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# Headache, cardiac arrest, and intracranial hemorrhage

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**Abstract** Headache is one of the most common manifestations of non-traumatic intracranial hemorrhage, which is an uncommon, but not rare, cause of cardiac arrest in adults. History of a sudden headache preceding collapse may be a helpful clue to estimate the cause of out-of-hospital cardiac arrest (OHCA). Medical records of witnessed OHCA patients were reviewed to identify those who complained of a sudden headache preceding collapse, and the incidence of intracranial hemorrhage among them as well as their clinical characteristics was investigated retrospectively. During the 12-month period, 124 patients who sustained a witnessed OHCA were treated. Among them, 74 (60%) collapsed without any pain complaint, and only 6 (5%) complained of a sudden headache preceding collapse. All of the six patients were resuscitated: four had a severe subarachnoid hemorrhage (SAH), while the other two had a massive cerebellar hemorrhage. By contrast, 39 of the 74 patients who collapsed without any pain were resuscitated. Among them, another six patients were found to harbor an SAH. Thus, a total of 12 among the 124 witnessed OHCA (10%) sustained a fatal intracranial hemorrhage. While OHCA patients who collapse complaining of a sudden headache are uncommonly seen in the emergency room, they have a high likelihood of harboring a severe intracranial hemorrhage. It should also be reminded that approximately half of patients whose cardiac arrest is due to an intracranial hemorrhage may collapse without complaining of a headache. The prognosis of those with cerebral origin of OHCA is invariably poor, although they may relatively easily be resuscitated temporarily. Focus needs to be directed to avoid sudden death from a potentially treatable cerebral lesion, and public education to promote the awareness for the symptoms of potentially lethal hemorrhagic stroke is warranted.

**Keywords** Headache · Out-of-hospital cardiac arrest · Intracranial hemorrhage · Subarachnoid hemorrhage

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## Introduction

Sudden headache is one of the most common manifestations of non-traumatic intracranial hemorrhage: more than 90% of patients with subarachnoid hemorrhage (SAH) and 30–40% of those with intracerebral hemorrhage (ICH) complain of a sudden headache on admission [1, 2], and the presence of altered mental status preceded by a sudden headache suggests that the patient may sustain a severe intracranial hemorrhage. Not surprisingly, intracranial hemorrhage is also an uncommon, but not rare, cause of cardiac arrest and/or sudden death in adults [3–7]. It is likely that patients with an intracranial hemorrhage who present with out-of-hospital cardiac arrest (OHCA) may experience a sudden headache before collapse, as chest pain is often a prodrome in cardiogenic OHCA patients. Although it is often difficult to identify the cause of cardiac

arrest in emergency settings, a sudden headache preceding collapse may be a helpful clue to estimate its cause. We conducted a retrospective study to evaluate the relationship between sudden headache and cerebral origin of OHCA, as there has been few data on the subject in the literature.

## Patients and methods

This is a retrospective study conducted in a single institution, which is a tertiary referral center and covers a local population of approximately 500,000. The observation period was between 1 January and 31 December 2007. In all circumstances, OHCA patients were transferred by emergency medical services and cardiopulmonary resuscitation (CPR) was performed in accordance with the latest Basic/Advanced Cardiac Life Support guidelines [8]. As soon as patients were successfully resuscitated, a brain computed tomography (CT) scan was obtained to rule out the presence of intracranial pathologies. The witnesses (family members, friends, colleagues, or passersby) were interviewed in the Emergency Department, and the information of when and how they collapsed was recorded in a medical chart by emergency medicine residents. The charts were reviewed to identify OHCA patients who complained of a sudden headache within 2 h of collapse, and the incidence of intracranial hemorrhage as well as their clinical characteristics was investigated. Only patients whose collapse had been witnessed were included in the evaluation. Patients who sustained a witnessed OHCA with a nontraumatic but exogenous cause, such as asphyxia, were also excluded from the study.

