Int J Clin Exp Med 2014;7(3):719-727
www.ijcem.com /ISSN:1940-5901/IJCEM1401036

Original Article

Evaluation of heart function with impedance cardiography in acute myocardial infarction patients

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Received January 17, 2014; Accepted February 20, 2014; Epub March 15, 2014; Published March 30, 2014

Abstract: To evaluate the capability of impedance cardiography (ICG) in reflecting the cardiac functions of acute myocardial infarction (AMI) patients. Methods: 99 inpatients with initial AMI were recruited. Venous blood was obtained for detection of N-terminal brain-type natriuretic peptide (NT-proBNP), B-Type natriuretic peptide (BNP) and c troponin-T (cTnT) followed by ICG. Thorax fluid capacity (TFC), pre-ejection period (PEP), left ventricular ejection fraction (LVEF), cardiac output (CO), stroke volume (SV), stroke index (SI), systemic vascular resistance (SVR), systemic vascular resistance index (SVRI), cardiac index (CI), end-diastolic volume (EDV) and systolic time ratio (STR) were measured. All these patients underwent ICG and echocardiography 2 days after surgery. Results: Our results indicated NT-proBNP and BNP were associated with SVR, SVRI, PEP and STR, independently ($P < 0.05$). cTnT was associated with SVR and SVRI ($P < 0.05$). And the outcomes showed correlation between ICG and echocardiography in SV, SI, EDV, LEVT, STR, LVEF ($P < 0.01$), CO and CI ($P < 0.05$). However, no correlation was noted in PEP. In addition, changes were also found in the blood pressure and heart rate 7 days after PCI. Conclusion: May be ICG data could reflect the early cardiac functions of AMI patients, but the accuracy of ICG in evaluating cardiac functions should be combined with detection of blood NT-proBNP, BNP and cTnT and echocardiography.

Keywords: Acute myocardial infarction, impedance cardiography, brain natriuretic peptide, echocardiography

Introduction

Acute myocardial infarction (AMI) is one of important problems threatening the health of subjects in developed countries and the incidence of AMI is also increasing in developing countries over year. AMI refers to the ischemia-induced myocardial necrosis due to coronary thrombosis. At the early stage of AMI, the compensatory contraction of myocardium around the necrotic tissues increases, the ventricular remodeling is absent and the contraction remains normal. With the development of AMI, the myocardial contraction decreases. However, the incidence of complications including re-infarction, sudden death and heart failure are still at a high level, even in patients survive from AMI following treatment. Therefore, it is imperative to stratify the risks at the early stage of AMI and to evaluate the heart function in a real-time manner.

A variety of methods have been developed to assess the myocardial function, including three-dimensional ultrasonography, magnetic resonance imaging and myocardial perfusion imaging. Although these techniques are comprehensive and accurate in the evaluation of myocardial function, detection with these methods requires skilled personnel and is costly. In China, massive application of these techniques is limited. Among numerous peripheral blood parameters, N-terminal brain-type natriuretic peptide (NT-proBNP), B-Type natriuretic peptide (BNP) and c troponin-T (cTnT) have been studied extensively in AMI and results reveal these parameters are closely related to the myocardial function. BNP levels are closely related to NYHA functional class and to individual prognosis [1, 2]. Impedance cardiography (ICG) is a noninvasive modality that uses changes in impedance across the thorax to assess hemodynamic parameters and has been widely applied in old patients with heart failure and hypertension patients, but seldom used in AMI patients. It allowed for assessment of thorax fluid capacity (TFC), pre-ejection period (PEP), left ventricular ejection fraction (LVEF), left ven-

Table 1. Patients' characteristics and information on treatment

Characteristics	n = 99
Age	62.3 ± 11.2
Gender (male, %)	76 (76%)
High presser	56 (56%)
Diabetes	61 (61%)
Hyperlipidemia	44 (44%)
Medication	
Aspirin	98 (99%)
ACEI or ARB	89 (89%)
β-blocker	78 (78%)
Ca^{2+} antagonist	68 (68%)
Diuretic	23 (23%)
Statins	98 (99%)
Nitrate drug	99 (100%)

Note: The main risk factors, characteristics and treatment about the recruited patients. n: number; ACEI: angiotensin-converting enzyme inhibitors; ARB: angiotensin receptor blocker.

