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Case Report

Transient left ventricular apical ballooning in a patient with cardiac arrest after subarachnoid hemorrhage

Wataru Mitsuma (MD)[a,b,*], Masahiro Ito (MD)[a], Makoto Tomita (MD)[a], Komei Tanaka (MD)[a], Junichiro James Kazama (MD)[b], Tadayuki Honda (MD)[b], Satoru Hirono (MD)[a], Hiroshi Endoh (MD)[b], Makoto Kodama (MD)[a], Yoshifusa Aizawa (MD FJCC)[a]

[a] *Division of Cardiology, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan*
[b] *Advanced Disaster Medical & Emergency Critical Care Center, Niigata University Graduate School of Medical and Dental Hospital, Niigata, Japan*

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KEYWORDS
Subarachnoid hemorrhage;
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Summary Subarachnoid hemorrhage (SAH) often accompanies cardiac abnormalities. Sudden cardiac arrest is also known to occur after SAH. A 32-year-old woman was admitted to our hospital because of cardiac arrest immediately after the onset of SAH. Return of spontaneous circulation was obtained by conventional advanced cardiovascular life support. After resuscitation, her echocardiogram showed left ventricular apical ballooning, which improved within 7 days. This is the first report presenting both sudden cardiac arrest and transient left ventricular apical ballooning after SAH.

Introduction

Spontaneous subarachnoid hemorrhage (SAH) often accompanies electrocardiographic and/or echocardiographic abnormalities [1]. These cardiac findings are more likely to occur with increasing neurological deficits [1]. On the other hand, cardiac arrest is also known to occur after SAH [2,3]. About 4% of patients with SAH experience cardiac arrest immediately after the onset [2]. However, the mechanism of cardiac arrest after SAH remains uncertain. One hypothesis is that SAH leads to cardiac arrest directly. The other hypothesis is that myocardial damage is elicited by SAH, and then leads to cardiac arrest. Here, we describe a woman with cardiac arrest after SAH, who presented with transient left ventricular apical ballooning, so-called Takotsubo cardiomyopathy, after resuscitation.

* Corresponding author at: Division of Cardiology, Niigata University Graduate School of Medical and Dental Sciences, 1-754 Asahimachi dori, Chuoku, Niigata 951-8510, Japan.
Tel.: +81 25 227 2185; fax: +81 25 227 0774.
E-mail addresses: mwataru@med.niigata-u.ac.jp, mwataru@hotmail.com (W. Mitsuma).

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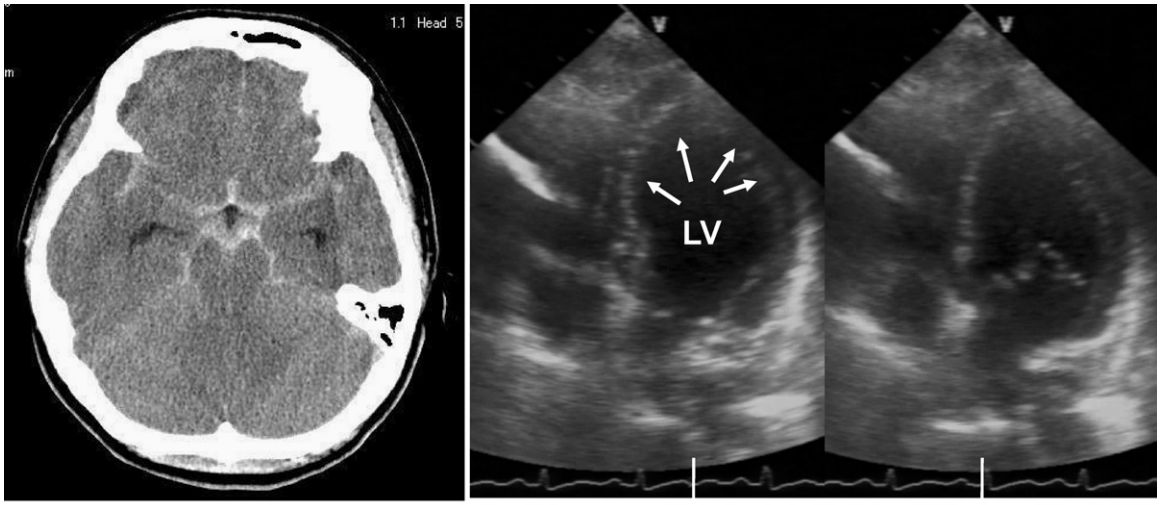

