INTRODUCTION

A 78-year-old woman entered cardiac arrest during intravascular treatment for pseudoaneurysm. Although extracorporeal cardiopulmonary resuscitation with extracorporeal membrane oxygenation (ECMO) is considered to be contraindicated in patients at risk of bleeding complications, we successfully restored spontaneous circulation via ECMO, and the intravascular treatment was completed without anticoagulants or other complications.

Extracorporeal cardiopulmonary resuscitation (ECPR) is a resuscitation method with a percutaneous cardiopulmonary support device that performs extracorporeal membrane oxygenation (ECMO) in combination with normal cardiopulmonary resuscitation. ECMO can assist cardiorespiratory function by passing blood removed via a centrifugal pump through an oxygenator, then returning it through cannulas percutaneously inserted into the femoral artery and vein.1 In cardiogenic cardiac arrest, ECPR significantly improved the 3-month survival rate from 9.9% to 22.7%, compared to conventional CPR.2 In the 2015 American Heart Association Guidelines,3 although routine use for cardiac arrest patients is not recommended, it may be considered for cases where ECPR is readily available and the cause of cardiac arrest can be identified. However, the effectiveness of ECPR for noncardiogenic cardiac arrest has been reported only with certain diseases, such as pulmonary thromboembolism,4 incidental hypothermia,5 and addiction,6 and its adaptation to other conditions is currently limited. Anticoagulants are required during ECMO, and significant bleeding complications occur in 27% of patients.3 Hence, there are few reports on the effectiveness of ECPR for hemorrhagic shock, and at present, it is unclear whether ECPR should be administered to such patients. In this paper, we present a case in which we administered ECPR for hemorrhagic shock and were able to get a good clinical outcome.

CASE HISTORY/EXAMINATION

We report a case of a 78-year-old woman taking 220 mg of dabigatran for atrial fibrillation. She had undergone tricuspid valve replacement surgery 1 month prior, during which a central venous catheter was inserted in the right internal jugular vein during the perioperative period. Her
postoperative course was good, and she was discharged. Then, 300 mL fresh blood hematemesis occurred without any discernable cause and she was transported to a nearby clinic. Upper gastrointestinal endoscopy was unable to identify the source of bleeding, and she was thus referred to our hospital. At the clinic, blood loss had reached an estimated 2 L, and 6 units in total of packed red blood cell transfusions had been administered by the time of arrival at our hospital. On admission, she was alert and conscious, and had a respiratory rate of 23 breaths/min, oxygen saturation of 100% under 2 L/min oxygen administration, blood pressure of 108/57 mm Hg, and pulse rate of 139 beats/min. Continuous fresh bleeding emitted from the oral cavity. The hemoglobin level had decreased to 6.2 from 9 g/dL at the previous clinic (Table 1).

3 | INVESTIGATIONS AND TREATMENT

Cervical-pelvic contrast computed tomography (CT) was performed to identify the source of bleeding, and an extravasation (Ev) image of the contrast agent was detected in the vicinity of the right papilla (Figure 1A-C). It was difficult to secure a visual field with nasopharyngoscopy due to the continuous bleeding, which we were unable to stop. Thus, we tried to perform intravascular treatment. Prior to the treatment, we administered 50 mg of ketamine for tracheal intubation and placed her on a ventilator. First, we placed a 5Fr sheath in the right femoral artery, and the right common carotid artery was contrast-enhanced with a 5Fr Simmons-type catheter. We then detected a pseudoaneurysm about 4.5 x 2 mm in size in the vicinity of the right facial artery, a branch of the external carotid artery (Figure 2A). At the time of imaging, Ev was not detected but we considered the lesion as the bleeding source. While we were attempting to access the bleeding source with a microcatheter, the patient went into cardiac arrest after bradycardia. We immediately started cardiopulmonary resuscitation, administered a total of two mg of adrenaline intravenously, and continued resuscitation for five more minutes. However, spontaneous circulation did not return, and we decided to start ECPR. ECMO was initiated 17 minutes after starting cardiopulmonary resuscitation with a right femoral 20Fr drainage cannula (Flexmate®, Toyobo) and a right femoral 16Fr return cannula (Flexmate®, Toyobo). With the aid of fluoroscopy, the tip of a drainage cannula was placed in the right atrium. Spontaneous circulation returned soon after, and we performed the intravascular treatment under ECMO. We selected the right facial artery with the microcatheter, reached the vicinity of the pseudoaneurysm, and embolized with n-butyl-2-cyanoacrylate (NBCA) diluted five times with lipiodol (Figure 2B).

| TABLE 1 | Patient's clinical parameters |
| Complete blood count | |
| WBC | 7700 | /µL |
| RBC | 197 | x10^6/µL |
| Hb | 6.2 | g/dL |
| Ht | 18.3 | % |
| Plt | 13.3 | x10^4/µL |

| Coagulation | |
| APTT | 48.8 | sec |
| PT | 24 | % |
| PT-INR | 2.41 |
| Fib | 165 | mg/dL |
| FDP | 12.4 | µg/mL |
| Ddimer | 5.21 | µg/mL |

| Arterial blood gas (10 L/min oxygenation) | |
| PH | 7.345 |
| PO₂ | 17.7 | mm Hg |
| PCO₂ | 20.9 | mm Hg |
| HCO₃⁻ | 28.4 | mmol/L |
| BE | 4.1 | mmol/L |
| Lac | 43.4 | mg/dL |

| Chemistry | |
| TP | 3.0 | g/dL |
| Alb | 1.9 | g/dL |
| BUN | 23.1 | mg/dL |
| Cre | 0.96 | mg/dL |
| Na | 132 | mEq/L |
| K | 5.4 | mEq/L |
| Cl | 106 | mEq/L |
| Ca | 6.6 | mg/dL |
| P | 3.6 | mg/dL |
| Mg | 1.9 | mg/dL |
| AST | 19 | U/L |
| ALT | 11 | U/L |
| LDH | 170 | U/L |
| ALP | 198 | U/L |
| T-Bil | 1.0 | mg/dL |
| CK | 30 | U/L |
| CRP | 0.06 | mg/dL |
| Fe | 103 | µg/dL |
| TIBC | 160 | µg/dL |
| ferritin | 61 | ng/mL |

