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Short paper

Collapse-related traumatic intracranial hemorrhage following out-of-hospital cardiac arrest: A multicenter retrospective cohort study



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Abstract

Background: Sudden loss of consciousness as a result of cardiac arrest can cause severe traumatic head injury. Collapse-related traumatic intracranial hemorrhage (CRTIH) following out-of-hospital cardiac arrest (OHCA) may be linked to poor neurological outcomes; however, there is a paucity of data on this entity. This study aimed to investigate the frequency, characteristics, and outcomes of CRTIH following OHCA.

Methods: Adult patients treated post-OHCA at 5 intensive care units who had head computed tomography (CT) scans were included in the study. CRTIH following OHCA was defined as a traumatic intracranial injury from collapse due to sudden loss of consciousness associated with OHCA. Patients with and without CRTIH were compared. The primary outcome assessed was the frequency of CRTIH following OHCA. Additionally, the clinical features, management, and consequences of CRTIH were analyzed descriptively.

Results: CRTIH following OHCA was observed in 8 of 345 enrolled patients (2.3%). CRTIH was more frequent after collapse outside the home, from a standing position, or due to cardiac arrest with a cardiac etiology. Intracranial hematoma expansion on follow up CT was seen in 2 patients; both received anticoagulant therapy, and one required surgical evacuation. Three patients (37.5%) with CRTIH had favorable neurological outcomes 28 days after collapse.

Conclusions: Despite its rare occurrence, physicians should pay special attention to CRTIH following OHCA during the post-resuscitation care period. Larger prospective studies are warranted to provide a more explicit picture of this clinical condition.

Keywords: Cardiac arrest, Computed tomography, Craniocerebral trauma, Intracranial hemorrhage, Out-of-hospital cardiac arrest

Introduction

Abrupt loss of consciousness following sudden cardiac arrest typically results in collapse, potentially leading to serious injury to the head, similar to that experienced following a collapse episode

caused by syncope or seizure. Collapse-related traumatic intracranial hemorrhage (CRTIH) may differ in incidence or outcomes based on the cause of collapse. Severe head trauma occurred in 2.6% of patients who experienced transient loss of consciousness secondary to syncope.¹ Furthermore, patients with severe head injury due to a fall secondary to a seizure comprised 3.8% of admitted patients with

Abbreviations: AAD, ascending aortic dissection, AMI, acute myocardial infarction, APTT, activated partial thromboplastin time, AS, aortic stenosis, CAG, coronary angiography, CPC, cerebral performance category, CPR, cardiopulmonary resuscitation, CRTIH, collapse-related traumatic intracranial hemorrhage, CT, computed tomography, ECMO, extracorporeal membrane oxygenation, ECPR, extracorporeal cardiopulmonary resuscitation, EMS, emergency medical service, F, female, IABP, intra-aortic balloon pumping, ICU, intensive care unit, IQR, interquartile range, M, male, OHCA, out-of-hospital cardiac arrest, PCI, percutaneous coronary intervention, PLT, platelet, PT, prothrombin time, ROSC, return of spontaneous circulation, VF, ventricular fibrillation.

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<https://doi.org/10.1016/j.resplu.2023.100418>

Received 27 May 2023; Received in revised form 6 June 2023; Accepted 8 June 2023

a head injury, and required neurosurgical intervention far more frequently than patients with head injury from a fall due to another cause (81.8% vs 32.3%).² To our knowledge, only one study has reported the incidence of CRTIH in patients admitted in the intensive care unit (ICU) following non-traumatic out-of-hospital cardiac arrest (OHCA).³ This single-center study found CRTIH in 1.1% of patients after OHCA (14/1310), but did not evaluate the characteristics of these individuals or the consequences of their injuries.

Importantly, patients with OHCA who require percutaneous coronary intervention (PCI), treatment for pulmonary embolism, or extracorporeal membrane oxygenation (ECMO) support are exposed to antiplatelet and anticoagulation agents.⁴⁻⁶ These therapies may potentially exacerbate intracranial hemorrhage, leading to poor neurological outcomes, and several reports have noted this challenge in treating patients after cardiac arrest.^{7,8} Thus, a better understanding CRTIH after OHCA is crucial to provide appropriate post-resuscitation care. This study aimed to investigate the frequency, characteristics, and outcomes of CRTIH following OHCA.

