Resuscitation 80 (2009) 977–980

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Clinical paper

Subarachnoid haemorrhage as a cause of out-of-hospital cardiac arrest: A prospective computed tomography study☆

Joji Inamasu[a,b,*], Satoru Miyatake[a], Hideto Tomioka[a], Masaru Suzuki[a], Masashi Nakatsukasa[b], Naoki Maeda[c], Takeshi Ito[c], Kunihiko Arai[c], Masahiro Komura[c], Kenichi Kase[a], Kenji Kobayashi[a]

[a] Department of Emergency Medicine, Saiseikai Utsunomiya Hospital, Utsunomiya, Japan
[b] Department of Neurosurgery, Saiseikai Utsunomiya Hospital, Utsunomiya, Japan
[c] Department of General Internal Medicine, Saiseikai Utsunomiya Hospital, Utsunomiya, Japan

ARTICLE INFO

Article history:
Received 16 January 2009
Received in revised form 2 May 2009
Accepted 11 May 2009

Keywords:
Brain computed tomography
Out-of-hospital cardiac arrest
Subarachnoid haemorrhage
Ventricular fibrillation

ABSTRACT

Aim: Aneurysmal subarachnoid haemorrhage (SAH) is a relatively common cause of out-of-hospital cardiac arrest (OHCA). Early identification of SAH-induced OHCA with the use of brain computed tomography (CT) scan obtained immediately after resuscitation may help emergency physicians make therapeutic decision as quickly as they can.
Methods: During the 4-year observation period, brain CT scan was obtained prospectively in 142 witnessed non-traumatic OHCA survivors who remained haemodynamically stable after resuscitation. Demographics and clinical characteristics of SAH-induced OHCA survivors were compared with those with "negative" CT finding.
Results: Brain CT scan was feasible with an average door-to-CT time of 40.0 min. SAH was found in 16.2% of the 142 OHCA survivors. Compared with 116 survivors who were negative for SAH, SAH-induced OHCA survivors were significantly more likely to be female, to have experienced a sudden headache, and trended to have achieved return of spontaneous circulation (ROSC) prior to arrival in the emergency department less frequently. Ventricular fibrillation (VF) was significantly less likely to be seen in SAH-induced than SAH-negative OHCA (OR, 0.06; 95% CI, 0.01–0.46). Similarly, Cardiac Trop-T assay was significantly less likely to be positive in SAH-induced OHCA (OR, 0.08; 95% CI, 0.01–0.61).
Conclusion: Aneurysmal SAH causes OHCA more frequently than had been believed. Immediate brain CT scan may particularly be useful in excluding SAH-induced OHCA from thrombolytic trial enrollment, for whom the use of thrombolytics is contraindicated. The low VF incidence suggests that VF by itself may not be a common cause of SAH-induced OHCA.

1. Introduction

Treatment of out-of-hospital cardiac arrest (OHCA) survivors has changed dramatically in the last decade. Aggressive therapeutic intervention, such as therapeutic hypothermia, immediately after resuscitation has been shown to improve the outcome of OHCA survivors, particularly when OHCA is in ventricular fibrillation (VF) rhythm of cardiac origin.[1,2] It is often difficult to distinguish accurately in emergency settings, however, whether an OHCA survivor has a cardiac cause or not: for example, it has been reported that patients with aneurysmal subarachnoid haemorrhage (SAH) may collapse into coma and cardiac arrest without complaining of a preceding "warning" headache.[3] Since 2004, we have been performing a brain computed tomography (CT) scan prospectively for OHCA survivors immediately after resuscitation, with an expectation that early identification of SAH that causes OHCA may help emergency physicians make therapeutic decision as quickly as they can. The incidence and clinical characteristics of SAH-induced OHCA is evaluated. Furthermore, whether it is feasible to predict or rule out the presence of SAH among OHCA survivors without obtaining a brain CT scan is discussed. The term "survivor" used here indicates patients who are successfully resuscitated from OHCA, but does not necessarily mean that they remain alive in the long-term.