## Results

During the 12-month period, a total of 124 adult patients who sustained a witnessed OHCA and were transferred to our institution were identified. The male to female ratio was 1.4:1. The age ranged from 23 to 92 years, with the average of 68 years. Among the 124 patients, 74 (60%) collapsed suddenly without complaining of any pain. Only six patients (5%) complained of a sudden headache within 2 h of collapse. Other pain or complaints associated with collapse include: chest pain/discomfort in 28 (22%), abdominal pain in 5 (4%), back pain in 2 (2%), and others in 9 (7%). All six patients with a sudden headache were resuscitated with a sustainable blood pressure. The interval between OHCA and return of spontaneous circulation (ROSC) ranged from 10 to 41 min, with an average of 26 min. Brain CT scan demonstrated that four had a dense SAH in the basal cistern, while the other two patients had a massive cerebellar hemorrhage. Two of the four SAH patients had experienced several bouts of a sudden headache 6–24 h before collapse, while the other two SAH patients complained of a sudden headache only at the time of collapse. The two ICH patients collapsed 10–20 min after complaining of a sudden headache. Only two of the six patients were known to have suffered from a chronic headache, and none of the six patients had sought medical attention before collapse (Table 1).

Among the 74 OHCA patients who collapsed without any prodromal pain, 39 (53%) were resuscitated temporarily and underwent a brain CT scan. Among them, another six patients were found to have a dense SAH. The interval between OHCA and ROSC in the six patients

**Table 1** Demographics and clinical characteristics of 12 patients with an intracranial hemorrhage presenting with out-of-hospital cardiac arrest

| Group | Age, gender | Type of hemorrhage | Timing of HA versus collapse | OHCA–ROSC interval (min) | Hx of chronic HA | Outcome |
|---|---|---|---|---|---|---|
| HA (+) | 36 F | SAH | HA only at the time of collapse | 10 | Unknown | Death |
| | 53 F | SAH | Repeated HA episodes 12 h before collapse | 31 | Unknown | Death |
| | 68 F | SAH | Repeated HA episodes 6 h before collapse | 14 | Unknown | Death |
| | 91 F | SAH | HA only at the time of collapse | 25 | Known | Death |
| | 57 M | Cerebellar ICH | HA 10 min before collapse | 37 | Known | Death |
| | 64 M | Cerebellar ICH | HA 15 min before collapse | 41 | Unknown | Death |
| HA (−) | 54 F | SAH | N/A | 36 | N/A | Death |
| | 55 F | SAH | N/A | 24 | N/A | Death |
| | 66 M | SAH | N/A | 25 | N/A | Death |
| | 74 F | SAH | N/A | 31 | N/A | Death |
| | 77 F | SAH | N/A | 22 | N/A | Death |
| | 85 F | SAH | N/A | 38 | N/A | Death |

*HA* headache, *Hx* history, *ICH* intracerebral hemorrhage, *N/A* not available, *ROSC* return of spontaneous circulation, *SAH* subarachnoid hemorrhage

ranged from 22 to 38 min, with an average of 29 min. Therefore, a total of 12 among the 124 witnessed OHCA patients (10%) had sustained a fatal intracranial hemorrhage. The incidence of SAH was 8%, or 10 in the 124 patients. Demographics and clinical characteristics of the 12 intracranial hemorrhage patients are summarized in Table 1. Nine of the ten SAH patients were female. None of the 12 patients survived despite intensive conservative care. Autopsy to clarify the cause of death in OHCA patients was not performed routinely in our institution.

## Discussion

Intracranial hemorrhage is an uncommon, but not rare, cause of sudden death in adults [3–7]. The definition of sudden death is, however, rather ambiguous: although it occasionally means instantaneous death, moderately progressive death may also be regarded as sudden death, particularly when there are no witnesses of the collapse. In this context, the definition of sudden death is less strict and specific than witnessed OHCA. Although the terms "cardiac arrest" and "sudden death" have often been used interchangeably, the critical difference should be noted: technically, patients with OHCA may not be dead yet. The incidence of intracranial hemorrhage as a cause of sudden death in adults according to autopsy studies is variable, which ranges from 4% [9] to as high as 54% [10]. The substantial difference in the incidence is hard to explain, but may partly be attributable to ethnic difference, i.e., a higher incidence of fatal intracranial hemorrhage among Japanese compared with the Caucasian population [9, 10]. Unlike sudden death, however, the incidence of intracranial hemorrhage among patients who present with witnessed OHCA has rarely been reported. This study found that at least 12 patients, consisting of 10% of total witnessed OHCA in our institution, had been causally associated with an intracranial hemorrhage, the great majority of which were non-traumatic SAH (10 patients).

