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A history of smoking is associated with improved survival in patients treated with mild therapeutic hypothermia following cardiac arrest☆

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Abstract

Objectives—To assess the association between smoking and survival with a good neurologic outcome in patients following cardiac arrest treated with mild therapeutic hypothermia (TH).

Methods—We conducted a retrospective observational study of a prospectively collected cohort of 188 consecutive patients following cardiac arrest treated with TH between May 2007 and January 2012. Smoking status was retrospectively collected via chart review and was classified as “ever” or “never”. Primary endpoint was survival to hospital discharge with a good neurologic outcome and was compared between smokers and nonsmokers. Logistic regression analysis was used to assess the association between smoking status and neurologic outcome at hospital discharge; adjusting for age, initial rhythm, time to return of spontaneous circulation (ROSC), bystander CPR, and time to initiation of TH.

Results—Smokers were significantly more likely to survive to hospital discharge with good neurologic outcome compared to nonsmokers (50% vs. 28%, $p = 0.003$). After adjusting for age, initial rhythm, time to ROSC, bystander CPR, and time to initiation of TH, a history of smoking was associated with increased odds of survival to hospital discharge with good neurologic outcome (OR 3.54, 95% CI 1.41–8.84, $p = 0.007$).

Conclusions—Smoking is associated with improved survival with good neurologic outcome in patients following cardiac arrest. We hypothesize that our findings reflect global ischemic conditioning caused by smoking.

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Keywords

Smoking; Cardiac arrest; Post-resuscitation care; Therapeutic hypothermia

1. Introduction

The harmful effects of cigarette smoking on public health are well established.¹ Although cigarette smokers have a higher probability of experiencing an acute coronary syndrome, they also have a lower unadjusted mortality rate following an acute myocardial infarction (AMI).^{2–4} This phenomenon was recognized 25 years ago and was termed the “Smoker's Paradox”.^{5,6} Some studies attribute this observation to younger age and fewer co-morbidities in patients who smoke.^{4,7–9} However, other studies have shown that the association between smoking and improved outcome persists after adjusting for these variables.^{10–13} Since the introduction of the term “smoker's paradox”, it remains unclear if there is a direct causal relationship between a history of smoking and reduced mortality following AMI.

Little is known regarding the potential mechanisms that underlie the association between smoking and improved outcomes following AMI. Smoking causes low-levels of hypoxia in diffuse tissue beds,^{14,15} which may result in a greater tolerance of more severe, acute hypoxic injuries and attenuation of reperfusion injury. This effect, known as ischemic conditioning,¹⁶ has been demonstrated in numerous experimental models and has been shown to be both neuroprotective¹⁹ and cardioprotective.^{17–21} Reperfusion and reperfusion injury following AMI share many features of the reperfusion injury that occurs in cardiac arrest, albeit diffuse tissue beds are involved in the setting of cardiac arrest. Given the physiologic similarities and the previously described relationship between smoking and improved outcomes following AMI, we hypothesized that a history of smoking may be associated with improved outcomes following cardiac arrest in patients treated with mild therapeutic hypothermia (TH).

To the best of our knowledge, the effect of smoking has never been studied in patients following cardiac arrest. The aim of the study was to determine if a history of smoking is associated with improved survival with good neurologic outcome in patients following cardiac arrest treated with mild TH.

2. Methods

We conducted an observational study of a prospectively collected cohort of 188 consecutive patients following cardiac arrest who underwent mild TH in the cardiovascular care unit of an academic tertiary care hospital between May 2007 and January 2012. All cardiac arrest survivors, age 18 years or older, who were unresponsive or comatose after return of spontaneous circulation (ROSC) were eligible for the TH protocol. Brainstem reflexes (i.e. cough, gag, corneal) as well as pathologic/posturing movements were permissible. Exclusion criteria for TH included: spontaneous awakening with purposeful movement, known or suspected pregnancy, known terminal illness or limited life expectancy, do not resuscitate (DNR) orders, initial body temperature less than 34 °C (93.2 °F), recent major head/face trauma, cardiac arrest due to traumatic injury, and estimated time from arrest to ROSC of greater than 60 min. Relative exclusion criteria included known bleeding diathesis or ongoing bleeding, and when another cause of coma was present such as drug intoxication or pre-existing coma. The majority of the study patients were transferred from community emergency departments or directly to our institution from multiple emergency medical service (EMS) systems with unique pre-hospital protocols. Patients meeting TH criteria

were generally cooled with external icepacks and/or chilled intravenous saline en route. Post-cardiac arrest patients determined by their treating physician to be suitable for TH were cooled to a target temperature of 33 °C (± 1 °C) using an active surface cooling device (Arctic Sun, Medivance, Louisville, CO) for 24 h following the approximated time of ROSC. Target temperature was defined as the time a patient's core temperature reached 34 °C. After external cooling, all patients were actively rewarmed at 0.25 °C/h until normothermic (36 °C).²²