tricular ejection time (LVET), cardiac output (CO), stroke volume (SV), stroke index (SI), systemic vascular resistance (SVR), systemic vascular resistance index (SVRI), cardiac index (CI), end-diastolic volume (EDV) and systolic time ratio (STR). CO, CI, SV and SI can reflect the myocardial blood flow; SVR and SVRI can reflect systemic vascular resistance and cardiac afterload; PEP, LVEF, LVET and STR can reflect myocardial contraction (prolonged PEP, LVET shortening, increase of STR and decrease of LVEF represent the compromised cardiac function); TFC can reflect the extravascular, intravascular and chest water content [3, 4] and represent the degree of heart failure indirectly. With the deterioration of heart failure, the blood volume increases resulting in increase of cardiac preload and TFC. A previous study showed that before and after cardiac rehabilitation in heart failure patients, impedance cardiography revealed a significant change in STR, LVEF, TFC and PEP [5, 6]. While another study indicated that in hospitalized patients with advanced heart failure, ICG provided some information about CO but not left-sided filling pressures, which had no prognostic utility in this patient population [7].

Based on the findings above, the present study was designed to discuss the correlation between NT-proBNP, BNP, cTnT and ICG and the correlation between ICG and echocardiography and to evaluate the role of ICG in the assessment of myocardial function in AMI patients in early stage.

Material and methods

Subjects

A total of 99 patients undergoing PCI in the department were recruited from October 2010 to March 2012. There were 75 males and 24 females with a mean age of 62.3 ± 11.2 years (range: 38~78 years). The inclusion criteria were as follows: patients had no myocardial diseases, valvular diseases, ventricular septal defect, atrial fibrillation, cardiogenic shock (systolic pressure < 80 mmHg); patients had initial AMI; the ischemic chest pain lasted for 30 min; electrocardiography showed ST elevation myocardial infarction (STEMI), ST elevation ≥ 0.1 mV (limb leads) or > 0.2 mV (precordial leads) in adjacent 2 or more contiguous leads or new left bundle branch block. For patients with non-ST elevation myocardial infarction (NSTEMI), ST segment depression > ~l mV (or elevation in lead V1 or avR) or symmetric T wave inversion was noted in electrocardiography; the levels of cardiac markers at 4 h after disease onset support the diagnosis of AMI; all patients underwent PCI. Written informed consent was obtained before study. The exclusion criteria were in patients had cardiac insufficiency or old myocardial infarction, patients had renal failure on hospitalization; the height was lower than 120 cm or higher than 230 cm (Instruments limited); the body weight was higher than 155 kg or lower than 30 kg (Instruments limited); patients had concomitant severe aortic insufficiency; patients received pacemaker implantation; patients had concomitant pleural effusion or pulmonary diseases. The main risk factors and characteristics after hospitalization are listed in **Table 1**.

Instruments

ICG was performed with noninvasive hemodynamic detector (Bioz.com; CardioDynamics, San Diego, CA, USA) and echocardiography was done with ultrasound scanner (Vingmed Vivid Seven, General Electric-Vingmed, USA).

Bioimpedance cardiography

Patients were rested in the supine position for at least 15 min before data were collected.

Table 2. ICG data and blood NT-proBNP, BNP and cTnT

Parameter	X ± (s)	Normal range
HR (bpm)	76.82 ± 3.15	58-86
SBP (mmHg)	124.25 ± 3.43	100-140
DBP (mmHg)	72.93 ± 2.26	60-90
SV (ml)	61.26 ± 3.94	65-121
SI (ml/m^2)	33.43 ± 1.76	35-65
CO (l/min)	4.68 ± 0.21	4.7-7.8
CI (l/min/m^2)	2.55 ± 0.08	2.5-4.2
SVR (dyne * s * cm^{-5})	1616.61 ± 159.28	742-1378
SVRI (dyne * s * cm^{-5} * m^2)	3157.93 ± 197.35	1337-2483
VI (/1,000/s)	28.96 ± 1.88	33-65
ACI (/100/s^2)	51.64 ± 3.15	70-150
PEP (ms)	86.04 ± 3.30	-
LVET (ms)	301.36 ± 7.45	-
STR (-)	0.32 ± 0.02	0.3-0.5
LCWI (Kg * m/m^2)	2.88 ± 0.18	3.0-5.5
TFC (/kOhm)	31.59 ± 1.42	30-50
BNP (pg/ml)	799.23 ± 91.62	< 100
NT-proBNP (pg/ml)	6530.18 ± 916.31	< 270
cTnT (ng/ml)	2.00 ± 0.22	< 0.01