Methods

Study design and population

This was a multicenter, retrospective cohort study of patients admitted to 5 ICUs in Japan (1 academic medical center and 4 community medical centers) from January 1, 2007 to December 31, 2019. All adult patients (≥ 20 years of age) admitted in the ICU after OHCA were screened for eligibility. We excluded patients without head computed tomography (CT) scans on admission and patients whose cause of cardiac arrest was stroke or external (including asphyxia, trauma, drowning, and drug overdose or toxin ingestion). We further excluded bedridden patients with immobility prior to cardiac arrest and patients with a treatment restriction upon ICU admission. Study approval was obtained by the Ethics Committee of Okayama University Hospital (approval number: K2208-036). The requirement

for patient consent was waived because of the retrospective study design.

Definition

CRTIH following OHCA was defined as a traumatic intracranial injury as a result of collapse due to sudden loss of consciousness associated with OHCA. Traumatic intracranial injury included epidural hematoma, subdural hematoma, subarachnoid hemorrhage, and cerebral contusion/intraparenchymal hemorrhage as confirmed by CT imaging. The radiologist's report on each patient was thoroughly reviewed and the presence or absence of CRTIH was confirmed by emergency medicine physicians in each hospital.

Outcomes

The primary outcome assessed was the frequency of CRTIH following OHCA. Additionally, the clinical characteristics of the patients, particularly the patients with CRTIH, were analyzed including their situation at the time of collapse and in-hospital management.

Data collection

We systematically collected the following data from participants' medical records: clinical information (age, sex, medications), location of cardiac arrest, whether or not another individual witnessed the arrest, provision of bystander cardiopulmonary resuscitation (CPR), initial rhythm on scene, the etiology of cardiac arrest, resuscitation time (defined as the interval between the emergency medical services call and return of spontaneous circulation), emergency call time (daytime defined as 8 a.m. to 8 p.m. vs. nighttime defined as 8 p.m. to 8 a.m.⁹), laboratory data on emergency department arrival, whether or not patients received targeted temperature management, extracorporeal cardiopulmonary resuscitation (ECPR), and 28-day neurological outcomes. A favorable neurological outcome was defined as a cerebral performance category (CPC) score of 1 or 2. We collected additional information on individuals in the CRTIH group: position before collapse, ground materials at the collapse

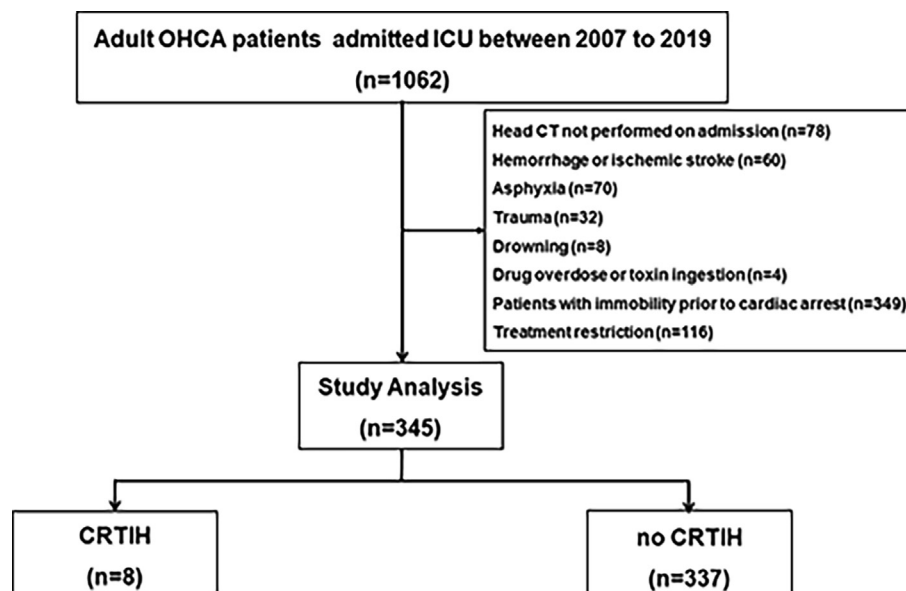


Fig. 1 – Flow diagram of study participants. OHCA, out-of-hospital cardiac arrest; ICU, intensive care unit; CT, computed tomography; CRTIH, collapse-related traumatic intracranial hemorrhage.

location, external signs of head trauma, CT findings, and post-resuscitation care and consequences.

Statistical analysis

Continuous variables are presented as median and interquartile range (IQR); categorical variables are presented as numbers

and percentages. Categorical variables were compared using the Fisher's exact probability test. The Mann-Whitney U test was used to evaluate variables with non-normal distributions. All statistical analyses were performed using the Stata version 17 (StataCorp LP, College Station, TX). Statistical significance was set at $P < 0.05$.

