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Changes in surface electrocardiogram in patients with chronic thromboembolic pulmonary hypertension undergoing pulmonary endarterectomy. Correlations with hemodynamic and echocardiographic improvements after surgery☆,☆☆

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Abstract

Background: The aim of the present study was to evaluate the changes of electrocardiographic (ECG) markers of right ventricular (RV) hypertrophy/overload in patients with chronic thromboembolic pulmonary hypertension (CTEPH) undergoing pulmonary endarterectomy (PEA).

Methods and results: We evaluated 99 CTEPH patients who underwent PEA. P wave amplitude in DII, R wave amplitude in V1 and the number of patients with negative T wave in V1–V3 decreased significantly at 1 month after surgery with no further change at 1 year, in parallel with the rapid improvement in right heart hemodynamics. S wave amplitude in V1, R:S wave ratio in lead V6 and prevalence of SIQIII pattern improved significantly at 1 year, in parallel with the progressive reverse remodeling of the right ventricle at echocardiography.

Conclusions: The study shows that some of the ECG markers of RV hypertrophy/overload better reflect RV hemodynamic overload while others better reflect the pathologic remodeling of the right ventricle.

Keywords: Pulmonary hypertension; Electrocardiogram

Introduction

Several electrocardiographic (ECG) criteria have been proposed to identify patients with right ventricular (RV) hypertrophy, mostly derived from the amplitude of R and S waves in precordial leads V1 and V6 [1]. ECG criteria for RV hypertrophy are also frequently associated with ST depression and T-wave inversion in right precordial leads, usually referred to as right ventricular strain and with P waves >2.5 mV in lead II, which are suggestive of right atrial enlargement. However, such ECG criteria were mostly derived from pathologic studies attempting to find ECG correlates of RV hypertrophy in patients with previously diagnosed, clinically advanced cardiopulmonary disease [2–6]. Several studies have evaluated the association between RV structure and ECG alterations in patients with either chronic thromboembolic pulmonaryhypertension (CTEPH) or pulmonary arterial hypertension (PAH), which could be useful for diagnostic or prognostic purposes [7–12]. However, it is still unknown to what extent the different ECG criteria reflect the changes in mass and volume of the right ventricle and to what extent they are related to the RV overload determined by the elevated pressures in the pulmonary circulation.

Unlike other types of pulmonary hypertension, chronic thromboembolic pulmonary hypertension (CTEPH) can be successfully treated with surgery. It is well demonstrated that pulmonary endarterectomy (PEA) allows dramatic improvements of the right heart hemodynamic profile immediately after surgery [13,14]. On the contrary, regression of hypertrophy and restoration of normal RV systolic function require more time and take place mainly during the first postoperative year [15,16].

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We hypothesized that significant ECG changes would occur in CTEPH patients undergoing PEA, and that some would be related to the rapid relief of pulmonary hypertension determined by surgery and some would be more strongly associated with the progressive improvement of RV structure over time. The aim of the present study was therefore to evaluate the temporal evolution of ECG markers of right ventricle hypertrophy/overload after PEA to clarify which ECG signs better correlate with hemodynamic improvement determined by surgery and which with the progressive reverse remodeling of the right ventricle as assessed by echocardiography.

Materials and methods

Patients

The present study is a retrospective evaluation of 99 CTEPH patients who underwent PEA between 2006 and 2010 in the Division of Cardiac Surgery of Policlinico S.Matteo Hospital and were followed up for at least 1 year after surgery. Exclusion criteria were previous myocardial infarction, significant left valvular heart disease, congenital heart disease, necessity of additional cardiac surgery, persistent atrial fibrillation/flutter, significant chronic lung disease; the rationale was that in such conditions the ECG markers of RV hypertrophy and/or strain could either be masked or counterbalanced by concomitant left heart disease or sustained by the presence of lung disease. Patients' clinical characteristics are shown in Table 1. All patients underwent right heart catheterization, echocardiographic examination and a 12-lead ECG recording before surgery, at 1 month and 1 year after surgery as part of the routine PEA follow-up protocol at our Hospital. At each time point all examinations were performed within the same day.

ECG

Conventional 12-lead ECG was recorded with patient in supine position with commercially available electrocardiographs at a paper speed of 25 mm/s, sensitivity of 1 mV = 10 mm, sample frequency of 500 Hz. In the first 50 patients, the ECGs were scanned with a dedicated program (AMPS scan ECG: L) and then analyzed with a semi-automatic ECG analysis program (AMPS Cal ECG) that takes into account a single beat; every ECG recorded and analyzed was checked by an expert cardiologist to confirm the automatic analysis. In the second cohort of 49 patients, the ECG analysis was manually performed by an experienced cardiologist. The following parameters, known as markers of right ventricle hypertrophy/overload were measured: QRS axis (°); P wave amplitude in DII (μV); R wave amplitude in V1 (μV); S wave amplitude in V1 (μV); S wave amplitude in V6 (μV); right bundle branch block (presence or absence); negative T waves in V1–V3 (presence or absence); R:S wave ratio in V6; SIQIII pattern (presence or absence).

Table 1
Patients' clinical characteristics at baseline.

Age in years: mean, (SD)	57.0 (15)
Male sex (n)	49
WHO class II/III/IV (%)	11%, 59%, 30%
Previous deep venous thrombosis (n)	60
Chronic obstructive pulmonary disease (n)	9
Systemic hypertension (n)	28
Diabetes mellitus (n)	4
Ischemic heart disease (n)	6
Chronic renal insufficiency (n)	5

Echocardiography

Ultrasound examinations were performed using a commercially available echocardiographic equipment (Vivid 7 System, Vingmed-General Electric, Milwaukee, WI). The following parameters were measured to study the right ventricle: RV free wall thickness, RV end-diastolic diameter, RV end-diastolic area, RV end-systolic area, RV fractional area change, calculated as (end-diastolic area − end systolic area)/end diastolic area, tricuspid annular plane systolic excursion, degree of tricuspid regurgitation.

Right heart catheterization

Right heart catheterization was performed through the right internal jugular vein by using a flow-directed, balloon-tipped Swan–Ganz catheter (7.5 F; Edwards Lifesciences, Irvine, Calif). The following parameters were measured or calculated: cardiac output, cardiac index, right atrial pressure, pulmonary artery pressure, capillary wedge pressure, pulmonary vascular resistance.

Statistical analysis

All analyses were performed with Stata 13 (StataCorp, College Station, TX, USA). Data were described as mean and standard deviation (SD) if continuous and counts and percent if categorical. Changes over time were assessed with general linear models with robust standard errors clustered on patients to account for intra-patient correlation. Spearman correlations coefficients (r) and 95% confidence intervals were computed to assess the association of changes in ECG and hemodynamic changes at 1 month and the Student t test was used to compare echocardiographic parameters between patients with and without S wave normalization at 1 year. Since the changes over time of ECG parameters were similar in the cohort of patients analyzed manually and in the cohort of patients in whom the semi-automatic ECG analysis program was used (data not shown), these two groups were pooled together for final analysis. A 2-sided p-value <0.05 was considered statistically significant.

Results

The patient's clinical characteristic are shown in Table 1: half of the patients was of male gender, mean age was 57 years, the vast majority had severe symptoms, 60% had a history of deep vein thrombosis, few of them had associated diseases such as chronic obstructive pulmonary disease, diabetes mellitus, ischemic heart disease, systemic hypertension.