Journal of Cardiology 67 (2016) 352–357

Contents lists available at ScienceDirect

Journal of Cardiology

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

Original article

Abdominal admittance helps to predict the amount of fluid accumulation in patients with acute heart failure syndromes

Tatsunori Taniguchi (MD)[a,b], Go Hamano (MD)[a], Masao Koide (MD)[a], Keiji Hirooka (MD, PhD)[a], Yukihiro Koretsune (MD, PhD, FJCC)[a], Hideo Kusuoka (MD, PhD, FJCC)[a], Tomohito Ohtani (MD, PhD)[b], Yasushi Sakata (MD, PhD, FJCC)[b], Yoshio Yasumura (MD, PhD, FJCC)[a,*]

[a] *Department of Cardiology, National Hospital Organization Osaka National Hospital, Osaka, Japan*
[b] *Department of Cardiovascular Medicine, Osaka University Graduate School of Medicine, Suita, Japan*

ARTICLE INFO

Article history:
Received 10 December 2014
Received in revised form 5 April 2015
Accepted 30 April 2015
Available online 23 June 2015

Keywords:
Abdominal admittance
Acute decompensated heart failure
Fluid accumulation

ABSTRACT

Background: Predicting fluid volume that needs to be removed in acute heart failure syndromes (AHFS) patients remains challenging. Thoracic admittance (TA), the reciprocal of thoracic impedance measured by bioelectrical impedance, reflects the amount of fluid in the thorax. Abdominal organs play an important role in AHFS as systemic fluid reservoirs. We investigated the relationship between abdominal admittance (AA) at the time of admission for AHFS and net fluid loss (NFL) during hospitalization.
Methods: Sixty-two consecutive patients hospitalized for AHFS [age 71 ± 10 years, left ventricular ejection fraction (LVEF) $39 \pm 17\%$] were studied. The admittance values, i.e. the reciprocals of the impedance values, were derived using a BioZ® (CardioDynamics, San Diego, CA, USA). The change in weight from admission to discharge was used as a surrogate of amount of NFL.
Results: At the time of admission, a significant correlation was detected between TA and AA ($r = 0.46$, $p = 0.0001$). TA at admission was significantly correlated with the LV structural variables (end-diastolic dimension and end-systolic dimension), and serum sodium level. AA at admission was significantly correlated with New York Heart Association (NYHA) class and plasma BNP, and also correlated with LVEF and variables related to systemic congestion [minimal inferior vena cava (IVC) diameter and tricuspid regurgitation grade]. Neither TA nor AA values were significantly correlated with weight at admission. During hospitalization, TA and AA declined from 44 ± 8 kΩ^{-1} to 36 ± 6 kΩ^{-1} ($p < 0.0001$) and from 74 ± 25 kΩ^{-1} to 56 ± 17 kΩ^{-1} ($p < 0.0001$), respectively. Weight fell from 60.1 ± 10.8 kg to 54.5 ± 9.4 kg ($p < 0.0001$), while NFL was 5.8 kg (range, 0.1–17.5 kg). In univariate analyses, the admission NYHA class, TA, AA, weight, and IVC diameter correlated with NFL. Multivariate analysis demonstrated that only admission weight [standardized partial regression coefficient (SPRC) = 0.596], AA (SPRC = 0.529), and NYHA class (SPRC = 0.277) were independent predictors of NFL.
Conclusion: Abdominal admittance measurement helps to predict the amount of fluid volume to be removed in patients with AHFS.

Introduction

Congestion, along with low tissue perfusion, represents an important therapeutic target in acute heart failure syndromes (AHFS) [1,2]. A primary goal in the treatment of congestion is the correction of fluid accumulation. However, assessing fluid accumulation status prior to diuretic therapy remains challenging [1,3]. Bioelectrical impedance is a relatively simple, quick, and noninvasive technique for estimating the fluid volume in an electric field of interest [4,5]. Several previous studies have suggested that thoracic admittance (TA), the reciprocal of thoracic impedance measured by bioelectrical impedance, reflects the amount of fluid in the thorax [6]. However, information available for abdominal admittance measurements is lacking in AHFS. Abdominal organs are considered to play an important role as a systemic fluid reservoir in patients with AHFS [7]. In the present

* Corresponding author at: Division of Cardiology, National Hospital Organization Osaka National Hospital, 2-1-14 Hoenzaka Chuo-ku, Osaka 540-0006, Japan. Tel.: +81 6 6942 1331; fax: +81 6 6943 6467.
E-mail address: yasumu@oph.gr.jp (Y. Yasumura).

http://dx.doi.org/10.1016/j.jjcc.2015.04.018
0914-5087/

study, we applied the bioelectrical impedance and determined abdominal admittance (AA) in patients with AHFS. The purpose of this study was to examine whether the AA measurement helps to assess fluid accumulation status prior to treatment in AHFS. We focused on the relationship between the AA at the time of admission for AHFS and subsequent net fluid loss (NFL) with heart failure (HF) therapy during hospitalization.

