Published in final edited form as:

Am J Emerg Med. 2016 April ; 34(4): 722–725. doi:10.1016/j.ajem.2015.12.077.

The Impact of Body Mass Index On Patient Survival After Therapeutic Hypothermia Following Resuscitation

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Abstract

OBJECTIVES—Therapeutic hypothermia improves survival in patients following cardiac arrest; yet the impact of body mass index(BMI) on survival is lesser known. We hypothesized that non-obese patients would have greater survival post therapeutic hypothermia than obese patients.

METHODS—We retrospectively evaluated 164 patients who underwent therapeutic hypothermia following resuscitation for cardiac arrest from 1/2012-9/2014. Logistic regression analysis was used to assess for survival based upon BMI and comorbidities(odds ratio[OR], 95% confidence interval[CI]).

RESULTS—Forty-one percent of patients were obese. Obese patients presented less frequently with ventricular fibrillation($p=0.046$) but had similar rates of pulseless electrical activity($p=0.479$), and ventricular tachycardia($p=0.262$) as non-obese patients. In multivariable analysis, BMI $<30\text{kg/m}^2$, hypertension, presence of pacemaker/implantable cardioverter defibrillator(PM/ICD), high glomerular filtration rate(GFR), and low neuron-specific enolase(NSE) were all associated with increased survival post therapeutic hypothermia respectively: 0.36(0.16-0.78), 0.28(0.12-0.66), 0.23(0.08-0.62), 0.25(0.11-0.56), 0.37(0.14-0.96). Other comorbidities demonstrated no association with survival.

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Conflict of Interest

We otherwise have no disclosures.

CONCLUSIONS—BMI $\geq 30\text{kg/m}^2$ compared to BMI $<30\text{kg/m}^2$ was a significant risk factor for mortality post therapeutic hypothermia protocol. Absence of history of hypertension, lack of PM/ICD, high NSE, and renal disease had greater associations with death. Larger studies will be needed to validate these findings.

1. Introduction

Therapeutic hypothermia improves survival post cardiac arrest.[1,2] Known predictors of survival post therapeutic hypothermia include early bystander cardiopulmonary resuscitation and ventricular fibrillation arrest.[3–7] Despite increased utilization of therapeutic hypothermia, overall survival post cardiac arrest remains less than 50%.[2] Obesity is an emerging risk factor for sudden cardiac death.[8,9] As the prevalence of obesity rises in the United States, there will be an increase in the prevalence of sudden cardiac death and likely utilization of therapeutic hypothermia.[9,10] While there is growing medical and surgical literature showing that the time to target temperature takes longer for obese patient, little is known about the impact of obesity on survival post therapeutic hypothermia.[11–13] We hypothesized that body mass index (BMI) ≥ 30 kilogram/meter squared (kg/m^2) is associated with higher risk of mortality than BMI $<30\text{kg/m}^2$ after therapeutic hypothermia for cardiac arrest.

2. Methods

2.1 Study Design and Setting

A retrospective evaluation was conducted of 164 consecutive patients who underwent therapeutic hypothermia following resuscitation for cardiac arrest at the Ohio State University Wexner Medical Center from 1/2012 – 9/2014. The therapeutic hypothermia protocol is standardized to initiate therapy within 6 hours of arrest, maintain cooling temperature $89.6\text{--}93.2^\circ$ Fahrenheit using intravenous cooled fluid and external cooling blankets or intravascular cooling catheters for 24 hours, followed by controlled rewarming to body temperature over no sooner than 8 hours. Patients were stratified by the presence of clinical obesity, BMI $< 30\text{ kg/m}^2$ compared $\geq 30\text{ kg/m}^2$, which was calculated from admission weight and height in the electronic medical record. The following covariates were assessed for upon hospital discharge using the institutional Information Warehouse which is populated from the medical record: arrest etiology (cardiac), arrhythmia leading to arrest (other dysrhythmia, pulseless electrical activity, ventricular fibrillation, ventricular tachycardia), arrhythmia of past medical history (atrial fibrillation, atrial flutter), comorbidities of past medical history unless noted otherwise [chronic kidney disease (CKD)- National Kidney Foundation Stage III or higher, diabetes mellitus, hypertension, obstructive coronary artery disease- known or discovered during left heart catheterization, pacemaker/implantable cardioverter defibrillator (PM/ICD)], demographics (age, gender, race), left ventricular ejection fraction (pre-arrest and during hospitalization by either cardiac magnetic resonance imaging, contrast left ventriculography, echocardiogram, or nuclear imaging), maximum and minimum temperature during therapeutic hypothermia, and labs [brain natriuretic peptide (BNP), glomerular filtration rate (GFR) upon admission and

discharge, maximum neuron-specific enolase (NSE), and troponin]. Our institutional review board approved this study.