Table 1 – Patient Characteristics.

Variables	All (<i>n</i> = 345)	CRTIH (<i>n</i> = 8)	No CRTIH (<i>n</i> = 337)	<i>P</i> -value
Age, median years (IQR)	68 (58–75)	69 (56–72)	68 (58–76)	0.726
Sex				0.723
Male, <i>n</i> (%)	239 (69.3)	6 (75.0)	233 (69.1)	
Female, <i>n</i> (%)	106 (44.4)	2 (25.0)	104 (30.9)	
Timing of EMS call, <i>n</i> (%) ^a				0.170
Day	215 (64.6)	7 (87.5)	208 (64.0)	
Night	118 (35.4)	1 (12.5)	117 (36.0)	
Location of collapse, <i>n</i> (%) ^b				0.023
Home	178 (52.0)	1 (12.5)	177 (53.0)	
Other setting	164 (48.0)	7 (87.5)	157 (47.0)	
Medication, <i>n</i> (%)				
Anticoagulant agents ^c	36 (12.0)	1 (14.3)	35 (11.9)	0.534
Antiplatelet agents ^d	64 (21.3)	2 (28.6)	62 (21.2)	0.848
Witnessed cardiac arrest, <i>n</i> (%) ^e	250 (74.6)	8 (100)	242 (74.0)	0.095
Bystander CPR, <i>n</i> (%) ^f	175 (53.4)	6 (75.0)	169 (52.8)	0.214
Initial rhythm, <i>n</i> (%) ^g				0.886
Shockable	162 (47.5)	4 (50.0)	158 (47.4)	
Non-shockable	179 (52.5)	4 (50.0)	175 (52.6)	
Etiology of cardiac arrest, <i>n</i> (%) ^h				0.259
Cardiac	237 (69.3)	7 (87.5)	230 (68.9)	
Non-cardiac	105 (30.7)	1 (12.5)	104 (31.1)	
Prehospital ROSC, <i>n</i> (%)	96 (27.8)	3 (37.5)	93 (27.6)	0.537
Time interval from collapse to ROSC—median (IQR), min ⁱ	30 (20–45)	34 (18–49)	30 (20–45)	0.967
Laboratory data on admission				
Lactate—median (IQR), mmol/l ^j	10.5 (7.9–14.5)	10.5 (6.1–13.9)	10.5 (8.0–14.5)	0.689
PLT—median (IQR), 10 ⁴ /μL	16.1 (6.6–22.3)	20.1 (8.3–22.9)	16.0 (6.6–22.3)	0.496
PT—median (IQR), % ^k	13.5 (11.5–15.6)	13.7 (11.8–15.1)	13.4 (11.5–15.6)	0.709
APTT—median (IQR), sec ^l	37.0 (28.3–53.1)	35.0 (24.9–47.2)	37.3 (28.3–53.4)	0.533
Pre-CPC, <i>n</i> (%) ^m				0.216
CPC 1	289 (84.3)	8 (100)	281 (83.9)	
CPC 2	54 (15.7)	0 (0)	54 (16.1)	
Targeted temperature management, <i>n</i> (%)	196 (56.8)	6 (75.0)	190 (56.3)	0.293
ECPR, <i>n</i> (%)	47 (13.6)	3 (37.5)	44 (13.1)	0.046
Neurological Outcome at 28 days, <i>n</i> (%)				0.674
CPC 1–2	106 (30.7)	3 (37.5)	103 (30.6)	
CPC 3–5	239 (69.3)	5 (62.5)	234 (69.4)	

Data are presented as median [interquartile range] for continuous variables or as *N*(percentage) for categorical variables. *P* values were calculated using Fisher's exact probability test or Mann-Whitney U test.

APTT, activated partial thromboplastin time; CPC, cerebral performance category; CPR, cardiopulmonary resuscitation; CRTIH, collapse related traumatic intracranial hemorrhage; ECPR, extracorporeal cardiopulmonary resuscitation; EMS, emergency medical service; IQR, interquartile range; PT, prothrombin time; PLT, platelet; ROSC, return of spontaneous circulation.

^a Data missing in 12 patients.

^b Data missing in 3 patients.

^c Data missing in 44 patients.

^d Data missing in 45 patients.

^e Data missing in 10 patients.

^f Data missing in 17 patients.

^g Data missing in 4 patients.

^h Data missing in 3 patients.

ⁱ Data missing in 45 patients.

^j Data missing in 78 patients.