2. Methods

This study was carried out between January 2004 and December 2007 in a single institution, which is a tertiary referral center and covers a local population of approximately 500,000. Cardiopul-

☆ A Spanish translated version of the summary of this article appears as Appendix in the final online version at doi:10.1016/j.resuscitation.2009.05.010.

* Corresponding author at: Department of Emergency Medicine, Saiseikai Utsunomiya Hospital, 911-1 Takebayashi, Utsunomiya, Tochigi 321-0974, Japan. Tel.: +81 28 626 5500; fax: +81 28 626 5594.
E-mail address: ginamasu@aol.com (J. Inamasu).

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doi:10.1016/j.resuscitation.2009.05.010

monary resuscitation (CPR) for OHCA victims is performed in accordance with the latest Basic/Advanced Cardiac Life Support guidelines.[4,5] Prehospital data were documented in the Utstein style,[6] and prehospital ECG was recorded routinely by emergency medical services (EMS) consisting solely of properly trained paramedics. They routinely provide airway protection/oxygenation, standard chest compression and administration of intravenous (IV) saline. Automated external defibrillator is used for VF/ventricular tachycardia rhythm, and IV epinephrine is used for pulseless electrical activity (PEA) and asystole whenever necessary. After arrival in the emergency department (ED) and successful CPR/stabilization, i.e., return of spontaneous circulation (ROSC) and sustainable blood pressure (defined as systolic pressure >80 mmHg), non-traumatic OHCA survivors were brought immediately to a CT suite adjacent to the ED. Presence of possible myocardial injury was screened with Cardiac Trop-T sensitive assay (Roche Diagnostics, Tokyo, Japan) in most OHCA survivors. Exclusion criteria for the immediate brain CT scan protocol include: (1) when blood pressure cannot be maintained even with the use of maximal dose of vasopressors; and (2) when the cause of OHCA is non-traumatic but extrinsic, such as asphyxia, drug overdose, and near-drowning. The brain CT scan protocol for non-traumatic OHCA survivors was approved by the internal review board of our institution.

During the 4-year period, a total of 173 haemodynamically stable non-traumatic OHCA survivors underwent a brain CT scan immediately after resusciation. Among them, 31 survivors were excluded from the analysis because their collapse had not been witnessed, in whom estimation of time from OHCA to ROSC was impossible. For the remaining 142 witnessed OHCA survivors, medical records were reviewed thoroughly and brain CT images were evaluated by board-certified radiologists. Attention was paid to rule out pseudo-SAH, which is defined as hypoxia-induced high attenuation areas along the basal cisterns or cortical sulci mimicking SAH.[7,8] The condition has been thought to result from combination of decreased CT attenuation of the brain parenchyma and distension of the superficial vessels as a consequence of elevated intracranial pressure associated with severe brain edema.[7,8] Measurement of CT value may be useful in differentiating pseudo-SAH, whose values are lower than those of aneurysmal SAH.[8]

2.1. Statistical analysis

Continuous variables, expressed as mean ± SD, were compared using the Student's-*t* test, and categorical variables using chi-square test. Odds ratio (OR) and 95% confidence interval (CI) was calculated from 2 × 2 tables. Statistical analyses were performed using SPSS 13.0 for Windows, and $p < 0.05$ was considered statistically significant.

3. Results

3.1. Incidence of intracranial haemorrhage and SAH

During the 4-year observation period, the door-to-CT time for the 142 witnessed non-traumatic OHCA survivors ranged from 21 to 70 min, with the average of 40.0 min. No adverse events occurred during transfer to the CT suite or on the CT table. Twenty-six of the 142 survivors (18.3%) who underwent an immediate brain CT scan were shown to have an intracranial haemorrhage which was considered responsible for the OHCA. Among the 26 survivors, 23 had a non-traumatic SAH, 2 had a cerebellar haemorrhage, and one had a brainstem haemorrhage. Thus, the incidence of SAH among OHCA survivors was 16.2%. The three survivors with an intracerebral haemorrhage will not be discussed further.