2.2 Outcome Measures

The primary outcome was survival to hospital discharge after therapeutic hypothermia post cardiac arrest.

2.3 Statistical Analysis

Baseline patient characteristics were compared between stratified BMI groups ($< 30 \text{ kg/m}^2$ and $\geq 30 \text{ kg/m}^2$) using chi-square tests for categorical variables and analysis of variance for continuous variables. Normality testing was performed. Logarithmic transformation was applied when it was needed. Logistic regression analysis was used to estimate survival post therapeutic hypothermia based upon covariates (odds ratio [OR], 95% confidence interval [CI]). Kaplan-Meier analyses were performed to compare survival times. Statistical analyses were performed using SAS 9.4 (Cary, NC), and the significance level was set at 0.05 for all tests.

3. Results

Forty-one percent of patients who underwent therapeutic hypothermia were obese ($n=68$). Obesity was more common in patients with female sex ($p = 0.006$), presence of CKD ($p = 0.024$), diabetic status ($p < 0.0001$), hypertension ($p = 0.008$), worse discharge GFR (discharge $p = 0.046$), and smaller troponin ($p = 0.040$). Obese patients had a higher prevalence of the following arrhythmias past medical history of atrial fibrillation/flutter (atrial fibrillation $p = 0.013$, atrial flutter $p = 0.017$) and during arrest were less likely to have ventricular fibrillation ($p = 0.046$). Rates of pulseless electrical activity ($p = 0.479$), ventricular tachycardia ($p = 0.262$), and other unlisted cardiac arrhythmia (0.714) were similar between patients with BMI $< 30 \text{ kg/m}^2$ and $\geq 30 \text{ kg/m}^2$. Other demographics, comorbidities, lab/study findings were similar between both groups. (Table 1)

In univariable logistic regression analysis, BMI $< 30 \text{ kg/m}^2$, hypertension, presence of PM/ICD, high GFR, and low NSE were all associated with increased survival post therapeutic hypothermia [BMI $< 30 \text{ kg/m}^2$ versus ($\geq 30 \text{ kg/m}^2$) 0.50 (0.26 – 0.97), hypertension 0.48 (0.24 – 0.96), PM/ICD vs. no PM/ICD 0.28 (0.12 – 0.65), GFR discharge median vs. $<$ median 0.29 (0.14 – 0.58), NSE $<$ median vs. median 0.32 (0.13 – 0.77)]. In multivariable logistic regression analysis, BMI $< 30 \text{ kg/m}^2$, hypertension, presence of PM/ICD, high GFR, and low NSE were all associated with increased survival post therapeutic hypothermia [BMI $< 30 \text{ kg/m}^2$ vs. $\geq 30 \text{ kg/m}^2$ 0.36 (0.16 – 0.78), hypertension vs. no hypertension 0.28 (0.12 – 0.66), PM/ICD vs. no PM/ICD 0.23 (0.08 – 0.62), GFR discharge median vs. $<$ median 0.25 (0.11 – 0.56), NSE $<$ median vs. median 0.37 (0.14 – 0.96)]. Race, gender and other covariates had no association with survival. (Table 2, Figure 1)

4. Discussion

This study analyzed predictors for mortality in a population of patients with cardiac arrest managed with therapeutic hypothermia. In multivariable analysis, our main findings were

that obesity was associated with increased mortality post therapeutic hypothermia compared to non-obese patients, and the presence of a PM/ICD, hypertension, low NSE, and high GFR were protective. To our knowledge, this is the first study demonstrating worsened outcomes with obesity in this population.