Figure 1 Computed tomography (A), and echocardiogram of end systole (B) and end diastole (C) of the presented patient. Extensive subarachnoid hemorrhage with bilateral intraventricular hemorrhage was observed on computed tomography (A). Akinesis of the left ventricular apex and mid segments (white arrows) was found on the apical 4-chamber view after resuscitation. LV, left ventricle.

Case report

A 32-year-old woman, without any history of cardiovascular disease, was transferred to our emergency department because of cardiac arrest. Her initial cardiac rhythm, recorded by emergency medical service, showed pulseless electrical activity. Twenty minutes before arrival, she suddenly collapsed with a moan in her house, and her friend started cardiopulmonary resuscitation. Although return of spontaneous circulation (ROSC) was obtained following a 1 mg epinephrine bolus at the emergency department, she was still unconscious with anisocoria. A computed tomography of her head showed extensive SAH (Fig. 1A), and the severity of her clinical presentation was grade 5, as quantified by the World Federation of Neurological Surgeons guidelines. Her 12-lead electrocardiogram after ROSC showed sinus tachycardia with mild ST-segment depression on II, III, aVF, V3, V4, and V5 and ST-segment elevation in aVL (Fig. 2). Akinesis of the left ventricular apex and mid segments with normal contraction of basal segments was found on echocardiography, and the ejection fraction measured by modified Simpson's method was 19% (Fig. 1B and C). These left ventricular wall motion abnormalities resembled Takotsubo cardiomyopathy and could not be explained by a single epicardial coronary artery obstruction. Her chest X-ray showed bilateral pulmonary edema, and creatine kinase on admission was 71 IU/l (normal range <163 IU/l). Serial electrocardiographic and echocardiographic examinations were performed. Although poor R-wave progression in the precordial leads, ST-segment elevation, and T-wave inversion with QT prolongation (corrected QT interval 480 ms) were found without creatine kinase elevation on day 3, all electrocardiographic abnormalities normalized on day 9 (Fig. 2). Takotsubo-like wall motion abnormalities improved with disappearance of pulmonary edema on day 7. However, she died of brain death on day 10.

Discussion

In this case report, we describe a patient with cardiac arrest after SAH showing transient left ventricular apical ballooning, or so-called Takotsubo cardiomyopathy. Several cardiac abnormalities, including cardiac arrest, were observed after the onset of SAH [1—3]. Electrocardiographic findings, such as ST-T elevation/depression, T-wave inversion, QT prolongation, and supraventricular/ventricular arrhythmias, were found in more than 50% of SAH patients [1]. Global or segmental left ventricular dysfunction was observed on left ventriculography and/or echocardiography [4]. These electrocardiographic and echocardiographic abnormalities were more likely to occur with increasing neurological deficits, and 58% and 52% of patients with severe neurological deficits had electrocardiographic and echocardiographic abnormalities, respectively [1]. However, echocardiographic abnormalities do not always accompany electrocardiographic ones [1]. In recent reports, Takotsubo-like wall motion abnormalities were observed after SAH [5,6]. These cardiac findings were a transient phenomenon and irrelevant to coronary artery disease [4]. Although the pathophysiology of these abnormalities remains uncertain, the catecholamine surge following brainstem damage has been considered to produce neurogenic myocardial stunning and electrocardiographic abnormalities [6,7]. On the other hand, cardiac arrest is also known to occur after SAH [8]. SAH is found to be present in 4—10% of all out-of-hospital cardiac arrests, and about 4% of SAH patients experience cardiac arrest after the onset [2,3,9]. Direct catecholamine cardiotoxicity, same as left cardiac dysfunction after SAH, has been considered one of the mechanisms leading to cardiac arrest, while respiratory suppression by a sudden increase in intracranial pressure with brainstem herniation may also lead to cardiac arrest in some patients [2,6]. However, the association between cardiac