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CASE REPORT

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What should be done when appropriate antiplatelet therapy fails to prevent stent thrombosis? Two case reports

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Key Clinical Message

When performing noncardiac surgery in high-risk patients with PCI, anesthesiologists should be prepared for in-stent thrombosis, that do not respond to conventional treatments and cardiopulmonary resuscitation. VA-ECMO is an advantageous treatment method in those patients.

KEYWORDS

dual antiplatelet therapy, extracorporeal membrane oxygenation, major adverse cardiac events, noncardiac surgery, percutaneous coronary intervention

1 | INTRODUCTION

Perioperative management of patients with percutaneous coronary intervention (PCI) has been challenging.[1] The 2016 American College of Cardiology/American Heart Association (ACC/AHA) focused update on the duration of dual antiplatelet therapy (DAPT) in patients with coronary artery disease, recommends that noncardiac surgery can be performed in patients with drug-eluting stents (DESs) after 6 months of DAPT, and in patients with stable ischemic heart disease (Class IIb) after 5 days of DAPT cessation.[2] Recent reports support the safety of second-generation DESs and recommend DAPT cessation 180 days after PCI.[1,3,4] However, there are also reports that suggest that perioperative DAPT does not significantly decrease the occurrence of perioperative major adverse cardiac events (MACEs).[5] Moreover, the evidence regarding perioperative management of patients after PCI is insufficient and cannot guarantee patient safety.[5] Hence, if adverse cardiac events cannot be entirely prevented, it is imperative to prepare for their management. We encountered two cases of perioperative myocardial infarction (MI) in patients with PCI receiving DAPT for more than 6 months, and resuscitated them using extracorporeal membrane oxygenation (ECMO). We present these cases along with a brief review of the literature.

Institutional review board (IRB) approval was obtained from Dankook university hospital IRB (IRB approval number: 2022-04-006). Written informed consent was obtained from the patients for the publication of this study.

2 | CASE REPORTS

2.1 | Case 1

A 74-year-old man (American Society of Anesthesiologists class III; weight, 66.7 kg; height, 166.6 cm, nonsmoker) with a right distal ureter tumor was scheduled for distal ureterectomy. The patient had been treated for hypertension and chronic kidney disease (CKD).

In March 2020 (approximately 21 months before surgery), diagnostic coronary angiography (CAG) had revealed significant stenosis in proximal left anterior descending artery (p-LAD), proximal left circumflex artery (p-LCX) and middle right coronary artery (m-RCA). After dilation with Ikazuchi-Rev (Kaneka medical products) balloon catheter (2.0 × 15 mm, 10 atm), a 3.5 × 32 mm

