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Association of remote ischemic peri-conditioning with reduced incidence of clinical heart failure after primary percutaneous coronary intervention

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Abstract

Background—Clinical heart failure (HF) occurs frequently after ST-segment elevation myocardial infarction (STEMI), and is associated with increased mortality. We assessed the impact of remote ischemic peri-conditioning (RIPC) during inter-facility air medical transport of STEMI patients on clinical HF following primary percutaneous coronary intervention (pPCI).

Methods—Data from Acute Coronary Treatment and Intervention Outcomes Network Registry®-Get With the Guidelines™ (ACTION Registry-GWTG) from two PCI-hospitals that are utilizing RIPC during inter-facility helicopter transport of STEMI patients for pPCI between March, 2013 and September, 2015 were used for this study. The analyses were limited to inter-facility STEMI patients transported by helicopter with LVEF <55% after pPCI. The outcome measures were occurrence of clinical HF and serum level of brain-type natriuretic peptide (BNP).

Results—Out of the 150 STEMI patients in this analysis, 92 patients received RIPC and 58 did not. The RIPC and non-RIPC groups were generally similar in demographic and clinical characteristics except for lower incidence of cardiac arrest in the RIPC group (3/92 [3.3%] versus 13/58 [22.4%], p=0.002). STEMI patients who received RIPC were less likely to have in-hospital clinical HF compared to patients who did not receive RIPC (3/92 [3.3%] versus 7/58 [12.1%]; adjusted OR=0.22, 95% CI 0.05–0.92, p=0.038) after adjusting for baseline differences. In

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subgroup analysis, RIPC was associated with lower BNP (123 [interquartile range, 17.0–310] versus 319 [interquartile range, 106–552], p= 0.029).

Conclusion—RIPC applied during inter-facility air transport of STEMI patients for pPCI is associated with reduced incidence of clinical HF and serum BNP.

Keywords

remote ischemic conditioning; acute myocardial infarction; heart failure; brain-type natriuretic peptide

INTRODUCTION

Clinical heart failure is a common complication of ST-segment elevation myocardial infarction (STEMI), leading to prolonged hospital stay, consumption of healthcare resources, and increased morbidity and mortality [1–3]. While clinical heart failure (HF) and reduced left ventricular ejection fraction (LVEF) are not synonymous, most patients that develop clinical HF after STEMI have reduced LVEF, and some patients with reduced LVEF have no clinical signs or symptoms of HF. Although improvements in STEMI treatment and prompt primary percutaneous coronary intervention (pPCI) in the last decade have reduced the incidence and/or severity of cardiac dysfunction after STEMI, some studies have indicated that a significant proportion of patients still develop clinical HF [1,2], with a reported incidence rate of about 10–40% depending on the population studied [1,2]. There is an increasing concern that the reduction in mortality of STEMI patients might lead to increased numbers of patients surviving only to suffer severe HF [4], with significant global economic burden [3]. Therefore, there is a need to develop adjunctive therapy to pPCI that can prevent or reduce clinical HF in STEMI patients.

In this regard, emerging evidence suggests that remote ischemic conditioning (RIC) elicited by non-injurious, brief episodes of ischemia and reperfusion at a distant vascular bed may protect vital organs such as the heart from subsequent injury [5,6]. This non-invasive strategy appears to confer protection when applied prior to prolonged ischemic injury (pre-conditioning), during ischemic injury (peri-conditioning) and at the end of ischemic injury or onset of reperfusion (post-conditioning). In a recent pre-clinical study in rats, RIC reduced adverse left ventricular remodeling and oxidative stress induced by myocardial infarction [7]. However, the effect of RIC on clinical HF in humans remains unknown. Accordingly, this cross-sectional study examined for the first time the impact of remote ischemic peri-conditioning (RIPC) on in-hospital clinical HF and biomarker of cardiac dysfunction, brain-type natriuretic peptide (BNP), in a contemporary population of STEMI patients with reduced LVEF after pPCI.

