Case Report

Treatment of a patient with acute aortic dissection using extracorporeal cardiopulmonary resuscitation after an out-of-hospital cardiac arrest: a case report

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Case: Circulatory support using veno-arterial extracorporeal membrane oxygenation for aortic disease is conventionally contraindicated. In this case, a 66-year-old man experienced cardiopulmonary arrest caused by acute aortic dissection. When exercising in the gym, he experienced chest discomfort, so the staff immediately called an ambulance. While in the ambulance, he experienced cardiopulmonary arrest. His initial electrocardiogram showed ventricular fibrillation. At the emergency department, we immediately performed extracorporeal cardiopulmonary resuscitation. We suspected acute coronary syndrome, so coronary angiography was carried out. Enlargement of ascending aorta was noted. Whole-body enhanced computed tomography was subsequently performed, leading to a final diagnosis of acute aortic dissection.

Outcome: Emergency ascending aorta prosthesis implantation was performed. The patient received intensive care and was discharged on day 49 of hospitalization. His cerebral performance category score was 4 at discharge.

Conclusion: This case suggests that veno-arterial extracorporeal membrane oxygenation may be used for patients with aortic dissection presenting with cardiac arrest.

Key words: Aortic dissection, cardiopulmonary arrest, case report, coronary angiography, extracorporeal cardiopulmonary resuscitation

BACKGROUND

ACUTE AORTIC DISSECTION (AAD) has an extremely poor prognosis and high mortality rate. It requires early diagnosis and treatment. Circulatory support using veno-arterial extracorporeal membrane oxygenation (VA-ECMO) for aortic disease, particularly AAD, can exacerbate the false lumen or promote aortic valve regurgitation, possibly leading to a cardiac tamponade; therefore, it is not actively indicated for this condition. Veno-arterial ECMO is sometimes initiated when a patient experiences circulatory failure preoperatively, but such cases are extremely rare.[1]

Herein, we report a case of an out-of-hospital cardiac arrest (OHCA) that was successfully treated with extracorporeal cardiopulmonary resuscitation (ECPR) and VA-ECMO. Following various tests, AAD was diagnosed, and eventually lifesaving surgery was performed.

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CASE

THE PATIENT WAS a 66-year-old man who experienced chest discomfort while exercising at a gym, and developed presyncopal symptoms soon afterward. He developed ventricular fibrillation (VF) in the ambulance. Chest compressions were maintained, and defibrillation was performed; however, return of spontaneous circulation failed. The patient arrived at our hospital 27 min after the cardiac arrest (CA). His medical history was unremarkable.

On arrival at the hospital, the patient's Glasgow Coma Scale score was 3/15 (E1/V1/M1). The electrocardiogram showed pulseless electrical activity. No pericardial effusion was found on transthoracic echocardiography. Test results on admission are shown in Table 1.

Figure 1 outlines the clinical course and details of treatment. As VF indicated acute coronary syndrome (ACS),

Table 1. Results of clinical and radiological investigations in a 66-year-old man on hospital admission following an out-of-hospital cardiac arrest