^k Data missing in 78 patients.

^l Data missing in 74 patients.

^m Data missing in 2 patients.

Table 2 – Characteristics, and outcome of collapsed-related traumatic intracranial hemorrhage.

Patient	#1	#2	#3	#4	#5	#6	#7	#8
Sex	M	M	M	F	M	F	M	M
Age, years	<60	<60	60–69	60–69	>70	>70	>70	>70
Etiology	VF	AMI	VF	AMI	AAD	AMI	AMI	AS
Standing position at time of arrest	Yes	Yes	Yes	Yes	No	None noted	Yes	Yes
Location at time of arrest	Outdoor	Outdoor	Outdoor	Outdoor	Outdoor	Indoor	Indoor	Indoor
Ground composition at location of arrest	Concrete	Concrete	Concrete	Concrete	Sand	Flooring	Flooring	Flooring
External findings	Subcutaneous hematoma in occiput region	Subcutaneous hematoma	None noted	Frontal contusion	None noted	None noted	Subcutaneous hematoma	Subcutaneous hematoma in parietal region
CT timing	Following ECMO initiation	Following ROSC	Following ROSC	Following ROSC	Following ECMO initiation	Following ECMO initiation	Following ROSC	Following ROSC
CT findings	Subdural hematoma/ Subarachnoid hemorrhage	Subdural hematoma	Subarachnoid hemorrhage	Subarachnoid hemorrhage	Subdural hematoma	Cerebral contusion	Subdural hematoma/ Subarachnoid hemorrhage	Subdural hematoma
Rotterdam CT score	3	2	2	2	2	2	2	2
Anticoagulant use after admission	Yes	Yes	No	Yes	Yes	Yes	Yes	No
Antiplatelet use after admission	No	Yes	No	No	No	Yes	No	No
IABP	No	No	No	No	No	No	Yes	No
ECMO	Yes	No	No	No	Yes	Yes	No	No
CAG	Yes	Yes	Yes	Yes	No	Yes	Yes	No
PCI	No	Yes	No	Yes	No	Yes	No	No
Change in hematoma size on follow-up CT	Follow-up CT was not performed	Expansion	No change	No change	Reduction	No change	Expansion	No change
Neurosurgery	No	Craniotomy	No	No	No	No	No	No
CPC score at 28 days	5	1	1	4	2	5	5	4

AAD, ascending aortic dissection; AMI, acute myocardial infarction; AS, aortic stenosis; CAG, coronary angiography; CPC, cerebral performance category; CT, computed tomography; ECMO, extracorporeal membrane oxygenation; F, female; IABP, intra-aortic balloon pumping; M, male; PCI, percutaneous coronary intervention; VF, ventricular fibrillation.

Results

Of 1062 adult patients hospitalized in the ICU after OHCA, 345 patients met the inclusion criteria and were included in the final analysis (Fig. 1). Their median age was 68 years old (IQR 58–75 years); 239 (69.3%) were male, and 106 (30.7%) were female. Of the 345 patients, 8 (2.3%) were identified with CRTIH. Baseline characteristics of the patients are shown in Table 1. OHCA occurred outside home more frequently in the patients with CRTIH (87.5% vs. 47.0% with no CRTIH, $p = 0.023$), and bystanders witnessed the collapse of all patients with CRTIH. The initially documented heart rhythm and the proportion of patients who were on antiplatelet or anticoagulant agents at the time of collapse were not different between patients with and without CRTIH. Three patients with CRTIH received ECPR for circulatory support (Table 1).

Of the 8 patients with CRTIH, 7 experienced OHCA due to a cardiac etiology (Table 2). Six were standing before collapse. Half of the patients (4/8) were injured on a concrete floor. External signs of head trauma were noted in 5 patients. Patients received anticoagulant treatment (6 patients), antiplatelet treatment (2 patients), and extracorporeal membrane oxygenation (ECMO) (3 patients) during post-resuscitation care. Hematoma size increased between initial and follow-up CT in 2 patients; both received anticoagulation therapy post-resuscitation. One received antiplatelet therapy as well and required a craniotomy. Favorable neurological outcomes at 28 days post-collapse were observed in 3 patients (37.5%) including the patient who required craniotomy.

Discussion

In this study, CRTIH was observed in 2.3% of patients after OHCA. CRTIH after OHCA occurred most often in patients who collapsed from a standing position, outside the home, and was almost always after cardiac arrest of cardiac origin. ECMO was instituted post-OHCA in 3 of the 8 patients. CRTIH worsened during hospitalization in 2 patients, 1 of whom required neurosurgical intervention.