3.2. Clinical characteristics of SAH-induced OHCA

Clinical characteristics of those 23 SAH-induced OHCA survivors were evaluated. The male:female ratio was 5:18. The age ranged from 33 to 91 years, with the average of 65.3 years. Only two (8.7%) had been known to harbor a cerebral aneurysm which were treated conservatively. All had a CT-evidence of diffuse, thick SAH in the basal cistern and/or ventricular system: according to the Fisher SAH grading scale,[9] eight survivors (34.8%) had a Fisher grade III and the other 15 (65.2%) had a grade IV SAH. There were no patients who were diagnosed with a pseudo-SAH.

Eleven (47.8%) had complained of a sudden headache before collapse. Twelve (52.2%) had received a bystander CPR before EMS arrival. Estimated time from OHCA to ROSC ranged from 8 to 48 min, with the average of 26.4 min. Only three (13.0%) had achieved ROSC prior to ED arrival, and the other 20 (87.0%) achieved ROSC after CPR in the ED. Electrophysiologically, initial rhythms after collapse were PEA in 12 (52.2%), asystole in 10 (43.5%), and VF in 1 (4.3%), respectively. Bedside Cardiac Trop-T assay was performed in 19 (82.6%), and only one (5.3%) tested positive. All the 23 survivors died either in the intensive care unit or in the neurosurgical ward. Their survival period ranged from 1 day to 16 days, with the average of 4.2 days. Twenty-two (95.7%) were brain dead. The other (4.3%) restored some of brainstem functions, but eventually died of systemic infection. Autopsy was not carried out in any of them.

3.3. SAH-induced vs. SAH-negative OHCA

There were 116 OHCA survivors whose brain CT scan was negative for SAH. The male:female ratio was 70:46. The age ranged from 32 to 94 years, with the average of 68.3 years. Fifty survivors (43.1%) had received a bystander CPR before EMS arrival. Estimated time from OHCA to ROSC ranged from 6 to 51 min with the average of 28.0 min, and 40 (34.5%) had achieved ROSC prior to ED arrival. Only two (1.7%) had complained of a sudden headache before collapse. Initial rhythms were VF in 50 (43.1%), asystole in 36 (31.0%), and PEA in 30 (25.9%), respectively. Bedside Cardiac Trop-T assay was performed in 96 (82.8%), and among them, 40 (41.7%) tested positive. Twenty-three of the 116 survivors were discharged alive, with a survival rate of 19.8%.

The 23 SAH-induced and 116 SAH-negative survivors were compared (Table 1). The SAH-induced group was significantly more likely to be female-dominant ($p < 0.001$) and trended to have achieved ROSC prior to ED arrival less frequently (OR, 0.29; 95% CI, 0.08–1.02). Sudden headache before collapse was far more likely to be experienced in SAH-induced OHCA (OR, 52.25; 95% CI, 10.34–263.98). VF was significantly less likely to be seen in SAH-induced compared with SAH-negative OHCA (OR, 0.06; 95% CI, 0.01–0.46). Similarly, Cardiac Trop-T assay was significantly less likely to be positive in SAH-induced OHCA (OR, 0.08; 95% CI, 0.01–0.61). The survival rate to discharge was significantly lower for SAH-induced OHCA (0% vs. 19.8%, $p < 0.05$). Regarding other variables, i.e., age, estimated time from OHCA to ROSC, and frequency of bystander CPR, there were no significant differences between the two groups (Table 1).

4. Discussion

Intracranial haemorrhage is a relatively common cause of OHCA in adults, and among various subtypes of haemorrhage, aneurysmal SAH causes OHCA most frequently.[10] In this study, SAH was observed in as many as 16.2% of haemodynamically stable non-traumatic OHCA survivors who underwent an immediate brain CT scan. SAH was diffuse and thick in all the 23 survivors, whose Fisher SAH grade was either III or IV. Although there have been no