

# Spontaneous subarachnoid hemorrhage as a differential diagnosis of pre-hospital cardiac arrest

Sohil Pothiwala

## Abstract

Spontaneous subarachnoid hemorrhage is the most common neurological disorder leading to pre-hospital cardiac arrest. ECG changes in SAH may mimic myocardial infarction or ischemia, and thus lead to delayed treatment of the primary problem. Early identification of SAH-induced cardiac arrest with the use of computed tomography scan of the brain obtained immediately after resuscitation will aid emergency physicians make further decisions. The overall prognosis of patients who are resuscitated is extremely poor. But, prompt neurosurgical referral and multidisciplinary intensive care management can improve the survival rate and the functional outcome. Thus, physicians should consider SAH as a differential diagnosis in patients presenting with pre-hospital cardiac arrest.

**Keywords:** Cardiac arrest, cardiopulmonary resuscitation, subarachnoid hemorrhage, survival

## Access this article online

Website: [www.ijccm.org](http://www.ijccm.org)

DOI: 10.4103/0972-5229.106506

## Quick Response Code:



## Introduction

Spontaneous subarachnoid hemorrhage (SAH) is the most common neurological disorder leading to pre-hospital cardiac arrest.<sup>[1]</sup> 12% of the patients die before receiving medical treatment, 40% of the hospitalized patients die within the first month after the event, and more than one-third of the survivors suffer from major neurological deficit.<sup>[2]</sup> We report a case of a 57-year-old man presenting to the ED with pre-hospital cardiac arrest secondary to SAH. A high index of suspicion is required as the clinical presentation of cardiac arrest secondary to SAH can be obscure.

## Case Report

A 57-year-old man with no prior medical history complained of giddiness on waking up in the morning. He then fell backwards on the bed and was unresponsive. When the emergency medical services arrived at the scene

15 minutes later, the initial cardiac rhythm was pulseless electrical activity (PEA). There was no bystander CPR till then, and they immediately commenced CPR and inserted laryngeal mask airway.

On arrival in the ED, the patient was still in PEA. CPR was continued, and the patient was intubated. He had an episode of ventricular tachycardia, and after a single episode of defibrillation, he regained spontaneous circulation. Serial post-resuscitation ECGs showed ST elevation in aVR and ST depression in other leads [Figure 1]. Chest x-ray was suggestive of acute pulmonary edema. Bedside cardiac ultrasound showed normal LV contractility. Arterial blood gas analysis revealed metabolic acidosis. All the blood tests including cardiac enzymes were normal. In view of persistent hypotension, he was commenced on dopamine infusion and was mechanically ventilated.

The patient underwent CT scan of the brain, which showed subarachnoid hemorrhage with early ventricular extension and evolving hydrocephalus and also loss of grey-white differentiation, cerebral edema, and sulcal effacement [Figure 2]. After discussion with the neurosurgeon, cardiologist, and intensive care physician, the patient was admitted to neurosurgical

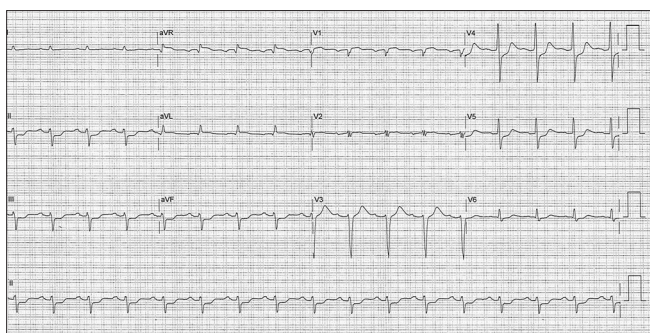
## From:

Department of Emergency Medicine, Singapore General Hospital, Singapore

## Correspondence:

Dr. Sohil Pothiwala, Department of Emergency Medicine, Singapore General Hospital, Outram Road, Singapore.