MATERIALS AND METHODS

Protocol Implementation and Data Sources

Data from the National Cardiovascular Data Registry® (NCDR) Acute Coronary Treatment and Intervention Outcomes Network Registry®-Get With the Guidelines™ (ACTION Registry–GWTG) from two tertiary care STEMI receiving centers (University of Pittsburgh

Medical Center Presbyterian and Passavant Hospital) in the United States that are utilizing RIPC during inter-facility helicopter transport of STEMI patients for pPCI were used for this study. The ACTION Registry–GWTG is a nationwide, voluntary, quality improvement registry sponsored by the American College of Cardiology and the American Heart Association [8]. The conduct of the RIPC protocol during helicopter transport of STEMI patients transferred to our two receiving facilities has been previously published [9]. In summary, RIPC was performed during inter-facility helicopter transport by repeated 5-minute cycles of inflation to 200 mmHg and deflation of a blood pressure cuff in the upper arm. A maximum of four cycles of RIPC were performed en route, and standard medical care for these patients and timeliness of transport were prioritized over RIPC. The protocol was approved by the University of Pittsburgh Institutional Review Board, the Pennsylvania Department of Health, the state EMS advisory council, and the regional Emergency Medical Services (EMS) council.

Patient Population

Patients with STEMI (n=536) that presented to the two PCI centers for pPCI from March 2013 through September 2015 were identified. Analyses were limited to STEMI patients with inter-facility air transport within the period that the RIPC quality improvement protocol was implemented. The protocol was first implemented at two air medical base sites as a pilot and then extended to 12 additional base sites, resulting in a cohort of patients that did or did not receive RIPC. STEMI was defined as chest pain or epigastric pain for more than 30 minutes and either (a) new ST elevation at the J point in two contiguous leads with the cut-off points of ≥0.2 mV in men or ≥0.15 mV in women in leads V2-V3 or ≥0.1 mV in all other leads or (b) new or presumed new left bundle branch block on electrocardiogram. Patients were excluded if they arrived directly to the Emergency Department of the two PCI centers (n=88), were transferred from the field directly to the two PCI centers (n=177), were transported by ground ambulance (n=49), and those with persistent hypotension at the time of air transport (n=3) were excluded. These patients were excluded because during the period of this study RIPC was not routinely performed in this EMS system for those transferred by ground ambulance and those with hypotension at the time of transfer. Additionally, those transferred from the field were excluded because the current analysis is limited to inter-facility transfers where multiple cycles of RIPC can be performed during transfer of the patients without delaying pPCI at the receiving facility. Given that patients with reduced LVEF are more likely to develop clinical HF symptoms, those with LVEF ≥55% (n=69) were excluded from 219 inter-facility STEMI patients. The final population (n=150) included in this analysis were inter-facility STEMI patients that were transported by air ambulance, received coronary stenting or plain old balloon angioplasty, and with LVEF <55% after pPCI.

Study Outcomes

The main study outcome measure was the occurrence of clinical HF, defined as physician report of one or more of the following: i) unusual dyspnea on exertion or dyspnea occurring in the supine position; ii) rales or jugular venous distention on physical examination; and iii) pulmonary edema on chest x-ray presumed to be due to cardiac dysfunction. The other outcome measure was the first BNP level during hospitalization. These definitions were

based on predefined criteria outlined in the ACTION Registry®–GWTG™ database. For outcomes analyses, we report i) the percentage of patients who experienced in-hospital clinical HF, and ii) the median BNP value.

Data Analysis

Descriptive characteristics of the study population are presented as n (%) for categorical variables and median (interquartile range) or mean (SD) for continuous variables. Baseline differences between patients that received RIPC and those that did not were tested using chi-squared tests for categorical variables (Fisher's Exact test for very small cells) and Wilcoxon rank-sum tests for continuous variables.

Although this analysis was based on registry data with modest sample size, there were only trivial differences in baseline characteristics between the patients that received RIPC and those that did not receive RIPC. Therefore, the conventional approach to assessing association in non-randomized studies (performing multivariable regression to "adjust" for characteristics that differ between the treatment groups) does not require that many covariates be accounted for with multivariable modeling. Nevertheless, to assess the robustness of our results when accounting for potential confounders, we present the effect of RIPC on clinical HF with a multivariable logistic regression model adjusted for variable with significant baseline difference: cardiac arrest on first medical contact. We also performed subgroup analysis excluding patients with cardiac arrest on first medical contact.