Blood tests at hospital admission					
WBC, /μL (2,700–10,300)	10,500	CK, IU/L (30–150)	92	PT, % (75.0–120.0)	88.6
Hb, g/dL (13.6–16.6)	13.2	CK-MB, IU/L (<20)	31	PT-INR	1.06
PLT, /μL (130,000–350,00)	190,000	AST, IU/L (10–37)	169	aPTT, s (39.0–24.0)	77.1
TP, g/dL (6.0–8.3)	5.1	ALT, IU/L (6–35)	192	Fib, mg/dL (200–400)	256
Alb, g/dL (3.6–5.1)	3.2	LDH, IU/L (115–250)	472	D-dimer, μg/mL (0.0–1.0)	6.9
BUN(mg/dL (7–20)	17	GGTP, IU/L (1–40)	32	FDP, μg/mL (0.0–5.0)	13.3
Cre, mg/dL (0.5–1.1)	1.3	T-Chol, mg/dL (130–220)	174	AT-III activity, % (80.0–120.0)	78.1
Na, meq/L (135–141)	141	Glu, mg/dL (60–110)	365	Blood gas analysis	
K, meq/L (3.6–5.1)	2.8	HbA1c, NGSP, % (4.6–6.2)	5.6	pH (7.340–7.440)	7.022
Cl, meq/L (98–108)	103	CRP, mg/dL (0.00–0.30)	0.07	PaO_2, Torr (69–116)	197
Ca, mg/dL (8.7–10.1)	8.6	BNP, pg/mL (0.0–18.4)	6.7	$PaCO_2$, Torr (35.0–45.0)	46.9
iP, mg/dL (2.5–4.5)	7.1			HCO_{3-}, mmol/L (23.0–28.0)	11.6
Mg, mg/dL (1.9–2.6)	2.4			Lac, mmol/L (0.0–1.5)	14.5

Radiological examination	
Chest X-ray	Cardiothoracic ratio of 0.41.
	Bilateral hilar vascular shadow enhancement.
	Bilateral pulmonary edema was observed.
Head CT	No coarse bleeding or infarction lesions were observed.
	Gray/white matter signal ratio was maintained (region of interest ratio = 1.34).
Abdominothoracic contrast CT	Aortic dissection (Stanford type A, DeBakey type IIIa) was indicated.
	Dissection entry tear was located at the distal end of the aortic arch.
	Dissection re-entry tear was located immediately above the bifurcation of the bilateral branches of the common iliac artery.
	Congestion was observed at the posterior aspect of the base of both lungs.

Reference intervals for blood tests are given in parentheses.
Alb, albumin; ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; AT-III, antithrombin III; BNP, brain natriuretic peptide; BUN, blood urea nitrogen; Ca, calcium; CK, creatine kinase; CK-MB, creatine kinase-MB; Cl, chloride; Cre, creatinine; CRP, C-reactive protein; CT, computed tomography; FDP, fibrinogen degradation products; Fib, fibrinogen; GGTP, γ-glutamyl transpeptidase; Glu, glucose; Hb, hemoglobin; HbA1c, glycated hemoglobin; iP, inorganic phosphate; K, potassium; Lac, Lactate; LDH, lactate dehydrogenase; Mg, magnesium; Na, sodium; NGSP, National Glycohemoglobin Standardization Program; PLT, platelet count; PT, prothrombin time; PT-INR, prothrombin time – international normalized ratio; T-Chol, total cholesterol; TP, total protein; WBC, white blood cell count.

ECPR was performed with advanced cardiopulmonary life support using a MERA CPB circuit (Senko Medical Instrument, Tokyo, Japan) primed using normal saline with 3,000 U heparin. Percutaneous cannulation was performed with NSH heparin-coated cannulae (size 16 and 22 French for the femoral artery and vein, respectively) using the Seldinger technique under ultrasonic and fluoroscopic guidance.

Veno-arterial ECMO was established 35 min after the CA with a starting flow of 3.5 L/min. Ventricular fibrillation persisted after VA-ECMO was established, and organized rhythm was observed after electrical defibrillation. A 12-lead electrocardiogram was performed immediately, and we strongly suspected ACS with lesions on the left main trunk of the coronary artery (Fig. 2A). Emergency coronary angiography (CAG) was undertaken 26 min after arrival. We unsuccessfully attempted to engage the left coronary artery (LCA) using a guiding catheter. When confirming the location of the entry point on the LCA using a contrast agent, we discovered an enlargement of the ascending aorta and abnormal pooling of contrast at the basal part of the aortic valve, suggesting AAD (Fig. 2B). Therefore, CAG was stopped, and abdominothoracic contrast CT was performed. The results indicated AAD, Stanford type A. The true lumen was patent along the main branch artery from the ascending aorta to the aortic arch, and the VA-ECMO arterial cannula was correctly inserted into the true lumen (Fig. 2C). Regional oxygen saturation was maintained at approximately 65% from 1 h after hospital arrival, so a good neurological outcome was expected. After consulting a cardiovascular surgeon, ascending aorta synthetic graft replacement surgery