SYNERGY™ (Boston Scientific) stent was implanted in the p-LAD, and a 3.5×16 mm SYNERGY™ stent was implanted in the m-RCA. Follow-up CAG in February 2021 (10 months before surgery) showed no lesion progression. Preoperative echocardiography showed regional wall motion abnormality (hypokinesia in the anteroseptal myocardium from mid to apex and antero-apex), and a left ventricular ejection fraction (LVEF) of 51%, with no interval change from the echocardiography done 10 months ago. Nineteen days before surgery, the patient underwent ureteroscopy and biopsy after cessation of DAPT for 5 days, which were uneventful. DAPT was reinitiated after this procedure. Preoperative laboratory findings were normal, with a hemoglobin level 13.3 mg/dL, a hematocrit level 40.6%, and a platelet count of 226,000/μL. Preoperative platelet function analysis results were also within the normal limits. Electrocardiography and chest radiography results were also unremarkable. After admission to the operating room, patient monitoring systems, including electrocardiogram, continuous blood pressure (BP), pulse oximetry, bispectral index (BIS), central venous pressure, and continuous arterial BP with radial artery catheter insertion, were instituted. His initial vital signs showed a BP of 133/70 mmHg, heart rate (HR) of 83 beats/min (bpm), and respiration rate of 23 breaths/min. General anesthesia was induced with an intravenous injection of 1 mg/kg lidocaine, 0.2 mg/kg etomidate, and 0.6 mg/kg rocuronium, followed by maintenance with 1.2 minimum alveolar concentration (MAC) desflurane. Conventional endotracheal intubation was performed with direct laryngoscopy, and a 7.5-mm plain tube was inserted. Approximately 5 h after the initiation of the surgical procedure, the patient developed sudden profound hypotension (BP, 65/50 mmHg; HR, 54 bpm; BIS, 38; and end-tidal carbon dioxide [$EtCO_2$], 26 mmHg) with ST-segment elevation (from −0.1 to 1.5 mm). Hypotension and bradycardia were treated with a fluid challenge of crystalloid solution (500 mL), and intravenous injections of 10 mg ephedrine and 100 μg phenylephrine; however, these interventions proved ineffective. Intravenous administration of 0.5 mg atropine and incremental doses of intravenous epinephrine (10 μg + 20 μg + 50 μg + 100 μg + 200 μg) was also ineffective. Dopamine (10 μg/kg/min), dobutamine (10 μg/kg/min), and epinephrine (0.1 μg/kg/min) were administered via infusion at the same time to treat hypotension. However, the BP decreased further to 50/40 mmHg, and his HR also decreased to 43 bpm. Coronary artery stent thrombosis was suspected, and the ECMO team was consulted. Cardiopulmonary resuscitation (CPR) was initiated, and 1 mg of epinephrine was administered intravenously three times alongside chest compressions; however, these interventions proved ineffective. Simultaneously with CPR, veno-arterial (VA) ECMO was performed on the right femoral artery and vein approximately 30 min after the occurrence of profound hypotension; subsequently, effective circulation was confirmed, and his mean arterial pressure (MAP) was restored to 65 mmHg. His BIS was 23–41 and $EtCO_2$ was 24–28 mmHg during the CPR.

The patient was transferred to the CAG room. CAG showed in-stent thrombosis and total occlusion in the left main coronary artery with 90% occlusion in the proximal left circumflex artery (LCX) (Figure 1). Mechanical thrombectomy and stent insertion were performed successfully in the proximal and mid-LAD approximately 75 min after the profound hypotension occurrence. After dilation with Ikazuchi-Rev balloon catheter (2.5×20 mm, 10 atm), a 3.25×28 mm Xience Sierra™ (Abbott) stent was implanted in the p-LAD, and a 2.75×48 mm Xience Sierra™ stent was implanted in the m-LAD. The patient was transferred to the intensive care unit. Ventricular tachycardia was noted and successfully controlled with direct-current (DC) cardioversion. ECMO weaning and removal were performed on postoperative day (POD) 2. The patient was extubated on POD 3 and transferred to the general ward on POD 6. Postoperative echocardiography showed aggravated regional wall motion abnormality (akinesia in the inferoseptal and anteroseptal regions from mid to apex and lateral apex) and an LVEF of 41%. The patient was discharged on POD 23 without further complications.

2.2 | Case 2

A 52-year-old woman (American Society of Anesthesiologists class, III; weight, 76.2 kg; height, 157.2 cm, nonsmoker) with a rotator cuff tear in her right shoulder was scheduled for arthroscopic rotator cuff tear repair. The patient had been treated for coronary artery disease, hypertension, and diabetes mellitus (DM).

She had multiple coronary artery stenotic lesions (proximal LAD, 40%; proximal LCX, 50%; distal LCX, 60%; first obtuse marginal branch (OM), 75%; proximal RCA, 30%; and right posterior descending artery, 90%). After dilation with Ikazuchi-Rev balloon catheter (2.0×15 mm, 6 atm), a 2.75×23 mm Xience Prime™ (Abbott) stent was implanted at the first OM near the p-LCX in July, 2012. In-stent restenosis (ISR) was diagnosed in December 2019 and treated with drug-eluting balloon angioplasty on the proximal LCX, and the first OM was managed with Ikazuchi-Rev balloon catheter (2.0×15 mm, 6 atm) and a Sequent® Please Neo (B Braun) drug-coated balloon catheter (2.75×20 mm). Approximately 6 months prior to this surgery, the patient had been scheduled for the same surgery, but she had profound hypotension just after the induction of general anesthesia and underwent CAG, which showed aggravated