E-mail: [drsohilpothiwala@yahoo.com](mailto:drsohilpothiwala@yahoo.com)



**Figure 1:** ECG showing ST elevation in aVR and ST depression in other leads

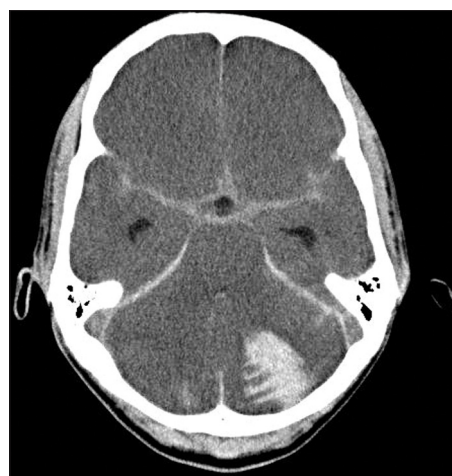
ICU for further management. Neurosurgical intervention was not considered to be indicated by the consulting neurosurgeon, and the patient died without regaining consciousness the following day.

## Discussion

SAH is a serious neurological event, which primarily occurs during adulthood in both the sexes. It carries a high mortality rate of around 35-40% despite neurosurgical treatment advances. The most common cause of SAH is rupture of an aneurysm in a blood vessel in the brain, but can also occur due to arterio-venous malformation or a traumatic event.

About 10% of the patients with spontaneous SAH present to the emergency department (ED) with out-of-hospital cardiac arrest.<sup>[3]</sup> Women are affected more frequently than men, and lower incidence of associated co-morbidities, with only a quarter of patients having cardiovascular risk factors. The mechanism of cardiac arrest secondary to SAH has been attributed to a sudden increase in intracranial pressure caused by massive SAH, which results in brainstem herniation leading to respiratory arrest. SAH also leads to sympathetic hyperstimulation and release of catecholamines, resulting in lethal cardiac arrhythmias and subsequent cardiac arrest.<sup>[4]</sup>

ECG changes, thought to be neurogenically mediated, are often pronounced in patients with SAH. ST-segment elevation or depression and abnormal T wave morphologies have been noted. The pattern of large inverted T waves along with prolonged QTc is commonly known as "cerebral" T wave. SAH also predisposes the patients to all cardiac arrhythmias, especially malignant ventricular arrhythmias like ventricular tachycardia, ventricular fibrillation, and torsades de pointes.<sup>[5]</sup> ECG in our case showed ST segment elevation in the aVR lead, leading to suspicion of acute myocardial infarction secondary to LM obstruction as a cause of cardiac arrest. Thus, ECG changes in SAH may mimic



**Figure 2:** Computed Tomography of brain showing acute subarachnoid hemorrhage with cerebral edema, sulcal effacement, and loss of grey-white differentiation

myocardial infarction or ischemia, and thus lead to delayed treatment of the primary problem.

PEA is found to be the initial cardiac rhythm in 44% of the SAH patients with cardiac arrest, with 48% having asystole and only 8% having ventricular fibrillation.<sup>[5]</sup> Thus, SAH should be considered as a differential diagnosis in pre-hospital cardiac arrest patients with PEA as the presenting rhythm.

A history of sudden onset of severe headache prior to the arrest is the key, but it has been reported that half of these patients lose consciousness without any prodromal symptoms.<sup>[6]</sup> Early identification of SAH-induced cardiac arrest with the use of computed tomography (CT) scan of the brain obtained immediately after resuscitation will aid emergency physicians make further decisions.<sup>[7]</sup> Prompt neurosurgical intervention is important for good neurological outcome. Moreover, post-resuscitation therapeutic hypothermia is unwarranted in patients with SAH as it would increase the bleeding tendency, which could prove to be detrimental.<sup>[8]</sup>

Patients with pre-hospital cardiac arrest due to spontaneous SAH have an extremely poor prognosis.<sup>[9]</sup> This can be attributed to the hypoxic brain damage secondary to cardiac arrest, in addition to raised intracranial pressure due to SAH. There have been reports of few cases who have survived the event, but the prognosis is poor. Some patients had complete neurological recovery, but most of them resulted in brain death, vegetative state or severe brain damage; and the chance of long term survival is small.<sup>[4,10-12]</sup> Patients who receive bystander CPR soon after arrest, those with early return of spontaneous circulation, early diagnosis and appropriate therapy have improved survival.<sup>[12]</sup>

In conclusion, physicians should consider SAH as a differential diagnosis in patients presenting with pre-hospital cardiac arrest, especially in the absence of risk factors. The overall prognosis of patients who are resuscitated is extremely poor with a low survival rate. But, prompt neurosurgical referral and multidisciplinary intensive care management can improve the survival rate and the functional outcome.

