary arterial pressures in patients with PE, most of them have been retrospective, have used relatively small numbers of patients, and have tended to focus only on patients diagnosed with PE or not compared the diagnostic value of ECG in appropriate in reference group (chronic overload). To our knowledge, this is the first study to examine the role of ECG in discriminating the pressure overload of RV whether it is acute or chronic. Our aim is not to discriminate the PE from PS based on single ECG data. PE could be easily discriminated from PS by the symptoms of PE (PE is usually presented with acute chest pain and acute dyspnea which are less common in PS in which chronic dyspnea is more common) and other imaging modalities i.e. Pure severe PS and/or PE patients were chosen to prevent the other contributing factors which may alter the ECG.

In our study model we investigated whether the ECG changes in acute pressure load states different from the chronic states. In contrast to previous data, it is shown that many of the classically described ECG changes are more prevalent in chronic RV pressure overload states than in patients with acute RV loading states. The relationship between ECG parameters and RV overload is well known (20-25). In spite of the great scattering of the ECG data, our study indicates that it is impossible to identify patients with pulmonary hypertension whether it is chronic or acute by ECG. Recognition of ECG findings of acute RV overload in patients could alter the estimated pretest probability of acute conditions such as PE and escalate the timing and type of treatment, and change the patient's disposition from the emergency department. However, very little evidence has been published to test the utility of the ECG as a diagnostic or risk-stratification instrument in undifferentiated patients for whom RV dysfunction neither diagnosed nor excluded.

Study limitations

There are a few limitations of the study worth mentioning. Due to experimental design, no definitive conclusions could be drawn based solely on the index data.

Although compatible in size with other similar investigations, this study was performed in a relatively small cohort, so chance represents a plausible alternative explanation. Finally, PS and PE are not unique diseases. RV dysfunction unquestionably has a complex pathogenesis, which is definitely not restricted to the observed changes of ECG. Obviously correlation of ECG indices with echocardiographic and hemodynamic variables is indeed important, however, our study is too small, and observational design precludes us to adequately assess these important links. These data most certainly should be confirmed in a larger, better randomized trial.

Table 2. Baseline ECG indices in PS and PE patients

ECG parameters	PE	PS	*p	OR, 95% CI
Rhythm disturbances, %				
Premature atrial contraction	5	39.1	0.008	12.2 [1.3-107]
Premature ventricular contraction	0	4.3	0.34	
Axis abnormalities, %				
Right axis deviation >90	15	78.3	<0.001	20.4 [4.2-98]
Indeterminate axis	30.4	80	0.001	0.11 [0.02-0.44]
QRS morphology, %				
Incomplete RBBB	40	73.9	0.02	4.2 [1.1-15.4]
Complete RBBB	10	13	0.75	
RBBB with associated ST-segment elevation and positive T wave in lead V1	0	8.7	0.17	
Low QRS voltage (<5 mm in limb leads)	15	8.7	0.52	
S wave in leads I and aVL >1.5 mm	40	65	0.10	
Shift in transition zone to V5	55	73.9	0.19	
Q waves in leads III and aVF but not in lead II	20	26.1	0.63	
S1Q3T3	20	39.1	0.17	
S slurred in V1 and V2	40	52.2	0.42	
Late R in aVR	30	78.3	0.001	8.4 [2.1-33.2]
Right ventricular hypertrophy criteria, %				
V1R	80	91.3	0.28	
V6S	50	69.6	0.19	
Upright T in V1	30	30.4	0.97	
Rsr in V1	40	30.4	0.51	
qR in V1	5	30.4	0.03	8.3 [1.2-74.8]
ST abnormalities, %				
ST change in any lead	45	34.8	0.49	
ST depression in any lead	25	17.4	0.54	
ST elevation in D1-2,V4-6	10	21.7	0.29	
ST depression in D1-2,V4-6	20	4.3	0.11	
T inversion in V2-3 T	45	34.8	0.49	
T inversion in V4-6 T	30	17.4	0.32	
T inverted in III and/or aVF	45	52.2	0.63	
T inverted in V1 or V2	65	73.9	0.52	
T flat or inverted in any lead	80	82.6	0.82	
Other, %				
P-Pulmonale	42.1	73.9	0.03	3.9 [1.1-14.3]

Data are presented as percentage

*Chi-square test and logistic regression analysis

CI - confident interval, ECG - electrocardiogram, OR-odds ratio, PE - pulmonary embolism, PS -pulmonary stenosis, RBBB - right bundle branch block

Conclusion

In conclusion, the ECG changes that attributed to the acute RV pressure loading states may be more prevalent in patients with chronic pressure loading states.

Conflict of interest: None declared.

Peer-review: Externally peer-reviewed.

Authorship contributions: Concept - M.M.C.; Design - M.M.C.; Supervision - M.M.C., M.B.; Resource - M.M.C., C.Ş., Ö.U., Z.I.;

Data collection&/or Processing - M.M.C., A.K.; Analysis &/or interpretation - Ö.U.; Literature search - M.M.C.; Writing - M.M.C.; Critical review - M.M.C., A.K.

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