Note: HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; SV: stroke volume; SI: stroke index; CO: cardiac output; CI: cardiac index; SVR: systemic vascular resistance; SVRI: systemic vascular resistance index; VI: velocity index; ACI: acceleration index; PEP: pre-ejection period; LVET: left ventricular ejection time; STR: systolic time ratio; LCWI: left cardiac work index; TFC: chest fluid capacity; BNP: B-type natriuretic peptide; NT-proBNP: N-terminal brain-type natriuretic peptide; cTnT: c troponin-T.

Four dual ICG sensors were placed: above the base of the neck and under each ear, and one on either side of the thorax in the mid-axillary line at the level of the xiphoid. A cable with eight ICG lead wires was attached to the individual sensor sites. An integrated oscillometric blood pressure cuff was connected to the patient's arm. The recording was performed for 10 min, and an average ICG status report was stored for analysis.

Echocardiographic measurements

Patients were imaged in the left lateral decubitus position using an ultrasound scanner system (Vingmed Vivid Seven, General Electric-Vingmed, USA). Images were obtained with a 3.5 MHz transducer, at a depth of 16 cm in the parasternal and apical views (standard long-axis and four-chamber images). Standard two-dimensional and colour Doppler data, triggered to the QRS complex, were saved in a cine-loop format. For each measurement, we averaged three cardiac cycles for patients in sinus rhythm and five for patients in atrial fibrillation. Measurements were obtained by two expert independent observers. End-diastolic and end-systolic left ventricular volumes and EF were determined by manual tracing of end-systolic and end-diastolic endocardial borders using apical four- and two-chamber views, employing the Simpson method for biplane assessment.

NT-proBNP, BNP, cTnT test

The venous blood was obtained for detection of NT-proBNP, BNP, cTnT, liver and kidney functions followed by ICG measurements. NT-proBNP was measured using N-terminal pro-brain natriuretic peptide, NT-proBNP ELISA kit, BNP detected with radioimmunoassay (phoenix USA), cTnT determined by an automatic electrochemiluminescence immunoassay analyzer (Eleesys 2010; Roche, Switzerland) with corresponding reagents. The associations between parameters determined by noninvasive hemodynamic monitor and blood NT-proBNP, BNP as well as cTnT were evaluated. The patients undergoing ICG at 48 hours after surgery followed by immediate echocardiography and the SV, SI, CO, CI, EDV, PEP, LVET, STR and LVEF were obtained. In addition, the relationships between impedance cardiography and echocardiography were also assessed based on the parameters (SV, SI, CO, CI, EDV, LEVT, STR, LVEF and PEP) (**Tables 2**-**4**).

Statistical analysis

Statistical analysis was performed with SPSS version 18.0. Data were expressed as mean ± standard deviation. Spearman correlation analysis was done to evaluate the correlation between the parameters determined by noninvasive hemodynamic monitor and blood NT-proBNP, BNP as well as cTnT. Linear regression analysis between variables was performed with a least square fitting routine (OriginPro 7.0, Microcal, USA) to assess the relationship between impedance cardiography and echocardiography. In addition, t test was used to compare the changes in parameters before

Table 3. Correlation analysis of ICG data and blood BNP, NT-proBNP and cTnT

Parameter	BNP	NT-proBNP	cTnT
HR (bpm)	0.187	0.241	0.141
SBP (mmHg)	0.256	0.187	0.104
DBP (mmHg)	0.306	0.244	0.266
SV (ml)	-0.898**	-0.736**	-0.741**
SI (ml/m^2)	-0.634**	-0.552**	-0.515**
CO (l/min)	-0.351*	-0.378*	-0.434**
CI (l/min/m^2)	-0.220	-0.346*	-0.312
SVR (dyne * s * cm^{-5})	0.865**	0.806**	0.669*
SVRI (dyne * s * cm^{-5} * m^2)	0.953**	0.769**	0.807**
VI (/1,000/s)	-0.235	-0.229	-0.158
ACI (/100/s^2)	-0.027	-0.143	-0.009
PEP (ms)	0.583**	0.433*	0.328
LVET (ms)	-0.895**	-0.813**	-0.825**
STR (-)	0.344**	0.325*	0.199
LCW (Kg * m)	-0.085	-0.231	-0.122
LCWI (Kg * m/m^2)	-0.210	-0.155	-0.098
TFC (/kOhm)	0.231	0.109	0.156