There is a strong association between obesity and sudden cardiac death, which is of paramount importance given the obesity epidemic in the United States.[14–16] The relationship between obesity and cardiovascular mortality and morbidity is complex, predominantly confounded by worsening cardiovascular risk factors in obese patients. [10,17,18] Despite the obesity paradox in cardiovascular disease, different aspects of obesity remain associated with worse outcomes.[19] Beyond BMI, the distribution of obesity is important; central obesity is associated with high risk of cardiac events.[19] The Paris Prospective Study found an increased risk of sudden cardiac death with rising sagittal abdominal diameter.[15] The Atherosclerosis Risk in Communities Study reported a two-fold increased risk of sudden cardiac death in patients with highest waist-hip ratio and concluded that BMI>35 was a significant predictor of SCD risk.[8] Visceral obesity also usually results in increased epicardial fat which has been strongly associated with arrhythmias.[20] Visceral adipose tissue has a constant flux of free fatty acids, which has been linked with risk of sudden cardiac death.[20] Unfortunately, obesity distribution data was unavailable for analysis in our cohort. However, these results revealed that obesity is associated with increased mortality post therapeutic hypothermia. Whether this reflects a primarily increased mortality driven by obesity within a sicker population with known higher rates of sudden cardiac death or an independent variable post therapeutic hypothermia requires additional investigation. While there are limited studies assessing survival based on BMI post therapeutic hypothermia, there are some studies that have tested the differences in time to target temperature based upon BMI in peri-operative and post-cardiac arrest settings. In these studies, obesity has been associated with longer duration of time to reach target temperature; although in one study there was no difference in survival. [11,12,21,22] It is acknowledged that fewer obese patients presented with ventricular fibrillation than non-obese patients, which may affect the findings. Nevertheless, we show contemporary results of patients stratified by BMI with similar percentages of non-shockable rhythms, ventricular tachycardia, and similar achievement of target temperatures; obesity was associated with reduced survival. This necessitates looking at the impact of BMI on survival post therapeutic hypothermia on a larger scale.

The presence of PM/ICD, low NSE, and high GFR are known predictors of survival post therapeutic hypothermia, but a history of hypertension is associated with an increased risk of sudden cardiac death.[4,13,23,24] However, induction of hypertension to increase cerebral perfusion has correlated with improved survival in post arrest cardiac models.[25] It is theorized that the latter factor could have led to the findings in this study. The association of hypertension with survival post therapeutic hypothermia requires additional study.

There are limitations in this sample size by including a single center. Yet, this center is a major tertiary referral center for the state of Ohio, serving a large heterogeneous population not dissimilar to other major hospitals. We were unable to determine the impact of time to target temperature or total arrest time before therapeutic hypothermia given limitations in

medical records. However differences in minimum temperature achieved based upon BMI were assessed, which provides incremental value. Additionally, given the retrospective nature of this study, systematic long-term follow-up was not available.

5. Conclusion

BMI < 30 kg/m² compared to ≥ 30 kg/m² was associated with survival post therapeutic hypothermia for cardiac arrest. This may be related to extended time to target temperature or an inherent risk of obesity. PM/ICD, low NSE, high GFR, and hypertension were also associated with higher survival. Further investigation on a national scale is necessary to assess the impact of these comorbidities on survival and further lead to tailoring of hospital-wide therapeutic hypothermia protocols.

Acknowledgments

The authors thank the Department of Internal Medicine and Division of Cardiovascular Medicine at The Ohio State University Wexner Medical Center for their support in proceeding with this study. We thank Ryan Ziegler for his efforts in assisting with data collection through our Information Warehouse.

Funding: NIH T32 Cardiovascular/Obesity Outcomes Fellowship 5T32HL116276-02 (KB)

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Figure. Time to Death Post Therapeutic Hypothermia Based Upon BMI

This is the multivariable model for time to death post therapeutic hypothermia for cardiac arrest based upon BMI < 30 kg/m² or BMI ≥ 30 kg/m². BMI indicates body mass index in kilogram/meter squared.

Table 1

Baseline Characteristics Stratified by BMI.