Following approval from the institutional review board, data were collected on all patients following cardiac arrest. In addition to baseline demographics, initial rhythm, time to ROSC, occurrence of bystander cardiopulmonary resuscitation (CPR), presence of shock at admission (defined as a SBP <90 or use of vasopressors), and Cerebral Performance Category (CPC) score at hospital discharge and follow up were collected. ROSC was estimated by retrospective review of the electronic chart and EMS "run sheets" and was defined as the time the patient was found pulseless to ROSC. Time to initiation of TH and time to reach target temperature were both calculated from time of ROSC. Time of initiation of cooling was defined as the time at which any cooling method was applied, either at our hospital or prior to arrival at our facility; commonly used methods included cold saline infusion, application of ice packs, or the application of our external cooling device.

Smoking status was retrospectively classified as "ever" or "never". Patients were considered to be smokers if they were ever labeled as a smoker in the electronic medical record by a health-care professional at any time, including during the index admission or during previous clinic visits or hospitalizations. The definition did not consider the amount smoked, or for former smokers the time since quitting. Patients were considered to be nonsmokers if they were labeled as a nonsmoker on the index admission and had never previously been labeled as a smoker in the medical record. Patients for whom smoking status was unknown, defined as never specifically mentioned in the electronic medical record, were excluded from this analysis. 82 patients were included prior to implementation of our CODE ICE standardized protocol, 56 (69%) were smokers. 106 patients were included after implementation of CODE ICE, 39 (39%) were smokers. Patients were considered to be previously healthy if they did not have a known chronic medical condition prior to cardiac arrest. Chronic medical conditions included coronary artery disease, congestive heart failure, arrhythmia, hypertension, chronic obstructive lung disease (COPD), chronic renal failure (CKD), neurologic disease, liver failure, disseminated malignancy, alcohol/drug abuse, obesity (BMI > 35), and insulin dependent diabetes mellitus (DM).

The primary endpoint was survival to hospital discharge with a good neurologic outcome. Neurologic outcome was assessed using the CPC score, with a "good" outcome defined as a score less than or equal to 2. The CPC score was developed as a measure of central nervous system function after cardiac arrest and has become the most commonly used outcome tool for this purpose.^{23–25}

Descriptive statistics are presented as the median (interquartile range) for continuous variables and frequency (percentage) for categorical variables. Wilcoxon rank sum test and Pearson Chi-square test were used to compare the baseline characteristics between smokers and non-smokers. Logistic regression analysis was used to assess the association between smoking status and neurologic outcome at hospital discharge while adjusting for age, initial rhythm, time to ROSC, bystander CPR, and time to initiation of TH. Another logistic model was fit to assess the association between smoking status and neurologic outcome with adjustment for the same set of covariates as well gender and comorbidities via a propensity score. A similar analysis was again done to analyze survival with good neurologic outcome in smokers and non-smokers at a median follow-up of 3 months. Odds ratios with 95%

confidence intervals are presented. All tests were two-tailed, with a significance level of 5%. All statistical analyses were performed using open source R statistical software (version 2.15.1, Vienna, Austria).

3. Results

Of 188 consecutive patients considered for inclusion, smoking status was determined in 181 patients (96%). Table 1 displays a comparison of baseline characteristics. Non-smokers had shorter times to initiation of TH (160 vs. 240 min, $p = 0.01$), shorter time to reach target temperature (320 vs. 450 min, $p < 0.01$), and spent more time at target temperature (21.0 vs. 17.0 h, $p < 0.01$). No statistically significant difference in age, gender, past medical history, initial rhythm following ROSC, sepsis, or shock on admission between smokers and non-smokers was observed.

The overall in-hospital mortality rate was 60% (108/181). Of the 181 patients, 110 (61%) had poor and 71 (39%) had good CPC neurologic status at time of discharge. Fig. 1 displays clinical outcomes based on smoking status. Smokers were significantly more likely to survive to hospital discharge with good neurologic outcome compared to nonsmokers (50% vs. 28%, $p = 0.003$). In addition, Fig. 1 compares outcomes in relation to CPC score for smokers versus non-smokers at both hospital discharge and at follow-up. At a median of 3 months follow-up, results were similar to hospital discharge: more smokers had a CPC score of 1–2 and more non-smokers had a CPC score of 3 or greater.