Note: Our results indicated NT-proBNP was negatively related to SV, SI, CO, CI and LVET, but positively associated with SVR, SVRI, PEP and STR. BNP was negatively related to SV, SI, CO and LVET, but positively associated with SVR, SVRI, PEP and STR. cTnT was negatively related to SV, SI, CO and LVET, but positively associated with SVR and SVRI. **$P < 0.01$; *$P < 0.05$. HR: Heart Rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; SV: stroke volume; SI: stroke index; CO: cardiac output; CI: cardiac index; SVR: systemic vascular resistance; SVRI: systemic vascular resistance index; VI: velocity index; ACI: acceleration index; PEP: pre-ejection period; LVET: left ventricular ejection time; STR: systolic time ratio; LCW: left cardiac work; LCWI: left cardiac work index; TFC: chest fluid capacity.

and after PCI. A value of $P < 0.05$ was considered statistically significant.

Results

Correlations between the parameters determined by noninvasive hemodynamic monitor and blood NT-proBNP, BNP as well as cTnT

Our results indicated NT-proBNP was negatively related to SV, SI, CO, CI and LVET (r = -0.736, -0.552, -0.378, -0.346, -0.813, $P < 0.05$), but positively associated with SVR, SVRI, PEP and STR (r = 0.806, 0.769, 0.433, 0.325, $P < 0.05$). BNP was negatively related to SV, SI, CO and LVET (r = -0.898, -0.634, -0.351, -0.895, $P < 0.05$), but positively associated with SVR, SVRI, PEP and STR (r = 0.865, 0.953, 0.583, 0.344, $P < 0.05$). cTnT was negatively related to SV, SI, CO and LVET (r = -0.741, -0.515, -0.434, -0.825, $P < 0.05$), but positively associated with SVR and SVRI (r = 0.669, 0.807, $P < 0.05$) (**Table 3**).

Relationship between impedance cardiography and echocardiography by linear correlation analysis and regression analysis

Linear correlation analysis and regression analysis showed there were obvious correlation between ICG and echocardiography in SV, SI, EDV, LEVT, STR, LVEF ($P < 0.01$), CO and CI ($P < 0.05$), but not in PEP (**Table 4**).

Changes in parameters of ICG after PCI

We also found that, blood pressure and heart rate (HR) significantly decreased 7 days after PCI compared to those before PCI, but other ICG parameters had no significant changes (**Table 5**).

Discussion

ICG, a noninvasive hemodynamic monitoring method, has been applied to monitor the hemodynamics based on the chest bioimpedance. In this study, by comparing the correlation of ICG with serum NT-proBNP, BNP, cTnT levels and echocardiography, we found that ICG would be available to monitor the change of heart function in AMI patients. The Bioz.com noninvasive hemodynamic monitoring system can detect 12 hemodynamic parameters, which can be applied in the evaluation of hemodynamics and ventricular function. These parameters include TFC, velocity index (VI), acceleration index (ACI), PEP, LVET, HR, blood pressure (BP), CO, SV, SVR, left cardiac work (LCW) and STR. The following parameters may be obtained through calculation: CI, SI, SVRI and left cardiac work index (LCWI). Among these parameters, CI, LVET, STR, VI, AVI and LCWI can be used to reflect cardiac output and myocardial contractility [8]. A study showed that the patients with low estimates of central hemodynamics evaluated by ICG are at an increased risk of the adverse in-hospital course of STEMI [9].