All of the six intracranial hemorrhage patients who collapsed complaining of a headache were resuscitated temporarily with an average OHCA–ROSC interval of 26 min, and the cause of their cardiac arrest was identified with the use of brain CT scan. This suggests that OHCA patients who collapse complaining of a sudden headache have a high likelihood of sustaining a fatal intracranial hemorrhage. The relative ease in resuscitating them is in sharp contrast with those who collapsed without any prodromal symptoms, in whom only 53% were resuscitated. Many of those who collapse without prodromal symptoms are likely to have a cardiac cause for their cardiac arrest [11], and the higher resuscitation rate in the former may be explained by the fact that cardiac damage resulting from an intracranial hemorrhage is generally smaller compared with that of an ischemic cardiac disease. Brain CT scan obtain routinely in resuscitated OHCA patients identified another six SAH patients who collapsed without complaining of a headache. Acquisition of CT scan in such circumstance is helpful not only in clarifying the cause of OHCA, but also in estimating the degree of anoxic brain damage [12, 13].

Two of the six intracranial hemorrhage patients who collapsed complaining of a headache actually had experienced "warning" headache within 6–12 h of their OHCA, in whom earlier intervention might have saved their lives. It was unclear why they did not seek medical attention, but in hindsight, it may be that intensity of headache might not have been so severe. Moreover, only one of the four SAH patients with a headache had experienced concomitant symptoms, which might have made it difficult for them to be diagnosed accurately even if they had been seen by general practitioners before collapse.

The authors suspect that most of the ten SAH-induced OHCA patients in this series had a ruptured cerebral aneurysm or other vascular malformations, because of the presence of a dense hematoma in the basal cistern and cerebral sulci. Interestingly, more than half (6 in 10) of them had not complained of a headache before or at the time of collapse. Although it is unclear why they had not complained of any headache, it is likely that the difference in the location of, or in the amount of bleeding from, a ruptured cerebral aneurysm may be responsible: particularly, massive bleeding from a posterior fossa aneurysm may cause instantaneous respiratory arrest because of its proximity to the medullary respiratory center, with no time left for the patient to report a headache [7]. In this regard, it is understandable that both of the patients with a cerebellar ICH had complained of a worsening headache before collapse, since increase of ICH volume is usually less rapid compared with that of SAH. Another pathophysiological mechanism, i.e., hyperacute myocardial stunning and lethal ventricular arrhythmia caused by SAH-induced catecholamine surge [14–16], may also be involved in the SAH-induced cardiac arrest. Statistical comparison between the four SAH patients with, and the six SAH patients without a headache complaint were not carried out, because of the small number of patients.

There are not a few limitations in this retrospective study. First, the information of whether or when the OHCA patient had complained of a headache or other pains depends only on the witnesses' account, which is susceptible to a recollection bias. Second, the current figure of 10% as the incidence of an intracranial hemorrhage among witnessed OHCA patients may not be definitive, because of lack of autopsy: if autopsy had been performed in all patients, the incidence of intracranial hemorrhage might

have been higher. Third, instead of aneurysmal SAH, some of the ten SAH-induced OHCA patients might actually have had a pseudo-SAH: 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 [17]. Finally, the dilemma is that identifying an intracranial hemorrhage in resuscitated OHCA patients does not necessarily translate into a clinical success, as reflected by their low survival rate [3, 7, 18, 19]. The relationship between sudden headache and cerebral origin of OHCA is difficult to evaluate or prove in a prospective fashion; however, we believe that a unique insight into the subject is provided by this study, despite the aforementioned limitations.

## Conclusions

Unlike acute chest pain, headache is an uncommon prodrome of cardiac arrest: only 5% of witnessed OHCA patients complained of a headache before collapse. Sudden headache preceding collapse seems to be a sensitive indicator that the cause of OHCA is of cerebral origin. Although they are relatively easily resuscitated from cardiac arrest, their prognosis is invariably poor. In addition, approximately half of patients whose cardiac arrest results from an intracranial hemorrhage may collapse without complaining of a sudden headache, and as a consequence, the total incidence of intracranial hemorrhage causally associated with witnessed OHCA amounted to 10%. Considering the difficulty in providing a meaningful survival for that population, focus needs to be directed to avoid sudden death from a potentially treatable lesion. Public education to promote the awareness of the symptoms of potentially lethal hemorrhagic stroke may also be warranted.

**Conflict of interest** None.

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