For outcomes analyses, we report i) the percentage of patients who experienced in-hospital clinical HF, and ii) the median BNP value. All statistical analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC). Two-sided p values <0.05 were considered as indicative of statistical significance.

RESULTS

Out of the 150 STEMI patients (median age, 62 years, interquartile range: 54–70 years; 74% male) included in this analysis, 92 patients received RIPC and 58 did not. Of the 92 patients that received RIPC, 70 patients (76.1%) received 3 or 4 cycles of RIPC while 22 patients (23.9%) received less than 3 cycles. The RIPC and non-RIPC groups were generally similar in demographic, clinical, angiographic and procedural characteristics except for significantly lower incidence of cardiac arrest in the RIPC group (3/92 [3.3%] versus 13/58 [22.4%], p=0.002) (Tables 1 and 2).

As shown in figure 1a. Despite having similar incidence of prior HF (3/92 [3.3%] versus 2/58 [3.4%], p=0.950), left main coronary artery disease (2/92 [2.2%] versus 2/58 [3.4%], p=0.637), anterior wall myocardial infarction (40/92 [43.5%] versus 24/58 [41.4%], p=0.467), left ventricular ejection fraction (45 [interquartile range, 38.0–50.0] versus 49 [interquartile range, 40–53], p=0.290), and door-to-balloon time (112 [interquartile range, 88–130] versus 112 [interquartile range, 88–130], p=0.552), STEMI patients who received RIPC were less likely to have in-hospital clinical HF compared to patients who did not receive RIPC (3/92 [3.3%] versus 7/58 [12.1%]; OR=0.25, 95% CI 0.06–0.99, p=0.035). In multivariate logistic regression analysis adjusting for baseline difference in incidence of

cardiac arrest, RIPC remained significantly associated with lower incidence of clinical HF (OR=0.22, 95% CI 0.05–0.92, p=0.038).

Further subgroup analyses restricted to patients without cardiac arrest (n=134) showed similar results (3/89 [3.4%] versus 6/45 [13.3%]; OR=0.28, 95% CI 0.05–0.95, p=0.030) (Figure 1b). Similarly, in a subgroup of patients that had serum BNP measured during hospitalization (n=41), RIPC, compared to non-RIPC group, was associated with lower serum BNP level (123 [interquartile range, 17.0–310] versus 319 [interquartile range, 106–552], p= 0.029).

DISCUSSION

This cross-sectional study of contemporary STEMI patients at two PCI centers in the United States suggests an association of RIPC applied during inter-facility air transport of STEMI patients with reduced incidence of in-hospital clinical HF, and lower serum BNP among patients with reduced LVEF after PCI. To the best of our knowledge, this is the first report on the impact of RIPC applied during interfacility air transport of STEMI patients on clinical HF after pPCI.

The occurrence of clinical HF in STEMI patients results in exercise intolerance, impairs quality of life, and has been associated with high morbidity and mortality [1,2]. Although improvements in STEMI treatment by prompt revascularization have reduced the incidence and severity of clinical HF after STEMI, a significant proportion of patients still develop clinical HF [1,2]. Therefore, there is a need to develop adjunctive therapy to prevent or reduce clinical HF in STEMI patients. The present observation of reduced clinical HF and serum BNP in STEMI patients that received RIPC calls for additional study on the possible cardio-protective effects of RIPC on ischemia HF in STEMI patients.