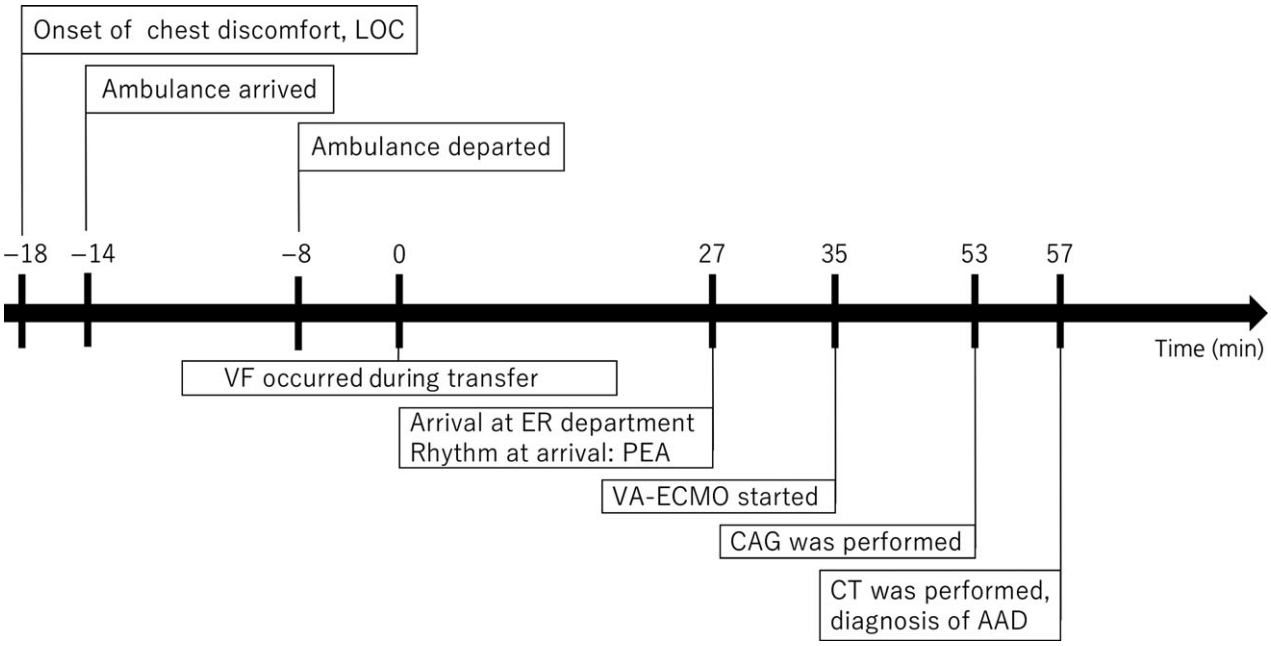

Fig. 1. Clinical course and treatment duration, from the onset of symptoms to diagnosis, in a 66-year-old man who suffered cardiopulmonary arrest caused by acute aortic dissection (AAD). Ventricular fibrillation (VF) occurred 18 min after onset of chest discomfort. The patient arrived in the emergency room (ER) 27 min after cardiopulmonary arrest. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was initiated 8 min after arrival. Coronary angiography (CAG) was carried out 18 min after VA-ECMO was initiated, once the patient's condition had stabilized. Computed tomography (CT) was carried out 22 min after the initiation of VA-ECMO. PEA, pulseless electrical activity.

was performed 41 min after hospital arrival. Radiography findings indicated a cardiothoracic ratio of 41%. Bilateral pulmonary edema was observed.

