Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/ihj

Original Article

Right ventricular outflow tract function in chronic heart failure

Bulent Deveci [a], *Kazim Baser* [b], *Murat Gul* [c], *Fatih Sen* [b], *Habibe Kafes* [b], *Sedat Avci* [d], *Orkun Temizer* [e], *Ozcan Ozeke* [b,*], *Omac Tufekcioglu* [b], *Zehra Golbasi* [b]

[a] *Akay Hospital, Department of Cardiology, Ankara, Turkey*
[b] *Turkiye Yuksek Ihtisas Training and Research Hospital, Department of Cardiology, Ankara, Turkey*
[c] *Van Ercis State Hospital, Department of Cardiology, Van, Turkey*
[d] *Tatvan State Hospital, Department of Cardiology, Bitlis, Turkey*
[e] *Burdur State Hospital, Department of Cardiology, Burdur, Turkey*

ARTICLE INFO

Article history:
Received 30 May 2015
Accepted 14 July 2015
Available online 10 November 2015

Keywords:
RVOT
Right ventricular function
Heart failure

ABSTRACT

Background: Heart failure (HF) is a common, progressive, complex clinical syndrome and a subset of HF patients has symptoms out of proportion to the resting hemodynamics and left ventricular ejection fraction (LVEF). Right ventricular (RV) function is a powerful prognostic factor in HF, but assessing it is a challenge because of the right ventricle's complex geometry.
Objective: The aim of this study was to investigate the clinical application value of RV outflow tract (RVOT) function measured by transthoracic echocardiography in HF patients.
Method: We prospectively investigated 36 chronic HF patients with dilated heart and LV systolic dysfunction and 21 healthy control subjects (normal ventricular function and ECG, and no cardiac risk factors). In addition to clinical and conventional echocardiographic parameters, RVOT size and fractional shortening (RVOT-FS) parameters were analyzed.
Results: The RVOT-FS was less in HF patients than healthy controls (18.8 $\pm$ 15.7 vs 55.8 $\pm$ 6.7, $p < 0.001$) and correlated positively with TAPSE ($r = 0.814$, $p < 0.001$) and inversely with SPAP ($r = -0.728$, $p < 0.001$) and functional capacity ($r = -0.842$, $p < 0.001$). There was a statistically significant difference in RVOT-FS among the HF subgroups with regard to NYHA functional capacity ($p < 0.001$), although there was no statistically significant difference with regard to LVEF.
Conclusion: Although the apparent discordance between LVEF and the degree of functional impairment in HF is not well understood, it may be explained in part by alterations in RV function. We found that the RVOT-FS was a noninvasive and easily applicable measure of RV function and might be used for a comprehensive evaluation and follow-up of HF patients with a combined assessment of RV by other RV parameters.

* *Corresponding author.*
E-mail address: ozcanozeke@gmail.com (O. Ozeke).
http://dx.doi.org/10.1016/j.ihj.2015.07.028

1. Introduction

Heart failure (HF) is a common, progressive, complex clinical syndrome with high morbidity and mortality.[1] Decreased exercise capacity is the main symptom in HF patients; therefore, the physician should provide an estimation of the functional class of the patient based on an assessment of the patient's daily activity and the limitations imposed by the patient's symptoms of HF. Although imperfect, the New York Heart Association (NYHA) classification has long been used to categorize HF patients, and this classification provides important prognostic information. Although HF is generally regarded as a hemodynamic disorder, many studies have indicated that there is a poor relation between measures of cardiac performance and the symptoms produced by the disease. However, a subset of patients with HF has symptoms out of proportion to the resting hemodynamics. Patients with a very low left ventricular ejection fraction (LVEF) may be asymptomatic, whereas patients with preserved LVEF may have severe disability. The apparent discordance between LVEF and the degree of functional impairment is not well understood but may be explained in part by alterations in ventricular distensibility, valvular regurgitation, pericardial restraint, cardiac rhythm abnormalities, and left atrial or right ventricular (RV) function.[1–3]

