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Successful Coil Embolization Using Percutaneous Cardiopulmonary Support in a Patient with Refractory Out-of-hospital Cardiac Arrest Caused by Aneurysmal Subarachnoid Hemorrhage

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Abstract

In recent years, extracorporeal cardiopulmonary resuscitation (ECPR) has been reported to be an effective alternative to conventional CPR for treating patients with reversible causes of cardiac arrest. Nevertheless, the definite indication for ECPR and also surgical interventions during ECPR treatment have not been established, especially in patients with out-of-hospital cardiac arrest (OHCA) caused by subarachnoid hemorrhage (SAH). We treated a comatose 50-year-old woman with refractory cardiac arrest due to aneurysmal SAH-induced takotsubo cardiomyopathy (TCM). The initial cardiac rhythm was ventricular fibrillation. This is the first case report on coil embolization being successfully performed on a patient undergoing ECPR and therapeutic hypothermia (TH) while the patient was still in cardiac arrest, which resulted in complete social rehabilitation. Moreover, the success of this treatment suggests that ECPR and endovascular therapy should be considered for highly selected patients when cardiopulmonary and neurological functions are potentially reversible even in the setting of SAH.

Keywords: subarachnoid hemorrhage, coil embolization, out-of-hospital cardiac arrest, percutaneous cardiopulmonary support, extracorporeal cardiopulmonary resuscitation

Introduction

Aneurysmal subarachnoid hemorrhage (SAH) is known to be one of the major causes of out-of-hospital cardiac arrest (OHCA). SAH accounts for 2%–11% of diseases that cause resuscitated OHCA.[1–6] The prognosis of patients with SAH associated with OHCA is extremely poor, ranging from 0% to 9% surviving, despite significant advances in basic and advanced life support.[1–7] Thus, almost all patients with SAH who are undergoing cardiopulmonary resuscitation (CPR) for refractory cardiac arrest at hospital arrival have been deemed futile.[1,3,5] Meanwhile, acute coronary syndrome (ACS) is the most common cause of OHCA, and extracorporeal CPR (ECPR) for patients with refractory cardiac arrest has been performed as an alternative to conventional CPR.[8,9] Recent resuscitation guidelines state that ECPR may be considered for selected patients for whom the suspected etiology of the cardiac arrest is potentially reversible.[10,11] ECPR has been considered to provide a bridge to the subsequent diagnosis and treatment of the underlying cause of cardiac arrest. However, especially in patients with SAH, the definite indication for ECPR and also surgical interventions that undergo ECPR treatment have not been well established.[10,11] The final decision to perform ECPR should be made on a case-by-case basis depending on the severity of the neurological cause and consequence of cardiac arrest. Considering the

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Table 1 CPR record from onset to the initiation of extracorporeal CPR

Response Time (min)	Event	Duration of CPR (min)	Duration of ROSC (min)
0	Sudden cardiac arrest. Bystander CPR was immediately performed	14	
5	Ambulance was called		
9	Ambulance arrived. Shocks were implemented for ventricular fibrillation		
14	ROSC occurred		36
40	Cardiac re-arrest (PEA). Epinephrine (1 mg) was administered	2	
42	ROSC occurred		18
60	Cardiac re-arrest (PEA). Epinephrine (1 mg) was administered	5	
65	ROSC occurred 1 min after arrival at hospital		1
66	Cardiac re-arrest (PEA). Epinephrine (1 mg) was administered	11	
68	ROSC did not occur. Decision was made to initiate extracorporeal CPR		
77	Extracorporeal CPR and therapeutic brain hypothermia were initiated		

CPR: cardiopulmonary resuscitation, PEA: pulseless electrical activity, ROSC: return of spontaneous circulation.

concurrence of an aneurysm rupture and sudden cardiac arrest, early aggressive cardiopulmonary and concomitant cerebral resuscitation, combined with early aneurysm repair may be necessary to maximize the chance of a meaningful survival.[12,13)]

We treated a critically ill patient with refractory cardiac arrest requiring ECPR, although the cause was determined as SAH due to cerebral aneurysm rupture. Therapeutic hypothermia (TH) and coil embolization were immediately performed even under conditions of ECPR while the patient was still in cardiac arrest; the patient dramatically recovered without any neurological sequelae. We hereby report such a case, as this strategy could become an option for treating severe SAH in the future.

Case Material

A 50-year-old previously healthy woman suddenly lost consciousness while on the toilet at home. Her husband, a firefighter, immediately performed CPR and called an ambulance. When the ambulance crew arrived, Glasgow coma scale score was 3, both pupils were 4.0 mm in diameter, and cardiac rhythm was ventricular fibrillation (VF). Therefore, shock resuscitation was implemented. The patient's heartbeat resumed in 5 min but she suffered repeated cardiac arrest during transport to the hospital and was in cardiac arrest on arrival at the hospital. Immediately after arrival, her heartbeat resumed but this was again followed by cardiac arrest. As the patient continued to repeatedly experience cardiac arrest, percutaneous cardiopulmonary support (PCPS) was initiated. Pupil contraction and spontaneous respiration subsequently resumed 77 min after onset (Table 1).