No accumulation of blood or fluid was observed inside the pericardium. A hematoma was found under the right side of the ascending aortic adventitia.

As no entry was found between the ascending aorta and left subclavian artery, the dissection was considered a retrograde extension with Stanford type B dissection. Three-quarters of the entire circumference of the aortic root had been dissected, but no dissection was observed in the LCA. Accordingly, the VF was assumed to result from compression of the right coronary artery due to the dissection.

After the aorta was declamped, the patient's own heart beat reappeared. The right ventricular pulse was found, but the left ventricle had poor contractility, particularly at the anteroseptal wall. Therefore, we reattached the peripheral VA-ECMO. No bleeding from the aortic anastomosis was observed; however, persistent bleeding was observed, particularly from the right internal thoracic artery. This bleeding was thought to result from damage to the right internal thoracic artery by chest compressions performed during cardiopulmonary resuscitation and triggered by coagulopathy, a complication of post-CA syndrome. Maintaining the ECMO flow was difficult due to the bleeding, and a large volume of blood products was required. Because of the coagulopathy, anticoagulation therapy was reluctantly discontinued.

The patient's circulatory status improved over time, and VA-ECMO was removed on day 4 of hospitalization. Findings from head CT carried out on the same day showed hypoxic encephalopathy, indicating a poor neurological prognosis. On day 49, the patient's condition had stabilized, so he was transferred to a recuperation hospital. His cerebral performance category score was 4 for both cerebral and overall performance at discharge.

DISCUSSION

ACCORDING TO HAGAN *et al.*,[2] the mortality rate for AAD ranges from 25% to 30%. Diagnosis from CT prior to CA is comparatively easy; however, once CA has occurred, the prognosis is often poor, and saving the patient is often difficult.

In the tertiary emergency center of our hospital, ECPR is performed for OHCA patients who meet certain criteria and