ICG data can used to identify the risk factors of hypertension patients and provide guidance for clinical treatment [10, 11]. In hypertension patients, the SI, CI, ACI and TFC are decreased and SVRI increased [12]. Nevertheless, in subjects with normal pressure, SVRI is relatively low while the SI, CI, ACI and TFC are dramatically increased [13]. The findings suggest coro-

Table 4. Linear correlation analysis and regression analysis of ICG and echocardiography

	x ($\bar{x}$ + SD)	y ($\bar{y}$ + SD)	r	Y = a + bX
SV (ml)	83.76 ± 21.77	74.01 ± 30.86	0.725**	Y = 12.743 + 1.027X**
SI (ml/m^2)	48.90 ± 13.30	42.96 ± 17.01	0.677**	Y = 0.587 + 0.899X**
CO (l/min)	5.88 ± 1.44	4.99 ± 1.58	0.468*	Y = 1.945 + 0.426X*
CI (l/min/m^2)	3.43 ± 0.94	2.95 ± 0.88	0.413*	Y = 1.546 + 0.445X*
EDV (ml)	120.85 ± 60.01	125.27 ± 50.35	0.514**	Y = 73.166 + 0.445*
PEP (ms)	48.05 ± 11.11	110.12 ± 35.5	0.120	Y = 0.056 + 0.635X
LVET (s)	265.12 ± 35.29	310.36 ± 45.5	0.579**	Y = 0.126 + 0.758X**
STR	0.191 ± 0.048	0.385 ± 0.12	0.550**	Y = 0.006 + 1.988X**
LVEF	0.56 ± 0.15	0.61 ± 0.14	0.786**	Y = 0.135 + 0.763X**

Note: Linear correlation analysis and regression analysis showed there was significant correlation between ICG and echocardiography in SV, SI, EDV, LEVT, STR, LVEF ($P < 0.01$), CO and CI ($P < 0.05$), but not in PEP. x: echocardiography data; y: ICG data; r: linear correlation coefficient; Y = a + bX: linear regression equation; **$P < 0.01$; *$P < 0.05$. SV: stroke volume; SI: stroke index; CO: cardiac output; CI: cardiac index; EDV: end-diastolic volume; PEP: pre-ejection period; LVET: left ventricular ejection time; STR: systolic time ratio; LVEF: left ventricular ejection fraction.

nary heart disease (CHD) patients have myocardial ischemia and compromised systolic function, which increases the left ventricular end-diastolic pressure and subsequently prolongs the PEP. PEP/LVET (STR) is a sensitive and liable indicator in the diagnosis of CHD. In CHD patients, the PEP is prolonged and LVET relatively shortened resulting in the increase of PEP/LVET. In addition, ICG data can also be applied for risk stratification for heart failure. The ICG data can be used in not only the assessment of hemodynamics but also the evaluation of systolic function of the heart.

Correlations between ICG data and blood NT-proBNP, BNP and cTnT

The detection of activities of myocardial proteins in the peripheral blood has been carried out to evaluate the AMI area for many years [14]. It has been confirmed that the cTnT is a preferred biomarker in the evaluation of AMI area [15]. Evidence also shows NT-proBNP is an indicator that can be applied to assess the infarct size and left ventricular function following AMI [16]. Steen et al investigated the role of NT-pro BNP and cardiac troponin T at 96 h for estimation of infarct size and left ventricular function after acute myocardial infarction. Patients with abnormal diastole had worse NYHA class, higher levels of BNP, and higher TFC [17]. Our results showed there was an inverse correlation between NT-pro BNP and LV-EF in STEMI (r = -0.67, $P = 0.0009$) and NSTEMI (r = -0.85, $P < 0.0001$). Likewise, cTnT showed an inverse correlation with LVEF in STEMI (r = -0.54, $P = 0.014$) but not in NSTEMI. With cTnT there was a linear correlation with infarct mass and relative infarct size in STEMI (r = 0.92, $P < 0.0001$) and NSTEMI (r = 0.59, $P < 0.0093$). NT-pro BNP demonstrated a good relationship with infarct mass (r = 0.79, $P < 0.0001$) and relative infarct size (r = 0.75, $P < 0.0001$) in STEMI, but not in NSTEMI. They proposed that NT-proBNP was mainly used to evaluate the LVEF and cTnT to assess the infarct mass and relative infarct size. In the present study, our results showed inverse correlations between NT-proBNP and ICG parameters (LVET, STR, PEP and LVEF) ($P < 0.05$). In addition, cTnT was negatively correlated with LVET and EF but not STR and PEP. Moreover, with the increase of NT-proBNP and cTnT, the SV, SI and CO decreased. Our findings demonstrate that, with the increase of infarct size in AMI patients, the NT-proBNP and cTnT increase, LVET shortens, STR elevates, PEP prolongs, LVEF decreases and the systolic function is compromised. Thus, similar to NT-proBNP and cTnT, ICG data can also be applied to indirectly evaluate the cardiac function of AMI patients.