Multivariable analysis was performed to assess the relationship between smoking history and clinical outcome. After adjusting for age, initial rhythm, time to ROSC, bystander CPR, and time to initiation of TH, a history of smoking was independently associated with increased odds of neurologically intact survival at the time of hospital discharge (OR 3.54, 95% CI 1.41–8.84, $p < 0.01$) (Table 2a) (Fig. 2). Sensitivity analysis using a propensity score for smoking while also adjusting for other potentially important co-variables demonstrated a similarly significant independent association between smoking and a good neurologic outcome at hospital discharge (OR 3.82, 95% CI 1.43–10.24, $p < 0.01$) (Table 2b).

4. Discussion

The primary finding of this study is that smoking is independently associated with improved survival with good neurologic outcome following cardiac arrest. To the best of our knowledge, this is the first study examining the effect of smoking in this specific population.

Aune et al. recently concluded that the favorable prognosis of smokers following potentially lethal ischemic events is likely due to confounders, including younger age and fewer comorbidities.⁴ In our analysis, improved survival with good neurologic outcome observed in smokers after cardiac arrest cannot be explained solely by clinical co-variables including age, gender and comorbidities. There were no significant differences in age or baseline comorbidities between smokers and nonsmokers treated with TH following cardiac arrest, suggesting that smoking may influence outcomes by mechanisms other than age or comorbid conditions. In addition, despite having a non-significant trend to be more likely to receive bystander CPR and having significantly shorter times to initiate and achieve hypothermia, both of which have previously been shown to increase survival to discharge,^{26,27} lifelong nonsmokers had decreased survival to discharge and to follow up at a median of 3 months compared to smokers.

While the exact mechanism underlying the independent association between smoking and good neurological outcome following cardiac arrest remains unknown, one possible explanation is that smoking results in physiologic conditions that may confer protection from ischemic injury. Hypoxia induced by smoking might create an ischemic conditioned state that could attenuate the lethal effects of reperfusion injury after a devastating ischemic event.

Ischemic conditioning was first described by Murry et al.¹⁶ finding that brief episodes of nonlethal ischemia and reperfusion applied to an organ or tissue bed conferred protection against future lethal reperfusion injuries.¹⁷ Ischemic conditioning has shown benefit in attenuating ischemic-reperfusion injury in a number of recent clinical studies.^{18–20,34} In addition, other studies have demonstrated that ischemic conditioning can be applied and subsequently be protective to multiple different organs including the heart and the brain.^{35,36}

Patients who survive cardiac arrest experience severe acute global hypoxia followed by acute reperfusion injury. Reperfusion is essential to maintaining viable tissue following ischemia. Paradoxically, the process of reperfusion induces injury that blunts its benefits. Our study shows an independent association between a history of smoking and survival to hospital discharge with a good neurologic outcome. It is possible that our findings reflect global ischemic conditioning caused by smoking: smoking causes hypoxia in tissue, which may create an ischemic conditioned state, thus attenuating the effects of ischemia and reperfusion caused by cardiac arrest.

This study has several limitations. The retrospective observational study design allows us to only suggest associations rather than causation; and due to the study design and size, we were unable to adjust for all potential confounders. Data for this study were collected in a registry from a single center that did not contain a formal severity of illness score, such as the APACHE score. We attempted to adjust for health status in the propensity-matched multivariable analysis by including the presence of comorbidities. However, severity of illness could remain a residual confounder. In addition, we studied a relatively small cohort who may not be generalizable to a larger population, or to patients who suffer cardiac arrest, or are not treated with TH. Due to the retrospective collection of smoking status, we were unable to obtain smoking status on a small percentage of our population (7/188, 3.7%); these patients were excluded from the analysis. In addition, we were unable to utilize a strict definition of current or former tobacco use. For the purpose of this study, we relied on the assessment of multiple healthcare providers to differentiate smokers from non-smokers. It is possible that some patients labeled as nonsmokers had previously smoked during their life but at the time of admission were not smoking. However, this misclassification would result in biasing the results toward the null, since smokers would be misclassified in the non-smoker cohort. In addition, former smokers were grouped with active smokers regardless of how long previously they had stopped smoking. This also might bias the results toward the null if the ischemic conditioning effects of smoking dissipate after stopping. The “ever” vs. “never” smoking classification, along with the size of our cohort, prevents us from assessing the potential “dose-response” relationship between smoking and outcome. Finally, smokers had numerically higher rates of VT/VF as initial rhythm, lower rates of shock on arrival, and a longer time to initiation of TH. It is possible that this could bias our results to the null and that a re-analysis with a larger cohort might not be reproducible. While these baseline differences might account for some differences in outcomes, we attempted to correct for any potential differences in important baseline variables in multivariable regression analyses.

