Recovery from Out-of-Hospital Cardiopulmonary Arrest Due to Type A Acute Aortic Dissection: A Case Report

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Abstract

Case: A 55-year-old man without past medical histories suffering from back pain for two weeks was successfully resuscitated from an 8-min cardiopulmonary arrest (CPA) and was brought to our hospital by ambulance. Computed tomography demonstrated type A acute aortic dissection (AAAD) with brachiocephalic artery occlusion. After admission, Glasgow Coma Scale score improved to E1VTM4, and voluntary movement was noted only in the right limbs.

Outcome: The patient underwent emergency grafting of the ascending aorta and innominate artery under deep hypothermic circulatory arrest. After surgery, the patient recovered with mild disorientation and left hemiplegia. Magnetic resonance imaging of the head revealed no large infarction but revealed multiple acute ischemic changes. One year later, the patient demonstrated independent walk and successfully returned to work life.

Conclusions: Immediate resuscitation and surgery resulted in good recovery from CPA after AAAD.

Keywords

Aortic Branch Artery Occlusion, Cerebral Ischemia, Malperfusion, Prognosis, Surgical Indication

1. Introduction

Type A acute aortic dissection (AAAD) requires emergency surgery as definitive treatment with a mortality rate of more than 40% after onset [1]. Poor prognosis has been reported with cardiopulmonary arrest (CPA) [2], aortic branch occlusion [3] [4] [5]
[6], severe consciousness disturbance [4], and cerebral vascular disorders (CVD) [4] in the preoperative period. We report a case of AAAD, requiring emergency surgery and recovering after out-of-hospital CPA with good neurological outcome.

2. Case Report

A 55-year-old man without past medical histories suffering from back pain for 2 weeks was brought to our hospital by ambulance because of dyspnea at his office. Following were the patient’s vital signs: Glasgow Coma Scale (GCS) score, 15; respiratory rate (RR), 36/min; pulse, 60/min; blood pressure (BP), 110/54 mmHg; and SpO₂, 60% (room air) at the scene (Normal vital sign ranges are; GCS 15, RR 12 - 18/min, pulse 60 - 100/min, BP 120 - 90/80 - 60 mmHg, SpO₂ 96% - 100% (room air)). However, vital signs deteriorated 3 min after ambulance arrival (GCS score, 3 and BP, immeasurable). Breathing ceased and pulseless electrical activity (PEA) was observed 9 min after ambulance arrival, and cardiopulmonary resuscitation (CPR) by chest compression and mask ventilation were started immediately by the emergency medical service (EMS). Seventeen minutes after ambulance arrival, the patient was resuscitated successfully with return of spontaneous circulation (ROSC) and respiration during transport and was brought to our emergency room (ER) 23 min after ambulance arrival. The patient had widened mediastinum, severe pulmonary edema, bilateral pulmonary effusion on chest X-ray (Figure 1(a)), severe aortic regurgitation and no pericardial effusion on echocardiography. Whole-body computed tomography (CT) demonstrated no obvious intracranial hemorrhage (Figure 1(b)) but AAAD from the ascending aorta to the bilateral common iliac arteries with brachiocephalic and right carotid artery occlusion by false lumen thrombosis (Figures 1(c)-(f)).

Forty minutes after ER arrival, GCS score improved to E1VTM4, and voluntary movement occurred only in the right limbs. Because a good prognosis seemed possible, emergency surgery for AAAD was performed, despite the predisposed risks. Under deep hypothermic circulatory arrest at rectal temperature of 20°C, emergency grafting of the ascending aorta and innominate artery was performed 3 h 48 min after ER arrival. For revascularization of the true lumen in the right carotid artery, the distal innominate artery at the bifurcation of the right subclavian and right common carotid arteries was transected and the thrombus was extracted from the false lumen of the proximal carotid artery. The AAAD did not extend to the root of the left carotid and bilateral coronary arteries. On postoperative day 3, after discontinuing all sedatives, the patient was extubated with restoration of consciousness (GCS score, E4V4M6) and left hemiplegia. There were no hemorrhagic intracranial lesions on head CT on postoperative day 5, and high-intensity spots and local areas were found in the anterior parietal lobes on head diffusion-weighted magnetic resonance imaging (MRI) on postoperative day 11 (Figure 2(a), Figure 2(b)). Large infarction was absent, but multiple acute ischemic changes were noted after the occlusion of the right carotid artery. The patient was able to walk for 30 meter with a walking-stick and mild disorientation. He was transferred to a rehabilitation hospital on postoperative day 25. The cerebral and overall
Figure 1. Preoperative radiological examinations. Chest X-ray and enhanced computed tomography (CT) scans showed widened mediastinum (black triangles), severe pulmonary edema (a), and bilateral pulmonary effusion (white triangles). There was no evidence of intracranial hemorrhage or large cerebral infarction on plain head CT (b). Type A acute aortic dissection from the ascending aorta to bilateral common iliac arteries with the brachiocephalic and right carotid artery occlusion (white arrow) by false lumen thrombosis on enhanced CT scans (c)-(f).

Figure 2. Postoperative diffusion-weighted magnetic resonance imaging demonstrates high-intensity spots (a), (b) and areas (b) in both anterior parietal lobes on postoperative day 11. There was no large infarction, but multiple acute ischemic changes were noted after right carotid artery occlusion associated with type A acute aortic dissection.

performance category (CPC and OPC) was grade 2 at hospital discharge. One year later, he was able to walk independently, supported by a walking-stick, and return to work (OPC1/CPC1).