Although, the mechanisms of these associations are not completely understood, studies have shown that that several signal transduction pathways are activated by RIPC, including generation of endogenous protective biomarkers in many organs, and specifically in the heart, nitric oxide and nitrite [10, 11]. A recent translational study in rat and humans found nitrite, the single electron oxidation product of nitric oxide, to be increased by about 50% with a single cycle of RIPC when normal reperfusion accompanied by reactive hyperemia was performed. Further experiments suggested that nitrite was both necessary and sufficient to confer cardio-protection when human plasma after RIPC was given to rat hearts in an ex vivo cardiac ischemia reperfusion model [11]. This endogenous, RIPC-induced nitrite may reduce clinical heart failure symptoms and serum BNP via vasodilatory activity in large veins and arteries resulting in reduced pre-load, after-load and ventricular wall stress. The present observation extends upon these findings and provides new insight by demonstrating that RIPC may protect against clinical HF in humans with STEMI undergoing pPCI.

The present study has some limitations. First, this is a cross-sectional analysis of a registry data and thus could not account for unmeasured cofounders including effect of medications such as ticagrelor on development of dyspnea. Second, the present study is limited to patients with reduced LVEF (<55%) after pPCI who are known to have higher incidence of

clinical HF. Third, our sample size is modest and outcome events are few thus limiting adjustment for multiple covariates. However, there were only trivial differences in baseline characteristics between the patients that received RIPC and those that did not receive RIPC. Therefore, the conventional approach to assessing association in non-randomized studies (performing propensity score matching or multivariable regression to "adjust" for characteristics that differ between the treatment groups) does not require that many covariates be accounted for with multivariable modeling. Nevertheless, we observed similar findings with multivariable adjustment for cardiac arrest and subgroup analysis excluding patients with cardiac arrest. Given the significance of clinical HF and the associated morbidity and mortality in STEMI patients, the observed reduced incidence of clinical HF in the RIPC group is an important hypothesis generating finding that warrants further in-depth investigation. To the best of our knowledge, this is the first report on impact of RIPC applied during inter-facility transfer on clinical HF in STEMI patients.

Conclusions

RIPC applied during inter-facility air transport is associated with lower incidence of in-hospital clinical HF and serum BNP level in contemporary population of STEMI patients with reduced LVEF after pPCI. Large randomized clinical trials are needed to determine the effects of RIPC on clinical HF in patients with coronary artery disease, and to discern the mechanisms of such effects.

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HIGHLIGHTS

- Clinical heart failure (HF) occurs frequently after ST-segment elevation myocardial infarction (STEMI), and is associated with increased mortality
- This study shows that remote ischemic peri-conditioning (RIPC) applied during inter-facility helicopter transfer of STEMI patients is associated with reduced incidence of clinical HF after primary percutaneous coronary intervention (pPCI)
- Large, multicenter randomized controlled studies are needed to determine the effects of RIPC on clinical HF in STEMI patients

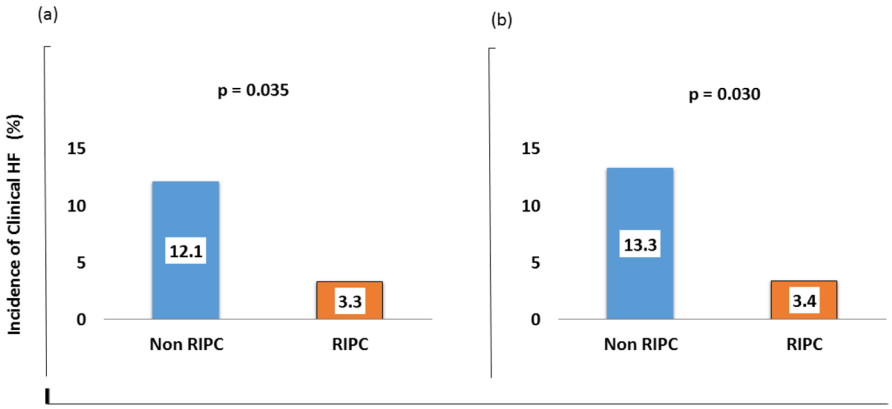

Figure 1.

Incidence of in-hospital clinical heart failure (HF) with reduced ejection fraction after primary percutaneous coronary intervention in the overall study population (a) and in those without cardiac arrest (b) stratified by whether they received or did not receive remote ischemic peri-conditioning (RIPC).