For many years, cardiologists were not interested in studying RV function and the role of the RV in HF. In recent years, RV function has been found to be a powerful prognostic factor in HF and pulmonary hypertension (PH),[4] but assessing it is a challenge because of the right ventricle's complex geometry, its interrelationship with the left ventricle (LV), its extreme sensitivity to loading conditions and to alterations in pulmonary pressure, and a limited understanding of underlying mechanisms of right HF.[5–7] Due to its widespread availability, echocardiography is used as the first line imaging modality for assessment of RV size and function; however, a single widely accepted and generally applicable index of RV function is not available.[5,7] The RV has 3 distinct features, i.e., the "inflow", "trabeculated apical", and "outflow tract" (infundibulum or conus) compartments, with different extent of contribution to the overall systolic function.[8–10] There are some data to suggest that the myocardium of the RV itself is intrinsically different to that of the LV.[11–13] The RV shortens in a circumferential direction during the isovolumic contraction controlled by subepicardial fibers and longitudinally during the ejection phase controlled by subendocardial fibers. The RV outflow tract (RVOT) has superficial circumferential muscle fibers, which causes radial RVOT contraction during systole.[11] It starts with a short contraction of the inlet region and ends with the contraction of the RVOT that is of longer duration. Since the onset of the RV ejection at RVOT occurs 25–50 ms after the contraction of the inflow tract, these result in overall peristalsis-like ventricular motion.[2,14–16] The RVOT function has been found to correlate closely with other anatomical, long axis as well as functional parameters and transtricuspid retrograde pressure gradient.[11–13]

Although the inlet part of the RV has a greater contribution to overall RV function compared with the infundibulum,[17–19] some studies have reported a possibility of using RVOT movement or contraction as a marker of RV systolic function.[11–13,20] Therefore, we aimed to evaluate the clinical and functional significance of RVOT in patients with HF.

2. Methods

We prospectively included HF patients. Inclusion criteria included a diagnosis of chronic HF for at least 12 months. All patients gave their written consent to participate in this study. All patients with arrhythmia, infectious disorders, malignant tumor, previous history of right heart failure or diagnosis of Group 1 PH (e.g., pulmonary arterial hypertension), Group 3 PH (PH associated with lung respiratory diseases and/or hypoxia), Group 4 PH (PH due to chronic thrombotic and/or embolic disease), and Group 5 PH (PH associated with a miscellaneous of rare diseases), and poor echocardiographic window were excluded. In keeping with current guidelines,[21] PH was defined using the pre-specified cut-off of PASP >50 mmHg at rest. In the present study, the term PH refers to an increased PASP associated with left heart diseases (Group 2 PH).

The study patients had a clinical diagnosis of HF made on the basis of compatible clinical presentation and history combined with documented systolic LV dysfunction (LVEF <50%) and dilation by transthoracic echocardiography. All patients were on standard HF therapy. Patients were divided into 3 groups according to their NYHA functional class (I, no symptoms with ordinary activity; II, mild limitation of physical activity and symptoms with ordinary physical activity; III, marked limitation of physical activity and symptoms with less than ordinary physical activity; and IV, symptoms with any physical activity or at rest). The patient groups were compared to a control group consisting of 21 age- and sex-matched, healthy control group.

Transthoracic echocardiography was performed by using a GE Vivid S5 with a 3.0 MHz phased-array transducer. Patients were examined in the left lateral decubitus position.

The SPAP was estimated by continuous wave Doppler evaluation of tricuspid regurgitation.[22,23]

RV long axis function via the tricuspid annular systolic excursion (TAPSE) was recorded from the apical four-chamber view with the M-mode cursor positioned at the free wall angle of the tricuspid valve. The distance between the tricuspid annulus and the RV apex was measured at end diastole and end systole of the same cardiac cycle, and TAPSE was calculated (in millimeters) as the difference between end-diastolic and end-systolic measurements.[24]

Two-dimensional echocardiograms of the parasternal short axis view at the level of the aortic root were obtained for the RVOT sizes and fractional shortening (RVOT-FS) values. M-mode recordings of the RVOT were obtained and dimensions were measured at end diastole (onset of the Q wave) and end systole (end of T-wave) using endocardial leading edge methodology. RVOT-FS was calculated as the percentage fall in RVOT diameter in systole with respect to that in diastole using the same M-mode images, as reported by Lindqvist et al.[13] (Fig. 1).

Data were analyzed by SPSS 16.0 (SPSS Inc., Chicago, IL, USA) software. Continuous variables were expressed as