Abbreviations: Alb, albumin; ALP, alkaline phosphatase; APTT, activated partial thromboplastin time; BE, base excess; BUN, blood urea nitrogen; CK, creatine kinase; Cre, creatinine; FDP, fibrin/fibrinogen degradation products; Fib, fibrinogen; Hb, hemoglobin; Ht, hematocrit; Lac, lactate; Plt, platelet; PT, prothrombin time; PT-INR, PT-international normalized ratio; RBC, red blood cell; T-Bil, total bilirubin; TP, total protein; WBC, white blood cell.
4 | OUTCOMES AND FOLLOW-UP

After the treatment, her hemodynamics stabilized, spontaneous circulation was sufficient, and she was weaned off ECMO six hours after hospitalization (5 hours 25 minutes from the start of ECMO). We did not use any anticoagulants during the ECMO management, but activated clotting time (ACT) was maintained at around 160 seconds. There were no neurologic sequelae associated with rebleeding or cardiac arrest, and she was transferred for rehabilitation on the 30th day of the disease.

5 | DISCUSSION

In this case, hemorrhagic shock occurred due to a pseudoaneurysm rupture in the superficial artery, a branch of the external carotid artery, and the patient went into cardiac arrest. She did not have any of the characteristic physical findings for vasculitis and was negative for antineutrophil cytoplasmic antibodies (ANCA). We considered that vasculitis was less likely to be the cause of the pseudoaneurysm; a collagen disease specialist agreed with this assessment. Thus, the pseudoaneurysm may have been a complication of the central venous catheter insertion during the tricuspid valve replacement. Arterial puncture is a complication in 10% of central venous catheter insertions. There are some reports on injuries of major arteries, such as the carotid, subclavian, and vertebral arteries,4-6 but few reports on injuries in the peripheral arteries. We found only three cases of internal jugular vein or subclavian vein puncture associated with pseudoaneurysms of the lower thyroid artery.7-9 In cardiac surgery, the central venous catheter is often inserted from the upper portion of the neck, in order to maintain an appropriate distance from the operative field. Therefore, clinicians should bear in mind the possibility of facial artery injury.

This patient did not respond to normal resuscitation; thus, we decided to perform ECPR. ECMO is considered to be contraindicated for cases with a risk of bleeding because anticoagulation therapy is required. However, recent reports show that ECMO could be safe and effective even in trauma cases,10 and there are several cases where ECMO was successfully performed despite hemorrhage or coagulopathy using a heparin-coated circuit. In these cases, the target ACT was shorter (160-180 seconds) than usual, sometimes even

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**FIGURE 1** Cervical-pelvic contrast computed tomography images in (A) noncontrast, (B) arterial, and (C) venous phases. An extravasation of the contrast agent was detected near the right papilla and was thought to be the source of bleeding at the time, indicated by a circle.

**FIGURE 2** A, A 4.5 × 2 mm pseudoaneurysm in the right facial artery in the right common carotid angiography. There was no obvious extravasation of the contrast agent at the time of imaging, but it was thought to be the source of bleeding. B, With a microcatheter, we reached the vicinity of the pseudoaneurysm and it was plugged with n-butyl-2-cyanoacrylate (NBCA) diluted five times with lipidol. C, Disappearance of the pseudoaneurysm was confirmed by imaging after embolization. Persistent bleeding from the oral cavity also ceased.
without anticoagulants, and bleeding complications were not reported.\textsuperscript{11} In fact, the incidence of bleeding-related deaths is <15\% in trauma cases using ECMO, and some authors have suggested that it is not necessarily contraindicated.\textsuperscript{12} We discovered only one case report of ECPR for amniotic fluid embolism with hemorrhagic shock, in which heparin was not administered for 48 hours from the start of ECMO, and the ACT was kept at around 150 seconds with a low dose of heparin at 800 IU/h.\textsuperscript{13} Usually, a 3000-unit bolus of unfractionated heparin is administered and continuous heparin dosage is adjusted to maintain an ACT of 180-200 seconds during ECMO. However, in the present case, we were able to avoid bleeding complications using a heparin-coated circuit without the need for anticoagulants because her hemodynamics were stable after intravascular therapy and she was weaned off ECMO early.

In the present case, we used NBCA, which is a permanent embolic substance that exerts an embolic effect by reacting with ions in blood or tissues during intravascular treatment. Because coils and gelatin sponges are not the embolic substance itself, and the embolic effect depends on coagulability, this effect decreases markedly due to the influence of anticoagulation.\textsuperscript{14} Therefore, NBCA may be effective in patients with coagulopathy or anticoagulants in situations where reliable hemostasis is required.

In conclusion, the present report demonstrates that even in cases of cardiac arrest caused by hemorrhagic shock, ECPR may be an effective treatment option, as long as the bleeding sources are controllable, anticoagulant therapy is well-managed, and the patient is weaned off ECMO early.

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**CONFLICT OF INTEREST**

None declared.

**AUTHOR CONTRIBUTIONS**

MO: contributed to attending physician and drafted the manuscript. TM: involved in literature review. DY: contributed to attending physician and drafted the manuscript. YK: revised the manuscript. HK: revised the manuscript. YA: contributed to final approval for publication.

**DATA AVAILABILITY STATEMENT**

All data of this case are included in this published article.

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