3. Discussion

There are two important points in this case. First, the AAAD patient was quickly resuscitated from an 8-min cardiac arrest probably due to severe hypoxia and pulmonary edema. Next, immediate surgery after ROSC resulted in good recovery of AAAD because
large cerebral infarction had been avoided.

Poor prognostic factors after surgical treatment of AAAD include cardiopulmonary arrest [2], severe disturbance of consciousness [4], and cerebrovascular disorder [4] in the preoperative period. Emergency surgery in patients with those risk factors usually is avoided because of high mortality and morbidity. One reason, avoiding surgery, is that anticoagulant use during intraoperative extracorporeal circulation may promote hemorrhagic cerebral infarction or posttraumatic intracranial hemorrhage [5]. Regarding our patient, despite three preoperative poor prognostic factors, the recovery suggests that the effects of these factors remained minimal.

Regarding the timing of surgery for AAAD, even in patients with CVD, the postoperative outcome was not affected by early operation [4] [7]. Moreover, some cases fully recovered by immediate surgery within 3 hour after ER arrival [5]. Our patient showed left hemiplegia perioperatively, which was not aggravated by immediate surgery, revealing minimal cerebral ischemic changes on MRI. Considering the duration of treatment by tissue-plasminogen activator for acute ischemic stroke, anticoagulant use by intraoperative extracorporeal circulation may not be harmful during immediate surgery for AAAD complicated by cerebral malperfusion.

Few cases of out-of-hospital CPA after AAAD have been reported worldwide except in Japan [8] [9] [10]. Four patients recovered from in- and out-of-hospital CPA after AAAD by emergency surgery (Table 1) [8] [9] [10]. Three patients received immediate CPR from healthcare providers, whereas one patient was given CPR by a swimming instructor.

Table 1. Past cases recovered after cardiopulmonary arrest because of type A acute aortic dissection.

<table>
<thead>
<tr>
<th>No</th>
<th>Author</th>
<th>Year</th>
<th>Age (years)</th>
<th>Sex</th>
<th>CA Site</th>
<th>Witness and Bystander CPR</th>
<th>Initial Rhythm after CA</th>
<th>CA Cause</th>
<th>CA Time</th>
<th>Post-CA Neurological Signs</th>
<th>Time to OR</th>
<th>Long Term Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Kurimoto</td>
<td>2002</td>
<td>71</td>
<td>Female</td>
<td>Prehospital</td>
<td>By a swimming instructor</td>
<td>NA</td>
<td>Cardiac tamponade</td>
<td>45 min</td>
<td>Coma, involuntary limb movement in response to noxious stimuli</td>
<td>NA</td>
<td>Recovered</td>
</tr>
<tr>
<td>2</td>
<td>Saito</td>
<td>2007</td>
<td>80</td>
<td>Male</td>
<td>ICU</td>
<td>By a cardiac surgeon</td>
<td>PEA</td>
<td>Cardiac tamponade</td>
<td>NA</td>
<td>Voluntary limb movement in response to verbal stimuli</td>
<td>NA</td>
<td>Recovered with left hemiplegia</td>
</tr>
<tr>
<td>3</td>
<td>Fujii</td>
<td>2014</td>
<td>70</td>
<td>Male</td>
<td>ER</td>
<td>By an ER physician</td>
<td>PEA</td>
<td>Cardiac tamponade</td>
<td>2 min</td>
<td>GCS M6</td>
<td>15 h</td>
<td>Partially recovered in the perioperative period but deceased because of pneumonia</td>
</tr>
<tr>
<td>4</td>
<td>Our case</td>
<td>2015</td>
<td>50</td>
<td>Male</td>
<td>Prehospital</td>
<td>By an EMS personnel</td>
<td>PEA</td>
<td>Unknown (hypoxia or transient aortic occlusion by flap)</td>
<td>8 min</td>
<td>GCS E1VTM4, left hemiplegia</td>
<td>3.5 h</td>
<td>Recovered</td>
</tr>
</tbody>
</table>

Abbreviations: CA: cardiac arrest, CPR: cardiopulmonary resuscitation, EMS: emergency medical system, ER: emergency room, ICU: intensive care unit, GCS: Glasgow coma scale, NA: not available, OR: operating room, PEA: pulseless electrical activities.
instructor who was well-trained in CPR procedures and also by healthcare providers. Thus, a witness, prompt high-quality CPR, and relieving CPA cause, including resuscitative pericardiocentesis if necessary, are considered essential for recovery from CPA after AAAD. A short duration until ROSC generally is favorable for predicting neurological outcome, but surprisingly, the duration varies between 2 and 45 min in the prior reports (Table 1). Therefore, the time window for favorable outcomes depends on the cause of CPA and subsequent resuscitative procedures after AAAD. After all, all patients had improved consciousness and response or limb movement to noxious stimuli within several hours after ROSC. Several factors, such as a witness, immediate high-quality CPR, quick removal of CPA cause, and neurologically-recovering symptoms in the early phase after ROSC, are necessary for recovery from CPA after AAAD. Conclusively, immediate resuscitation and surgery may improve an outcome of ROSC patients after AAAD with the several factors above.

4. Conclusion

We report a good recovery from CPA after AAAD by immediate resuscitation and surgery.

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References


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