Fig. 1 Head CT scan demonstrating diffuse thin SAH with slight intraventriclar hemorrhage. CT: computed tomography.

As the initial cardiac rhythm was VF, ACS was suspected and coronary angiography (CAG) was performed. However, no coronary stenosis or occlusion was observed. Although the heart exhibited almost complete apical and mid-ventricular akinesis during systole, a slight amount of wall motion was observed in the left ventricle base on left ventriculography, and takotsubo cardiomyopathy (TCM) was suspected. Head computed tomography (CT)

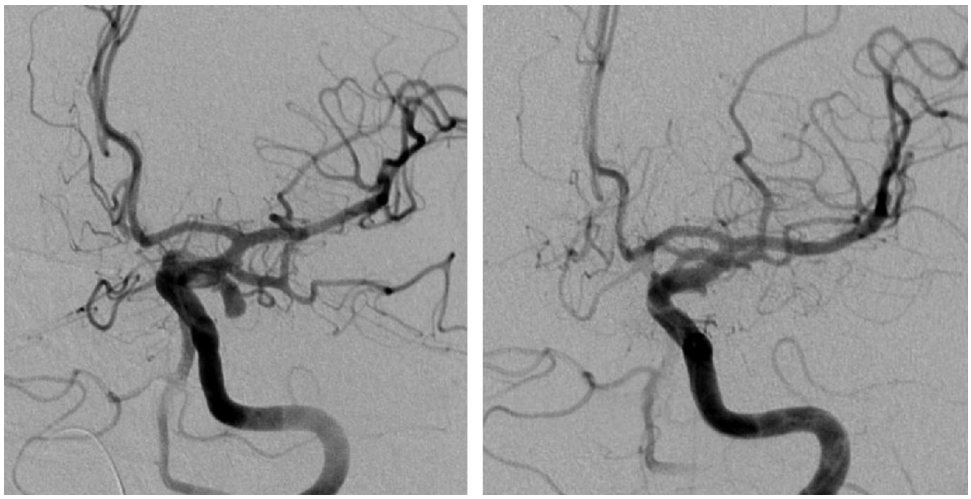

Fig. 2 Digital subtraction angiography. Left: Preoperative angiogram, left anterior oblique projection, showing a left internal carotid artery–posterior communicating artery aneurysm. Right: Postembolization angiogram, left anterior oblique projection, demonstrating complete occlusion of the aneurysm.

performed after CAG showed diffuse thin SAH with slight intraventriclar extension (Fig. 1). Subsequent three-dimensional CT angiography revealed a left internal carotid artery–posterior communicating artery aneurysm.

Although the patient remained in a comatose state, brainstem function improved, with pupil contraction, light reflex, and spontaneous respiration. Therefore, there was no reason to consider abandoning resuscitation. Because the use of anticoagulants during PCPS increases the risk of re-rupture, coil embolization was performed 5 h after the patient arrived at the hospital to completely occlude the aneurysm as an urgent measure to prevent re-rupture (Fig. 2).

The patient remained in cardiac arrest following surgery and was treated with PCPS with a target mean arterial pressure of 65 mmHg. For TH, the target of 34°C was reached 20 min after initiating PCPS and was maintained for 24 h. Nafamostat mesylate was continuously administered as an anticoagulant to maintain PCPS. CT performed on hospital day 2 revealed no presence of cerebral ischemia/edema, hemorrhagic complications, or hydrocephalus, and rewarming was implemented after TH at a speed of 0.1°C/h for 24 h. The patient's cardiac function rapidly improved and, on hospital day 3, she was successfully weaned from PCPS after 53 h since its initiation. She did not develop delayed ischemic neurological deficit due to vasospasm and rehabilitation was proactively continued. On hospital day 58, the patient was discharged home with a modified Rankin scale of 0 and no neurological sequelae.

Discussion

TCM, also referred to as neurogenic stress cardiomyopathy, is usually triggered by emotional or physical stress such as SAH predominantly in postmenopausal women.[14–16] The reported incidence of TCM in patients with SAH ranges from 2.2% to 15%.[14–16] Sudden cardiac arrest is an increasingly recognized presentation of TCM.[5,17–22] However, during ongoing CPR in the emergency department, accurately differentiating between comatose patients following SAH with TCM and ACS as a cause of OHCA is often challenging, especially those presenting with sudden collapse with no any prodromal symptoms or comorbidities.[1,20,22,23] Moreover, the initial cardiac rhythm of VF can be interpreted as an arrest of cardiac etiology because the cardiac rhythm was VF in only 0%–7.4% of cardiac arrest cases caused by SAH.[5,10,18,23] In this setting, the prompt decision to initiate ECPR for our patient with refractory cardiac arrest of presumed cardiac etiology was made based on favorable prognostic factors including younger age, shockable initial cardiac rhythm, witnessed bystander CPR, short no-flow time, and transitory return of spontaneous circulation (ROSC) events.[9–11]