Following myocardial infarction, a large amount of BNP is produced by the junction area between infarction region and non-infarction region the myocardium of where bares the high ventricular wall stress. Therefore, BNP can accurately reflect the local ventricular wall stress and indirectly reflect the cardiac preload [18, 19]. Therefore, detection of plasma BNP can predict both the infarction size and the left ventricular function [20]. Serum BNP level is a favorable indicator for the evaluation of heart failure, left ventricular function and myocardial remodeling after myocardial infarction [21]. In AMI patients, the BNP reaches a peak at 16 h

Table 5. ICG data before surgery and 7 days after surgery

Parameter	Before PCI	After PCI	T test
HR (bpm)	80.2 ± 4.19	73.15 ± 2.33	0.0076*
SBP (mmHg)	127.4 ± 3.56	117.2 ± 3.48	0.024*
DBP (mmHg)	75.9 ± 2.90	67 ± 2.01	0.008*
CI (l/min/m^2)	2.58 ± 0.14	2.49 ± 0.14	0.324
CO (l/min)	20.38 ± 15.56	4.53 ± 0.27	0.161
SI (ml/m^2)	47.72 ± 14.28	34.55 ± 1.96	0.186
SV (ml)	74.15 ± 13.24	62.6 ± 3.74	0.205
SVRI (dyne * s * cm^{-5} * m^2)	2820.25 ± 385.22	2656.4 ± 183.91	0.352
SVR (dyne * s * cm^{-5})	1570.80 ± 222.32	1475 ± 105.97	0.350
ACI (/100/s^2)	68.2 ± 13.70	53.75 ± 3.63	0.160
VI (/1,000/s)	46.15 ± 14.43	30.6 ± 2.05	0.149
TFC (/kOhm)	44.98 ± 14.36	32.57 ± 1.54	0.200
LCWI (Kg * m/m^2)	18.78 ± 15.64	2.64 ± 0.17	0.159
LCW (Kg * m)	21.24 ± 15.52	4.79 ± 0.38	0.151
STR (-)	16.08 ± 15.79	0.29 ± 0.02	0.165
PEP (ms)	96.75 ± 12.20	84.35 ± 4.39	0.174
LVET (ms)	301.8 ± 9.00	305.55 ± 7.33	0.374

Note: Blood pressure and heart rate significantly decreased 7 days after PCI, but other ICG parameters had no significant changes. *$P < 0.05$. HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; CI: cardiac index; CO: cardiac output; SI: stroke index; SV: stroke volume; SVRI: systemic vascular resistance index; SVR: systemic vascular resistance; ACI: acceleration index; VI: velocity index; TFC: chest fluid capacity; LCWI: left cardiac work index; LCW: left cardiac work; STR: systolic time ratio; PEP: pre-ejection period; LVET: left ventricular ejection time; PCI: percutaneous coronary intervention.

and then declines [22]. Cerisano et al [23] found that continuous increase of BNP following myocardial infarction was related to the delayed cardiovascular opening. When compared with low BNP patients, those with continuous increase of BNP had low LVEF and high left ventricular volume/mass index. BNP and left ventricular volume/mass index were two good indicators to evaluate the left ventricular expansion at early stage of myocardial infarction. Although the myocardium is not necrotic following myocardial infarction, BNP can predict the size and degree of myocardial infarction [22]. Compared to low BNP patients with myocardial infarction, continuous high BNP level is frequently observed in those with delayed cardiovascular opening, severely impaired left ventricular function and concomitant multi-vessel involvement [23]. BNP was measured in Chinese patients with or without PCI [24], and results showed the serum BNP reached a maximal level 12~24 h after emergency visit in patients receiving PCI, but the serum BNP level had two peaks in those without PCI: one at 12~24 h and the other 7 days after hospitalization. In addition, the BNP level in PCI patients was markedly lower than in non-PCI patients but there was no significant difference in the LVEF 3~5 days after hospitalization. 28 days after hospitalization, the LVEF in PCI patients was dramatically higher than in non-PCI patients. In addition, BNP was positively related to the congestive heart failure and poor prognosis following AMI [25]. Castellanos et al [26] investigated the role of BNP in combination with ICG data in the evaluation of risk events in heart failure patients. Their results showed BNP and STRI in ICG were important indicators predicting the long-term heart failure related events. Patients with BNP < 100 pg/ml and STRI > 0.45 s-1 had significantly lower incidence of adverse events when compared with those with high BNP level and low STRI (67% vs. 89%, $P = 0.001$). In a recent study, Havelka et al [27] evaluated the cardiac function of heart failure patients. Their results showed no correlations between BNP and CI or SVR, and there was 50% correlation between BNP and TFC. Our study revealed the correlations between cardiac markers (NT-proBNP, BNP and cTnT) and ICG data (SV, SI, CO, SVR, SVRI, LVET and STR) ($P < 0.05$). Moreover, with the increase of NT-proBNP, BNP and cTnT, the compromised cardiac function further deteriorates which is characterized by decrease of SV, SI, CO and LVET and increase of SVR, SVRI and STR.