In conclusion, the present study demonstrates the independent association of smoking with increased survival with good neurologic outcome in patients following cardiac arrest. It is possible that ischemic conditioning caused by smoking may be the mechanism behind this

association. These data are hypothesis generating in need of further investigation in larger prospective cohorts of patients undergoing TH after cardiac arrest.

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Fig. 1.

Frequency of ordinal CPC scores at hospital discharge and at follow-up. Median time to follow up for non-smokers and smokers was 3 months. CPC = cerebral performance score.

Fig. 2. Forrest plot depicting odds ratio of having a good neurologic outcome. Six variables included in the analysis were age, smoking status, initial rhythm, time to ROSC, presence of bystander CPR and time to initiation of TH.

Table 1

Baseline characteristics.

	Non-smokers <i>n</i> = 86	Smokers <i>n</i> = 95	<i>P</i>
Age (years)	62 (49–73)	59 (52–64)	0.34
Male gender (%)	50 (58)	66 (70)	0.11
Previously healthy (%)	14 (16)	12 (13)	0.48
Initial Rhythm VT ^a /VF ^b (%)	45 (54)	62 (67)	0.061
Received bystander CPR (%)	51 (59)	45 (47)	0.11
Time to ROSC ^c (min)	20 (13–30)	17 (11–26)	0.15
Shock at admission (%)	30 (35)	21 (22)	0.06
Time to initiation of TH ^d (min)	160 (75–300)	240 (120–360)	<0.01
Time to reach target temperature (min)	320 (210–480)	450 (330–660)	<0.01
Time spent at target temperature (h)	21 (17–24)	17 (14–20)	<0.01
Witnessed arrest (%)	72 (84)	80 (84)	0.93
Coronary angiography performed (%)	54 (63)	59 (62)	0.92
IABP ^e (%)	16 (19)	12 (13)	0.27
Peak troponin (μg/L)	3.0 (0.57–17)	5.7 (1.2–27)	0.15
Pneumonia during ICU (%)	23 (27)	32 (34)	0.31
Severe sepsis/septic shock during ICU (%)	15 (17)	15 (16)	0.77
Other serious infection during ICU (%)	5 (6)	5 (5)	0.87
Time from arrest to hospital discharge (days)	9.0 (8.0–14)	10 (7.0–16)	0.72
Time from arrest to follow up (months)	3.5 (1.0–7.0)	2.0 (1.0–6.0)	0.08
STEMI (%)	15 (17)	25 (26)	0.16
Good outcome (%) at discharge	24 (28)	47 (50)	<0.01
Good outcome (%) at follow-up ^f	14 (19)	34 (40)	<0.01
Transfer from outside hospital (%)	61 (71)	74 (78)	0.05
Core temperature (°C) at presentation ^g	35.4, 36.3, 35.8 (36.0 ± 1.2)	36.0, 36.4, 37.0 (36.3 ± 1.2)	0.12

^aVentricular tachycardia.^bVentricular fibrillation.^cReturn of spontaneous circulation.^dTherapeutic hypothermia.^eIntra-aortic balloon pump.^fFollow-up at a median of 3 months.^g*a*, *b*, *c* represent the lower quartile *a*, the median *b*, and the upper quartile *c* for continuous variables. *X* ± *s* represents *X* ± 1 SD.

Table 2a

Odds ratios of having a good neurologic outcome.

	OR	<i>p</i>-Value
Age (year)	0.97 (0.94–1)	0.03
Smoking Status (smoker)	3.54 (1.41–8.84)	<0.01
Initial rhythm (VT ^a /VF ^b)	6.81 (2.54–18.29)	<0.01
Time to ROSC (min)	0.88 (0.83–0.93)	<0.01
Bystander CPR	3.02 (1.15–7.93)	0.03
Time to initiation of TH ^c (min)	1 (0.99–1)	0.04

^aVentricular tachycardia.^bVentricular fibrillation.^cTherapeutic hypothermia.

Table 2bOdds ratios of having a good neurologic outcome (propensity score).^a

	OR	<i>p</i>-Value
Age (year)	0.97 (0.94–1)	0.03
Smoking status (smoker)	3.82 (1.43–10.24)	<0.01
Initial rhythm (VT/VF)	6.79 (2.53–18.22)	<0.01
Time to ROSC (min)	0.88 (0.83–0.93)	<0.01
Bystander CPR	2.99 (1.14–7.84)	0.03
Time to initiation of TH (min)	1 (0.99–1)	0.04

^a Odds ratio for the propensity score is 0.91 (0.58–1.41) with *p*-value 0.666.