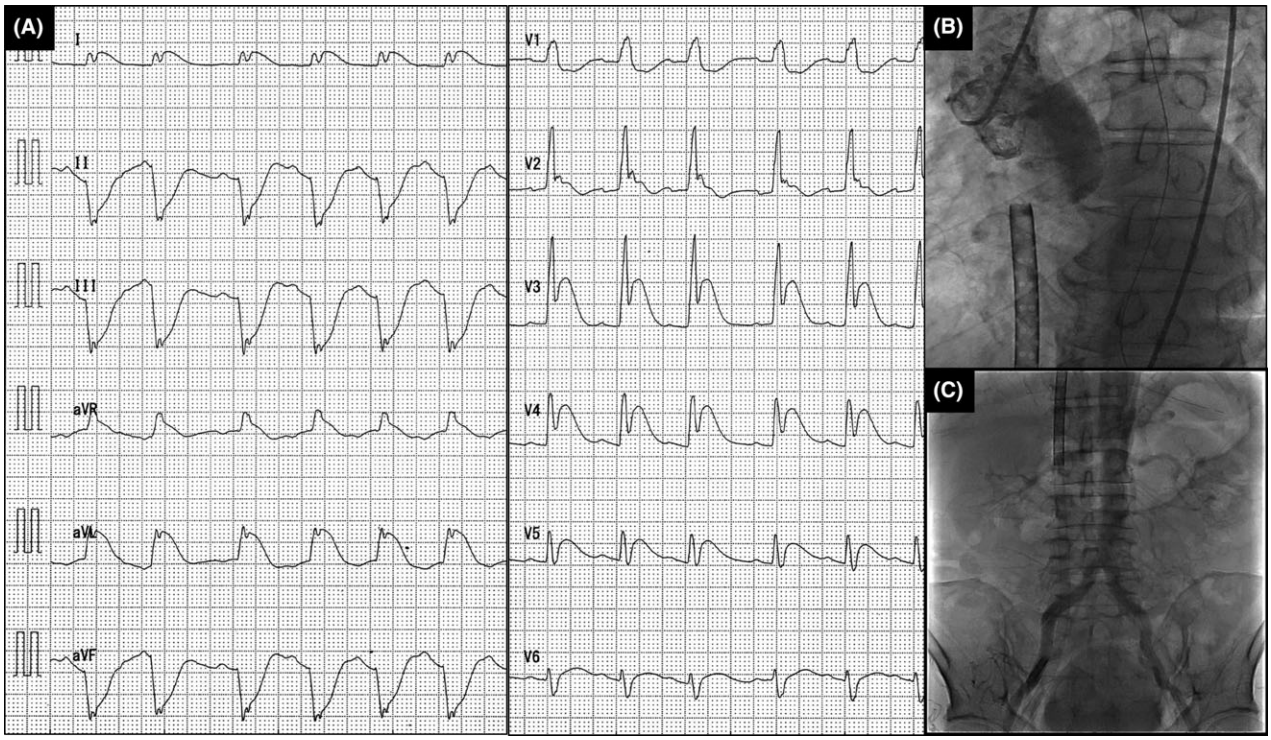

Fig. 2. Electrocardiogram and coronary angiograms of a 66-year-old man who suffered cardiopulmonary arrest caused by acute aortic dissection. A, Electrocardiogram shows QRS complex duration of 120 ms, ST elevation in left chest (I), left lateral arm (aVL), right lateral arm (aVR), and chest leads ($V_{3–6}$), and ST depression in left upper quadrant (II), right upper quadrant (III), and right lateral lower leg (aVF). B, Coronary angiogram showing enlargement of the ascending aorta and abnormal pooling of contrast at the basal part of the aortic valves. C, The arterial cannula was inserted correctly into the true lumen of the right femoral artery.

aims to achieve rehabilitation into society. Based on reports by the SAVE-J study group,[3] the indications for ECPR in our institution are as follows: (i) patients aged ≤65 years whose OHCA was witnessed by a bystander and in whom an initial shockable rhythm was found; or (ii) patients aged ≤70 years whose OHCA was witnessed by emergency medical service personnel and who were presumed to have a reversible underlying etiology, regardless of the initial rhythm. This case met almost all ECPR initiation criteria, and as the patient did not have pericardial effusion according to transthoracic echocardiography findings, his condition was not actively suspected to be an AAD; therefore, he was successfully resuscitated with ECPR.

In cases where a patient goes into cardiopulmonary arrest after the onset of VF while presenting with chest pain as the main complaint, ACS is the chief suspected condition. Hence, there are cases where the initial treatment intervention is started, and the aortic dissection is not discovered until after CAG has been started or completed.

Generally, VA-ECMO is contraindicated as circulatory support for aortic diseases. However, although AAD must be included in the differential diagnosis for causes of an OHCA, the limitations and treatment options for continuing cardiopulmonary resuscitation in a limited timeframe should also be considered. In this case, AAD was definitively diagnosed by contrast-enhanced CT after stabilizing the patient's circulatory status with ECPR. A good neurological prognosis was expected after establishing VA-ECMO, and we could treat the condition surgically, ultimately saving the patient. The patient survived because the ECMO arterial cannula was inserted into the true lumen of the aortic dissection, resulting in dilatation of the collapsed true lumen due to the ECMO counter flow. Fortunately, the true lumen was patent along the entire length of the main branch artery.

In conclusion, although ECPR is conventionally contraindicated for aortic diseases, there are some unavoidable situations that require OHCA resuscitation even before a diagnosis is made. This case presents an opportunity to reflect on ECPR as a treatment for AAD presenting with CA where the patient meets specific criteria before surgery.

DISCLOSURE

Ethics approval and consent to participate: This case report was approved by the institutional review board of Tokyo Metropolitan Bokutoh Hospital with a waiver of informed consent to protect participant anonymity. Ethics committee approval no. 29–35.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of interest: None declared.

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