Table 1

Baseline Clinical Characteristics and Risk Factors

	Overall	Non RIPC	RIPC	P-Value
# Patients	**150**	**58**	**92**	
Demographics				
Age (Years)	62.0 (54.0–70.0)	61.5 (54.0–70.0)	62.0 (53.5–71.0)	0.922
Gender (% Male)	111 (74.0%)	46 (79.3%)	65 (70.7%)	0.239
Height (cm)	173 (163–180)	173 (170–178)	173 (163–180)	0.251
Weight (kg)	84.5 (75.0–100)	83.5 (76.0–100)	86.0 (73.5–100)	0.769
Body Mass Index	29.1 (25.5–34.0)	27.5 (24.3–34.0)	29.5 (26.4–34.2)	0.071
Race				0.724
White	145 (96.7%)	57 (98.3%)	88 (95.7%)	
Black	3 (2.0%)	1 (1.7%)	2 (2.2%)	
Asian	1 (0.7%)	0 (0.0%)	1 (1.1%)	
Other	1 (0.7%)	0 (0.0%)	1 (1.1%)	
Insurance				0.658
Private	77 (51.3%)	29 (50.0%)	48 (52.2%)	
Public	59 (39.3%)	22 (37.9%)	37 (40.2%)	
None	14 (9.3%)	7 (12.1%)	7 (7.6%)	
Medical History				
Hypertension	85 (56.7%)	36 (62.1%)	49 (53.3%)	0.289
Dyslipidemia	74 (49.3%)	30 (51.7%)	44 (47.8%)	0.642
Current Dialysis	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Chronic Lung Disease	7 (4.7%)	3 (5.2%)	4 (4.3%)	0.845
Diabetes	30 (20.0%)	7 (12.1%)	23 (25.0%)	0.054
Prior Myocardial Infarction	22 (14.7%)	11 (19.0%)	11 (12.0%)	0.237
Prior Heart Failure	5 (3.3%)	2 (3.4%)	3 (3.3%)	0.950
Prior Percutaneous Coronary Intervention	25 (16.7%)	13 (22.4%)	12 (13.0%)	0.134
Prior Coronary Artery Bypass Graft	6 (4.0%)	2 (3.4%)	4 (4.3%)	0.784
Prior Atrial fibrillation	5 (3.3%)	1 (1.7%)	4 (4.3%)	0.383
Prior Stroke	5 (3.3%)	2 (3.4%)	3 (3.3%)	0.206
Prior Peripheral Artery Disease	3 (2.0%)	1 (1.7%)	2 (2.2%)	0.848
Home Medications				
Aspirin	45 (30.0%)	21 (36.2%)	24 (26.1%)	0.188
Clopidogrel	8 (5.3%)	3 (5.2%)	5 (5.4%)	0.945
Prasugrel	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Ticargrelor	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Warfarin	3 (2.0%)	1 (1.7%)	2 (2.2%)	0.848
Beta blocker	31 (20.7%)	14 (24.1%)	17 (18.5%)	0.405
Angiotensin Converting Enzyme Inhibitor	24 (16.0%)	8 (13.8%)	16 (17.4%)	0.558

	Overall	Non RIPC	RIPC	P-Value
# Patients	**150**	**58**	**92**	
Angiotensin Receptor Blocker	9 (6.0%)	2 (3.4%)	7 (7.6%)	0.289
Aldosterone Blocking Agent	3 (2.0%)	2 (3.4%)	1 (1.1%)	0.314
Statin	31 (20.7%)	13 (22.4%)	18 (19.6%)	0.675

Continuous variables presented as median (IQR) and categorical variables presented as n (%).