## References

1. Bonita R, Thomson S. Subarachnoid hemorrhage: Epidemiology, diagnosis, management and outcome. *Stroke* 1985;16:591-4.
2. Johnston SD, Robinson TJ. Subarachnoid haemorrhage: Difficulties in diagnosis and treatment. *Postgrad Med J* 1998;74:743-4.
3. Kitahara T, Masuda T, Soma S. The etiology of sudden cardiopulmonary arrest in subarachnoid hemorrhage. *No Shinkei Geka* 1996;21:781-6. In Japanese.
4. Inamasu J, Saito R, Nakamura Y, Ichikizaki K, Suga S, Kawase T, *et al.* Survival of a subarachnoid hemorrhage patient who presented with prehospital cardiopulmonary arrest: Case report and review of the literature. *Resuscitation* 2001;51:207-11.
5. Sommargren CE. Electrocardiographic abnormalities in patients with subarachnoid hemorrhage. *Am J Crit Care* 2002;11:48-56.
6. Kurkeiyan I, Meron G, Sterz F, Domanovits H, Tobler K, Laggner AN, *et al.* Spontaneous subarachnoid hemorrhage as a cause of out-of-hospital cardiac arrest: True or not? *Resuscitation* 2001;51:27-32.
7. Inamasu J, Miyatake S, Tomioka H, Suzuki M, Nakatsukasa M, Maeda N, *et al.* Subarachnoid hemorrhage as a cause of out-of-hospital cardiac arrest: A prospective computed tomography study. *Resuscitation* 2009;80:977-80.
8. Wolberg AS, Meng ZH, Monroe DM III, Hoffman M. A systemic evaluation of the effect of temperature on coagulation enzyme activity and platelet function. *J Trauma* 2004;56:1221-8.
9. Lassepas MR, Ahmed A. Cardiorespiratory arrest in aneurysmal subarachnoid hemorrhage. *J Stroke Cerebrovasc Dis* 1991;1:49-56.
10. Suzuki H, Sakurai M, Fujimoto M, Tsuchiya T, Sakaida H, Taki W. Complete recovery from aneurysmal subarachnoid hemorrhage associated with out-of-hospital cardiopulmonary arrest. *Eur J Emerg Med* 2010;17:42-4.
11. Toussaint LG 3<sup>rd</sup>, Friedman JA, Wijdicks EF, Piepgras DG, Pichelmann MA, McIver JI, *et al.* Survival of cardiac arrest after aneurysmal subarachnoid hemorrhage. *Neurosurgery* 2005;57:25-31.
12. Shapiro S. Management of subarachnoid hemorrhage patients who presented with respiratory arrest resuscitated with bystander CPR. *Stroke* 1996;27:1780-2.

**How to cite this article:** Pothiwala S. Spontaneous subarachnoid hemorrhage as a differential diagnosis of pre-hospital cardiac arrest. *Indian J Crit Care Med* 2012;16:216-8.

**Source of Support:** Nil, **Conflict of Interest:** None declared.

## New features on the journal's website

### Optimized content for mobile and hand-held devices

HTML pages have been optimized for mobile and other hand-held devices (such as iPad, Kindle, iPod) for faster browsing speed.

Click on [**Mobile Full text**] from Table of Contents page.

This is simple HTML version for faster download on mobiles (if viewed on desktop, it will be automatically redirected to full HTML version)

### E-Pub for hand-held devices

EPUB is an open e-book standard recommended by The International Digital Publishing Forum which is designed for reflowable content i.e. the text display can be optimized for a particular display device.

Click on [**EPub**] from Table of Contents page.

There are various e-Pub readers such as for Windows: Digital Editions, OS X: Calibre/Bookworm, iPhone/iPod Touch/iPad: Stanza, and Linux: Calibre/Bookworm.

### E-Book for desktop

One can also see the entire issue as printed here in a 'flip book' version on desktops.

Links are available from Current Issue as well as Archives pages.

Click on  View as eBook