	BMI <30 kg/m ² (N=96) N (%)	BMI ≥ 30 kg/m ² (N=68) N (%)	p-value
Female Gender	23 (24)	30 (44)	0.006
Age, Mean ± SD	54.8 ± 16.7	59.4 ± 13.7	0.064
Race			
African-American	23 (24)	25 (37)	0.107
Caucasian	65 (68)	41 (60)	
Other	8 (8)	2 (3)	
All Arrhythmia			
Atrial fibrillation	21 (22)	27 (40)	0.013
Atrial flutter	2 (2)	8 (12)	0.017
Other cardiac dysrhythmia	23 (24)	18 (26)	0.714
Pulseless electrical activity	5 (5)	2 (3)	0.479
Ventricular fibrillation	43 (45)	20 (29)	0.046
Ventricular tachycardia	30 (31)	27 (40)	0.262
Min Temperature F°, Mean ± SD	90.7 ± 3.2	89.7 ± 7.8	0.568
Max Temperature F°, Mean ± SD	100.0 ± 2.2	100.2 ± 1.8	0.520
Time to death after therapeutic, hypothermia, Mean ± SD	12.0 ± 15.3	7.7 ± 13.7	0.138
Medical Diseases			
Chronic kidney disease	20 (21)	25 (37)	0.024
Diabetes Mellitus	20 (21)	37 (54)	<0.0001
Hypertension	56 (58)	53 (78)	0.008
Obstructive CAD	55 (57)	44 (65)	0.338
Pacemaker/ICD	17 (18)	22 (17)	0.991
Labs/Studies, median (IQR)			
BNP pg/mL	175 (51, 408)	314 (191, 767)	0.113
GFR mL/min/1.73m ² Admission	57 (41, 60)	47 (31, 60)	0.080
GFR mL/min/1.73m ² Discharge	60 (38, 60)	51 (29, 60)	0.046
LVEF % pre	50 (40, 60)	55 (32, 60)	0.626
LVEF % during	45 (25, 60)	45 (25, 60)	0.755
Max NSE ng/mL	30 (18, 86)	29 (20, 62)	0.767
Troponin ng/mL	2.9 (0.4, 8.9)	1.1 (0.3, 4.9)	0.040

BMI indicates body mass index in kilogram/meter squared; BNP pg/mL, brain natriuretic peptide in picogram/milliliter; CAD, coronary artery disease; F°, Fahrenheit; GFR mL/min/1.73m², glomerular filtration rate in milliliter/minute/1.73meter squared; ICD, implantable cardioverter defibrillator; IQR, interquartile range first and third quartile; LVEF % pre, left ventricular ejection fraction percent pre therapeutic hypothermia; LVEF %during, left ventricular ejection fraction percent during therapeutic hypothermia; Max, maximum; Min, minimum; NSE ng/mL, neuron-specific enolase in nanogram/milliliter; SD, standard deviation. **Bold** indicates statistical significance p < 0.05.

Table 2

Univariable and Multivariable Analysis of Predictors of Survival Post Therapeutic Hypothermia

	Univariable Model Odds Ratio Mortality (95% CI)	Multivariable Model Odds Ratio Mortality (95% CI)
BMI <30 kg/m ² vs ≥30 kg/m ²	0.50 (0.26 – 0.97)	0.36 (0.16 - 0.78)
Woman vs Man	1.48 (0.75 – 2.92)	
Age <60 vs ≥60	0.85 (0.45 - 1.58)	
Race African-American versus Caucasian	0.95 (0.47 – 1.91)	
Other versus Caucasian	1.59 (0.39 - 6.50)	
Cardiac Arrest versus non-cardiac arrest	0.84 (0.39 – 1.83)	
Max Temperature <median vs ≥median	1.80 (0.96 - 3.40)	1.88 (0.90 - 3.94)
Min Temperature <median vs ≥median	1.25 (0.67 - 2.34)	
Medical Diseases		
CKD vs no CKD	0.78 (0.39 - 1.57)	
DM vs no DM	1.76 (0.89 - 3.46)	
Hypertension vs no hypertension	0.48 (0.24 – 0.96)	0.28 (0.12 - 0.66)
Obstructive CAD vs no obstructive CAD	0.57 (0.29 - 1.10)	
Pacemaker/ICD vs no Pacemaker/ICD	0.28 (0.12 - 0.65)	0.23 (0.08 - 0.62)
Labs/Studies		
BNP <median vs ≥median	0.50 (0.20 - 1.22)	
GFR admission ≥median vs <median	1.15 (0.61 – 2.17)	
GFR discharge ≥median vs <median	0.29 (0.14 - 0.58)	0.25 (0.11 - 0.56)
LVEF pre <median vs ≥median	1.14 (0.53 - 2.44)	
LVEF during <median vs ≥median	1.31 (0.68 - 2.53)	
NSE <median vs ≥median	0.32 (0.13 - 0.77)	0.37 (0.14 - 0.96)
Troponin <median vs ≥median	1.30 (0.69 - 2.43)	

BMI indicates body mass index in kilogram/meter squared; BNP, brain natriuretic peptide; CAD, coronary artery disease; CKD, chronic kidney disease; CI, confidence interval; DM; diabetes mellitus; GFR, glomerular filtration rate; ICD, implantable cardioverter defibrillator; LVEF pre, left ventricular ejection fraction pre therapeutic hypothermia; LVEF during, left ventricular ejection fraction during therapeutic hypothermia; Max, maximum; Min, minimum; NSE, neuron-specific enolase; VS, versus. **Bold** indicates statistical significance $p < 0.05$.