Table 2

Clinical Presentation, Reperfusion Time, Biomarkers and Angiographic Characteristics

	Overall	Non RIPC	RIPC	P-Value
# Patients	**150**	**58**	**92**	
Signs at Presentation				
Heart Rate (beats per minute)	80.0 (69.0–94.5)	86.5 (69.0–98.5)	78.0 (69.5–90.5)	0.065
Systolic Blood Pressure (mmHg)	145 (124–161)	137 (116–168)	146 (127–159)	0.228
Shock on First Medical Contact	5 (3.3%)	3 (5.2%)	2 (2.2%)	0.319
Cardiac Arrest on First Medical Contact	16 (10.7%)	13 (22.4%)	3 (3.3%)	0.0002
Diseased Vessels and Cardiac Function				
Vessels with Significant Disease				
Left Main	4 (2.7%)	2 (3.4%)	2 (2.2%)	0.637
Proximal Left Anterior Descending	34 (22.7%)	14 (24.1%)	20 (21.7%)	0.733
Mid/Distal Left Anterior Descending	49 (32.7%)	21 (36.2%)	28 (30.4%)	0.463
Left Circumflex	41 (27.3%)	15 (25.9%)	26 (28.3%)	0.748
Right coronary	70 (46.7%)	32 (55.2%)	38 (41.3%)	0.097
Ramus Intermedius	6 (4.0%)	3 (5.2%)	3 (3.3%)	0.561
Number of Diseased Vessels				0.424
1	102 (68.0%)	39 (67.2%)	63 (68.5%)	
2	30 (20.0%)	14 (24.1%)	16 (17.4%)	
3	18 (12.0%)	5 (8.6%)	13 (14.1%)	
Left ventricular Ejection Fraction	45.0 (38.0–53.0)	49.0 (40.0–53.0)	45.0 (38.0–50.0)	0.290
Culprit Vessels				0.467
Left Anterior Descending	64 (42.7%)	24 (41.4%)	40 (43.5%)	
Non Left Anterior Descending	86 (57.3%)	34 (58.6%)	52 (56.5)	
Coronary Intervention/Stent Type				0.250
Plain Old Balloon Angioplasty	15 (10.0%)	5 (8.6%)	10 (10.9%)	
Bare Metal Stent	38 (25.3%)	19 (32.8%)	19 (20.7%)	
Drug Eluting Stent	97 (64.7%)	34 (58.6%)	63 (68.5%)	
Cardiac and Renal Biomarkers				
Initial Troponin (ng/mL)	0.9 (0.1–8.3)	0.7 (0.1–9.0)	0.9 (0.1–7.9)	0.727
Peak Troponin (ng/mL)	54.0 (18.6–112)	46.2 (18.3–130)	56.7 (19.8–111)	0.749
Initial Creatinine (mg/dL)	1.0 (0.8–1.2)	1.0 (0.8–1.2)	1.0 (0.8–1.1)	0.602
Initial BUN (mg/dL)	16.0 (13.0–20.0)	16.0 (13.0–18.0)	17.0 (13.0–22.0)	0.192
Initial eGFR by MDRD (ml/min/1.73m^2)	77.8 (64.4–93.9)	76.9 (62.0–95.8)	78.0 (65.1–93.8)	0.649
Total Contrast Used (mL)	175 (150–200)	175 (155–200)	175 (138–220)	0.879
Reperfusion Time Intervals				
Door-In-Door-Out (minutes)	54.0 (41.0–75.0)	59.0 (43.0–97.0)	52.0 (41.0–71.0)	0.170
Transport Time (minutes)	29.0 (19.0–35.0)	25.0 (16.0–33.0)	30.0 (22.0–35.0)	0.159
Procedure Time (minutes)	27.0 (22.6–40.0)	26.0 (23.0–44.0)	27.5 (22.0–37.9)	0.543

	Overall	Non RIPC	RIPC	P-Value
# Patients	**150**	**58**	**92**	
Door-To-Balloon Time (minutes)	112 (88.0–138)	112 (88.0–148)	112 (88.0–130)	0.552
Door-To-Balloon Time Goal Category				0.844
<120 minutes	92 (61.3%)	35 (60.3%)	57 (62.0%)	
>120 minutes	58 (38.7%)	23 (39.7%)	35 (38.0%)	

Continuous variables presented as median (IQR) and categorical variables presented as n (%).

eGFR = estimated glomerular filtration rate; MDRD = modification of diet in renal disease.