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Left Ventricular End Diastolic Pressure and Serum Brain Natriuretic Peptide Levels in Patients with Abnormal Impedance Cardiography Parameters

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Background. Distinct hemodynamic patterns determined by impedance cardiography (ICG) have been found to be superior to clinical assessment for the identification of patients at risk for heart failure decompensation in the outpatient setting. Correlation of these hemodynamic patterns with serum brain natriuretic peptides (BNP) and left ventricular end diastolic pressure (LVEDP) has not been established. We evaluated the correlation of low-, intermediate- and high-risk groups for acute decompensation of heart failure (ADHF) as determined by ICG parameters with LVEDP and serum BNP.

Methods. Consecutive patients referred for cardiac catheterization with echocardiographic diagnosis of left ventricle dysfunction (systolic or diastolic) or history of congestive heart failure (CHF) underwent ICG evaluation, serum BNP measurement, and LVEDP by cardiac catheterization. Three groups at different levels of risk for ADHF were determined according to ICG parameters: thoracic fluid content (TFC) and stroke volume index (SVI); low risk (low TFC, high SVI), intermediate risk (low-low or high-high TFC and SVI, respectively), and high risk (high TFC and low SVI).

Results. Sixty three patients were included in the present study. Mean LVEDP and serum BNP levels were 20.2 $\pm$ 8.2 mmHg and 814 $\pm$ 1005 pg/mL, respectively, in the high-risk group in comparison to 12.3 $\pm$ 6.2 mmHg and 53 $\pm$ 38 pg/mL in the low-risk group ($p = 0.01$ and $p = 0.009$).

Conclusions. Patients with ICG parameters that represent high risk for ADHF have higher levels of serum BNP and LVEDP in comparison with patients who have intermediate- or low-risk ICG parameters for ADHF.

Key Words: Impedance cardiography, Decompensated heart failure, Hemodynamics, Brain natriuretic peptide.

Introduction

Episodes of acute decompensation of heart failure (ADHF) lead to an increase in morbidity and mortality (1) and are the most common Medicare diagnosis-related group (i.e., hospital discharge diagnosis) (2) and the major expenditure for heart failure care (3).

Findings of the physical exam have a good correlation with hemodynamic parameters in patients already in ADHF. On the other hand, the weeks preceding the acute decompensated state are characterized by a host of systemic compensatory mechanisms that cause disparities such as absence of rales and peripheral edema, despite significant elevation of ventricular filling pressures (4), and this negatively impacts the sensitivity and specificity of the physical exam to identify this state of impending decompensation. A need exists to evaluate patients beyond the physical exam to assess the hemodynamic status in the chronic compensated

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state. Invasive measurement of hemodynamic variables is the gold standard but its inherent risks make this approach unsuitable for periodic assessment of patients with chronic heart failure.

Newer implantable cardioverter defibrillators (ICD) and pacemaker devices are equipped with continuous monitors of intrathoracic impedance. The MID-HeFT study demonstrated an inverse correlation between intrathoracic average daily impedance (ADI) and intracardiac filling pressures (measured by pulmonary capillary wedge pressure) ($r = -0.61$, $p < 0.001$) for patients in the acute decompensated state during hospitalization. In the outpatient phase of the study, it was shown that intrathoracic ADI decreased by 12.3 ± 5.3% ($p < 0.001$) and 15.3 ± 10.6 days on average before symptom onset for all heart failure-related hospitalizations (5).

At present, intrathoracic impedance monitors are available only for patients already scheduled for an ICD or pacemaker implantation. A purely diagnostic device (Chronicle Implantable Hemodynamic Monitoring System; Medtronic, Inc., Minneapolis, MN) is currently under study and has not yet received FDA approval (6). This makes intrathoracic impedance monitors unavailable for the vast majority of patients at risk for ADHF. In this context, different methods have been devised in an attempt to non-invasively estimate hemodynamic status of patients and left ventricular function.

Serum brain natriuretic peptide (BNP) values have been found to correlate strongly with left ventricular end diastolic pressure (LVEDP), left ventricular ejection fraction (LVEF), and exercise performance (7).

Impedance cardiography (ICG) is a noninvasive method for estimating several hemodynamic variables including stroke volume index (SVI) and thoracic fluid content (TFC). ICG determines these parameters by measuring the change in conduction (impedance) of an alternating current as a function of fluid (blood) shifts in the thoracic cavity and the great vessels during the cardiac cycle (8).

In a recent study by Packer et al. (9), different levels of risk (low, intermediate, and high) for ADHF were identified based on serial outpatient ICG evaluations in 212 patients. A four-quadrant classification was developed by arranging ICG variables reflecting both inotropic qualities (SVI) and fluid status (TFC) to stratify different levels of risk for ADHF. Low-risk patients have low TFC and high SVI as opposed to high-risk patients who have high TFC and low SVI. The risk for the composite end-point in this study (death, hospitalization and emergency room visits) was 0.9% (95% CI 0.3−2%) in the low-risk group as opposed to 6.5% (95% CI 4.4−9.2%) in the high-risk group. ICG variables provided short-term prognostic information that appeared to be incremental to that available from a physician's clinical evaluation.

Further studies are needed to determine if the identification of patients at high risk can prevent episodes of ADHF. A thorough understanding of the physiopathology responsible for the different levels of risk conferred by ICG is needed in order to devise specific therapeutic interventions. In this study, we evaluated patients at different risks for ADHF as determined by ICG in order to determine LVEDP and serum BNP levels to contribute to this understanding.

Materials and Methods

Study Patients

Ninety consecutive patients referred to our Cardiac Catheterization Laboratory (CCL) for diagnostic catheterization and who fulfilled criteria for heart failure (Table 1) were included in the study between August and November of 2006. All patients gave written informed consent.

Exclusion criteria were height <120 or >230 cm, weight <30 or >155 kg, hemodynamically significant aortic regurgitation, acute coronary syndrome, left ventricular assist device, or pacemaker. Criteria to withdraw patients from the study included technical difficulties in measuring LVEDP, obtaining arterial blood sample for serum BNP and inability to perform ICG measurements prior to angiographic evaluation.

Five patients were excluded (three due to paced rhythm and two due to weight over the accepted range for ICG). Twenty two patients were withdrawn from the study after ICG was performed, 10 for inability to secure a blood sample prior to intravenous contrast administration and 12 due to lack of left ventricular end diastolic pressure

Table 1. Criteria for heart failure

Evidence by 2D echocardiographic study of either systolic or diastolic dysfunction, or clinical criteria for congestive heart failure (Framingham criteria, 2 major or 1 major plus 2 minor criteria).
Major criteria
Paroxysmal nocturnal dyspnea
Neck vein distension
Rales
Radiographic cardiomegaly (increasing heart size on chest x-ray)
Acute pulmonary edema
S3 gallop
Increased central venous pressure (>16 cm H_2O at right atrium)
Hepatojugular reflux
Weight loss >4.5 kg in response to treatment
Minor criteria[§]
Bilateral ankle edema
Nocturnal cough
Dyspnea on ordinary exertion
Hepatomegaly
Pleural effusion
Decreased vital capacity by one third from maximum recorded
Tachycardia (heart rate >120 beats/min)

[§]Minor criteria are acceptable only if they cannot be attributed to another medical condition (such as pulmonary hypertension, chronic lung disease, cirrhosis, ascites, or nephrotic syndrome).