The frequency of CRTIH following OHCA that we observed (2.3%) is fairly consistent with that seen by Champigneulle and colleagues (CRTIH in 1.1% of patients after OHCA).³ Our findings are also in line with studies in patients with syncope who can suddenly lose consciousness, potentially resulting in CRTIH by a similar mechanism as cardiac arrest^{1,10}. In a recent study of patients with syncope, 12% experienced injuries, and 4% had injuries classified as severe, which included, but was not limited to, head injuries.¹⁰

As expected, CRTIH was seen more frequently when a patient collapsed from a standing position, on a hard surface, outside the home, and when OHCA was caused by a cardiac etiology resulting in abrupt cessation of cerebral circulation.¹¹ Although we did not observe differences in proportions of patients with and without CRTIH who were taking anticoagulant or antiplatelet agents prior to collapse, these drugs are known to increase the risk of CRTIH and hematoma expansion.^{12,13} Thorough external physical examination, collection of information, including the patient's medication history and the circumstances at the moment of cardiac arrest, is essential to recognize patients at risk for CRTIH following OHCA.

CRTIH may also develop or progress during the post-resuscitation period due to use of anticoagulant or antiplatelet agents

after cardiac arrest. A comprehensive review that developed a consensus definition of patients at high bleeding risk post-PCI included recent traumatic intracranial hemorrhage as a major criteria for high bleeding risk. CRTIH was not considered as a separate entity, however.¹⁴ Moreover, special medical attention is needed in patients supported with ECMO, during which anticoagulants are normally mandatory. In this study, ECPR was administered in 3 patients, and although we believe, based on the pathophysiology, that intracranial hemorrhage occurred because of the event of hitting the head, we cannot rule out the possibility that ECPR led to hemorrhage. Several studies suggest that routine whole-body CT after ECPR is reasonable to detect any intracranial hemorrhage, which could have a huge impact on outcomes after OHCA.¹⁵

Limitations

Our study has several limitations. We focused on patients who were admitted to the ICU without severe disability before the cardiac event and without treatment restrictions. This excluded some patients who may have had CRTIH. We also excluded patients without brain CT scans, and some relevant information about the patients or the arrest events may be lacking due to retrospective design of the study. Thus, the prevalence of CRTIH found in this study may not be precise. Additionally, without more sensitive imaging modalities, such as digital subtraction angiography or magnetic resonance imaging, we cannot be absolutely certain that all subarachnoid hemorrhages classified as CRTIH were traumatic in origin. Finally, because only a small number of patients with CRTIH were identified, we could not perform multivariable logistic regression analysis to explore the risk factors associated with CRTIH, and clinically relevant conclusions that can be taken from this work are limited. However, by examining the characteristics of patients who developed CRTIH and the consequences of CRTIH in these patients for the first time, this study provides highly pragmatic information on the management of CRTIH during post-resuscitation care, despite these limitations.

Conclusion

Although CRTIH following OHCA might not be common, we advocate that physicians recognize CRTIH in patients with OHCA and pay attention to its potentially profound impact on neurological outcomes and the potential for devastating neurological damage if left untreated.

Ethics approval and consent to participate

This study conforms to the principles outlined in the Declaration of Helsinki and was approved by ethics committee of the Okayama University Hospital (approval number: K2208-036). Patient consent was waived for all participants enrolled in this study because of the retrospective study design.

Consent for publication

Consent for publication was waived.

Availability of data and materials

The datasets from this study are available from the corresponding author upon request.

Funding

No funding was received for this work.

CRedit authorship contribution statement

Fumiya Inoue: Conceptualization, Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Takashi Hongo:** Conceptualization, Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Toshihisa Ichiba:** Conceptualization, Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Takayuki Otani:** Conceptualization, Formal analysis, Writing – original draft, Writing – review & editing. **Hiroshi Naito:** Formal analysis, Writing – original draft, Writing – review & editing. **Yoshinori Kosaki:** Data curation, Writing – review & editing. **Yuya Murakami:** Data curation, Writing – review & editing. **Atsuyoshi Iida:** Data curation, Writing – review & editing. **Tetsuya Yumoto:** Formal analysis, Writing – review & editing. **Hirohichi Naito:** Conceptualization, Data curation, Formal analysis, Writing – original draft, Writing – review & editing, Supervision, Project administration. **Atsunori Nakao:** Writing – original draft, Writing – review & editing, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

The authors thank Shannon Wyzomierski for editing the manuscript.

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