Limited clinical data regarding the characteristics of favorable survivors from OHCA caused by SAH are available. A nationwide population-based study in Japan showed that in stroke-related OHCA cases, having a younger age, witness, and shockable initial documented rhythm were associated with improved outcomes.[2] Moreover, only five cases with good neurological outcomes have been reported with detailed descriptions of clinical course.[4,20,24–26]

Interestingly, these cases with favorable prognoses also had the common characteristics such as younger age, shockable initial cardiac rhythm, or extremely short no-flow time, including less severe SAH on initial CT scans.[4,20,24,26] However, overall CPR durations before ECPR in our patient were relatively longer than other cases. Nevertheless, repeated ROSC episodes with higher-quality CPR might be considered for ECPR even after prolonged periods of CPR.[27] The occurrence of any ROSC event during CPR, even if short or unstable, can ensure a markedly higher tissue perfusion pressure than that in chest compressions.[27]

The pathophysiological mechanisms of SAH-induced TCM have not been clearly elucidated.[16,28] The most widely accepted mechanism is an excessive release of catecholamines, leading to transient left ventricular dysfunction without the evidence of obstructive coronary artery disease.[16,28] TCM typically resolves in a few days or weeks, and its management is supportive.[14,16,29] However, life-threatening cardiac complications including ventricular arrhythmias, cardiogenic shock, thrombus formation, and even sudden cardiac arrest may develop in the acute phase.[30] However, despite its severity of cardiac dysfunction, aggressive intervention is justified in patients with severely impaired cardiac function due to SAH-induced TCM because the cardiac impairment is almost invariably transient.[29] ECPR using PCPS could serve as a bridge to recovery in severe TCM.[31,32] In a recent systemic review and meta-analysis of the available literature regarding the use of mechanical circulatory support in TCM patients with cardiogenic shock including cardiac arrest of 22.2%, median time on mechanical circulatory support was 3 days with an overall survival of 94.6%.[32] The prognosis might be dependent on the potentially possible reversibility of the primary hemorrhagic and secondary ischemic brain damages if the patient is successfully resuscitated from cardiac arrest. Even less severe SAH can trigger fulminant TCM, as in our patient.[15,16,20] Accordingly, non-resumption of the patient's heartbeat after starting PCPS is not considered a reason for abandoning further aggressive treatment.

PCPS is a rapidly deployable and easily transportable temporary assist system, providing sufficient oxygenated and decarboxylated blood flow to vital organs including the brain. Rapid hemodynamic stability provided by PCPS also facilitated a multidisciplinary approach to neurocritical and concomitant post-cardiac arrest care including TH.[8,33] In recent years, there have been reports on endovascular cooling methods for TH using extracorporeal circulation with the aim of a more rapid and precise cooling.[34] Due to cooling by the pump oxygenator, the bladder temperature reached 34°C at 20 min after PCPS was initiated. Fortunately, her circulation flow was stable soon after PCPS was initiated, and the mean arterial pressure could be maintained at the target level (BP ≥ 65 mmHg). Early TH receiving ECPR might be a potential neuroprotective strategy against post-cardiac arrest brain injury even in cases of refractory OHCA associated with SAH.[33]

Aneurysm rebleeding is associated with very high mortality and poor prognosis for functional recovery in survivors.[35,36] The risk of rebleeding is maximal in the first 2–12 h, with reported rates of occurrence between 4% and 13.6% within the first 24 h.[35] This early rebleeding is more common in patients with poor-grade SAH.[35,36] Additionally, the use of systemic anticoagulants while the patient is being treated with PCPS makes the patient prone to bleeding. Hypothermia can also impair blood coagulation system and induces platelet dysfunction at a temperature below 35°C.[37] Thus, our patient might be considered to be at higher risk for rebleeding. Therefore, aneurysm should be treated as early as feasible to reduce the risk of rebleeding after SAH.[35] As improvement in brainstem function was observed during ECPR, the patient was proactively judged to be eligible for securing aneurysm. Minimally invasive endovascular coiling is a much better indication than invasive surgical clipping in these complex clinical situations.[29,38] Ultra-early endovascular coiling was technically safe and feasible with no particular perioperative complications while the patient hemodynamically stable with PCPS.

Conclusions

To our knowledge, there has been no reported case on endovascular coiling being successfully performed on a comatose patient undergoing ECPR and TH while still in cardiac arrest caused by SAH. Furthermore, this early aggressive cardiopulmonary and cerebral resuscitation with beneficial neurological outcome suggests that ECPR and endovascular therapy should be considered for highly selected patients when the etiology of cardiac arrest is potentially reversible even in the setting of SAH.

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Conflicts of Interest Disclosure

The authors report no conflict of interest to declare in this paper.

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