TFC is an indicator reflecting the thoracic fluid content and can used to indirectly evaluate the degree of heart failure. These may be the main causes of no correlations between TFC and NT-proBNP, BNP or cTnT. In patients with chronic heart failure, the application of vasodilators depends on systolic blood pressure (SBP). However, SBP can no accurately reflect the real vasodilatation [28]. Excessive volume load induced chronic heart failure can be treated

with diuretics; angiotensin-converting enzyme inhibitors (ACEI) and/or angiotensin receptor blocker (ARB) and these drugs can effectively improve the afterload and subsequently the cardiac function. In the present study, except for patients with sever renal dysfunction or low blood pressure; the remaining patients were treated with ACEI or ARB. Results showed the SVR and SVRI were markedly increased and higher than the normal limit. Thus, the SCRI can be applied as an indicator to guide the clinical application of vasodilators, which is beneficial for improvement of left ventricular remodeling and of great clinical value.

ICG and echocardiography

Nowadays, transthoracic echocardiography has been a preferred strategy in the evaluation of cardiac function. However, the high cost and complicate manipulation significantly limit its wide application in China. LVEF is a main indicator for evaluation of left ventricular function in heart failure patients and often obtained by echocardiography or left ventricular angiography. However, in clinical practice, both techniques are costly and require trained personnel, which also limit their application. ICG is a non-invasive technique that can monitor the hemodynamics in a real time manner and this technique is relative affordable and simple. A study comparing the ICG and echocardiography showed the correlation between both methods in the CO, SV and EDV. The CI and STR from ICG are closely related to the LVEF from echocardiography ($r = 0.85$) [29]. Brenda et al [30] proposed STR from ICG was an alternative to LVEF in the evaluation of cardiac function in heart failure patients and the correlation coefficient between STR and LVEF was -0.55 ($P < 0.001$). The sensitivity, specificity, positive rate and negative rate of STR ≥ 0.50 were 92%, 85%, 95% and 79%, respectively in the evaluation of cardiac function. Thus, STR ≥ 0.50 was regarded as a cutoff value in the assessment of left ventricular dysfunction. In the evaluation of cardiac function of chronic heart failure, Thompson et al also found the STR in the ICG was negatively related to the EF in the echocardiography ($r = -0.54$; $P < 0.001$) [31]. In the present study, both ICG and echocardiography were employed to detect the SV, SI, CO, CI, EDV, LEVT, STR, LVEF and PEP. Our results revealed there were correlations between two methods in SV, SI, EDV, LEVT, STR, LVEF ($P < 0.01$), CO and CI ($P < 0.05$). This finding suggests the ICG data were related to those in echocardiography and ICG can be used as a portable technique to timely evaluate the hemodynamics of AMI patients.

Conclusion

The present study investigated the role of ICG in the evaluation of hemodynamics and cardiac function in AMI patients. Our results show that there had correlation in AMI patients on hospitalization although they do no present symptoms of heart failure. In addition, the parameters reflecting the hemodynamics are closely related to the cardiac markers (BNP, NT-proBNP and cTnT) and correlation is also noted between echocardiography and ICG in the evaluation of hemodynamics of AMI patients. Therefore, in the absence of echocardiography or blood detection, ICG can be applied as an effective and real time method to evaluate the cardiac function. However, more multi-center and double blind clinical studies with large sample size are required to quantify the cardiac function according to the ICG data.

Disclosure of conflict of interest

None.

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