Methods

Study population

This is a dual-center, retrospective cohort study approved by the institutional ethics committees. The study protocol conformed to the principles outlined in the Declaration of Helsinki. The patient population consisted of 62 consecutive AHFS patients who were referred to Osaka National Hospital and Osaka University Hospital for intensive medical therapy over a period of 8 months. The diagnosis of AHFS was primarily based on signs and symptoms derived from a thorough history and physical examination [8]. Discharge was decided after confirming adequate relief of signs/symptoms related to HF. Patients with AHFS secondary to acute coronary syndromes, myocarditis, and cardiac tamponade, as well as those with pulmonary emboli, end-stage renal failure, and cardiogenic shock, were excluded from the study.

Study design

Transthoracic and abdominal impedance analyses were performed at admission and prior to discharge. Attending physicians were blinded to the impedance data. We first examined the association of the impedance data with echocardiographic parameters, plasma B-type natriuretic peptide (BNP) concentration, and other clinical variables, and compared the significance of each admittance measurement for assessing fluid accumulation status. Furthermore, we examined potential predictors at admission for subsequent NFL during hospitalization. The change in weight during patients' hospital stays was used as a surrogate for NFL, which was defined as the difference between admission weight and discharge weight (in kilograms).

Clinical assessment

Standard monitoring included the evaluation of clinical signs and symptoms related to HF, chest X-ray, and weight change. For ambulatory patients, weight was measured using appropriately calibrated scales just after admission and before administration of the first dose of diuretics and was subsequently determined daily in the morning before breakfast. For non-ambulatory patients, weight was measured using the Scale-Tronix 2001 sling scale model (Scaletronix, New York, NY, USA).

Impedance measurement

Bioelectrical impedance measurements were performed using a BioZ Impedance device (CardioDynamics, San Diego, CA, USA) with the patient in a supine to 15° upright position. The procedure was performed according to the indications of the National Institutes of Health Technology Assessment Conference Statement [4]. For thoracic admittance measurements, two sets of dual sensor patches were placed on the patient; one pair was placed at the base of the neck under each ear, and the other pair was placed at the mid-axillary line at the level of the xiphoid process as described elsewhere [6]. The outer sensors transmit a low-amplitude (4.0 mA), high frequency (70 kHz) current, and the inner sensors determine the impedance. After all data were collected, thoracic baseline impedance (tZ_0) was determined. TA was derived assuming $TA = 1000/tZ_0$. For abdominal admittance measurements, electrodes were placed bilaterally on the mid-axillary lines, at the levels of the xiphoid process and the anterior superior iliac spine (Fig. 1). This electrode configuration is a novel approach to measure abdominal impedance and has not been reported to date. Abdominal baseline impedance (aZ_0) was measured, and AA was determined as the reciprocal of baseline impedance, using the formula: $AA = 1000/aZ_0$. Both TA and AA were determined just after admission, after a certain improvement in patient's discomfort by nitroglycerin administration and/or bi-level positive airway pressure ventilation, especially in patients with flash pulmonary edema.

Intraobserver variability for AA values was calculated from repeat measurements of 10 randomly selected subjects, and interobserver variability was calculated from analysis of AA values in the same subjects by a second investigator.

Echocardiography

Each patient underwent two-dimensional (2-D) and Doppler transthoracic echocardiography (TTE) performed at bedside with a Vivid i device equipped with a 1.5–3.6 MHz phased-array sector scanner transducer (GE Healthcare, Milwaukee, WI, USA). All echocardiographic examinations were performed at baseline (on admission) and discharge. Left ventricular (LV) function variables, LV end-diastolic dimension, end-systolic dimension, and left atrium dimensions were routinely determined from 2-D TTE images in parasternal long-axis views. The LV ejection fraction was calculated using Simpson's method with two- and four-chamber apical views. The severity of mitral regurgitation (MR) and tricuspid regurgitation (TR) was semi-quantitatively graded from color-flow Doppler data [9]. The systolic pressure gradient across the tricuspid valve (TR pressure gradient) was derived from the peak velocity of TR using a modified Bernoulli equation. Systolic pulmonary artery pressure was estimated by adding the TR pressure gradient value to the estimated right atrial pressure [10]. As for right-heart function variables, right atrial pressure was estimated using the diameter of the inferior vena cava (IVC) and the response to changes in respiration [11].

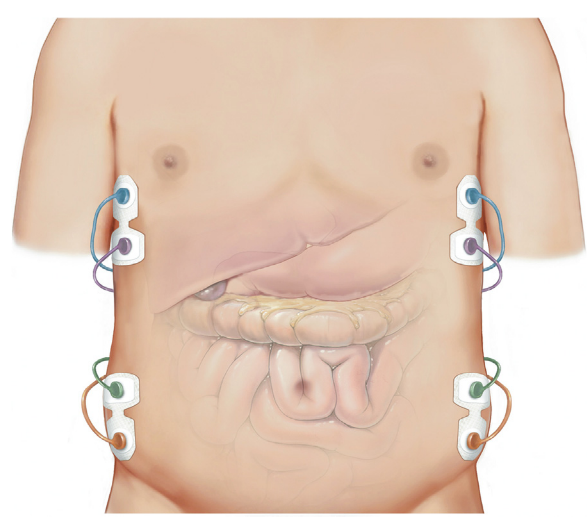

Fig. 1. Position of sensor electrodes for measurement of abdominal impedance. Abdominal impedance was measured using two sets of sensors placed bilaterally at the mid-axillary line, at the levels of the xiphoid process and anterior superior iliac spine.