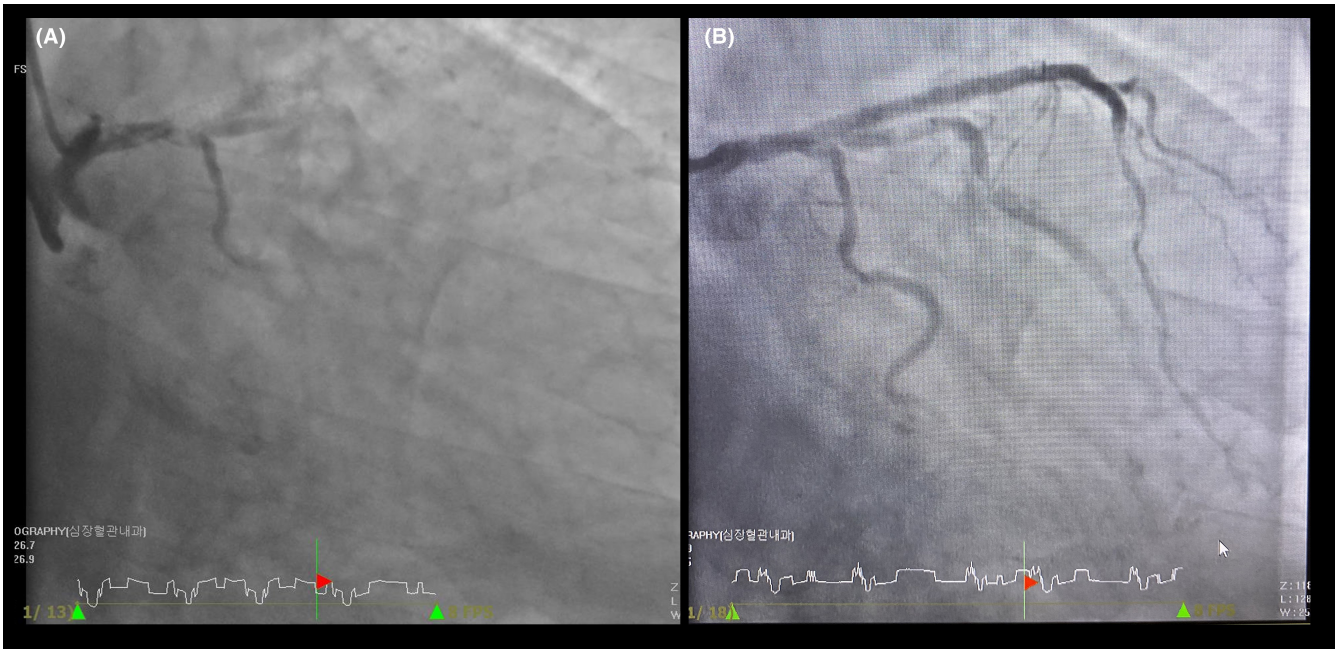

FIGURE 1 (A) Coronary artery angiogram shows left main coronary artery stent thrombosis and total occlusion, proximal LCX with 90% occlusion. (B) Normal blood flow after revascularization.

coronary artery disease (proximal LAD, 40%; proximal LCX, 70%; distal LCX, 75%; first OM, 90%; and proximal-RCA, 40%) and ISR in the first OM again. After dilation with Ikazuchi-Rev balloon catheter (2.0 × 15 mm, 6 atm), a 2.75 × 20 mm Orsiro® Mission (Biotronik) stent was implanted at the first OM.

Preoperative echocardiography showed no regional wall motion abnormality and an LVEF of 75%. Preoperative laboratory findings were normal (hemoglobin, 13.5 mg/dL; hematocrit, 39.3%; and platelet count, 209,000/μL). Electrocardiography showed a first-degree atrioventricular block, and chest radiography results were unremarkable. The cardiology team recommended administering a perioperative nitroglycerine infusion to decrease the risk of coronary artery vasospasm. Nitroglycerin infusion was started the day before surgery and DAPT was continued until the day of surgery.

After admission to the operating room, patient monitoring systems, including electrocardiogram, continuous BP, pulse oximetry, BIS, and continuous arterial BP with radial artery catheter insertion, were instituted. Her initial vital signs showed a BP of 140/90 mmHg, HR of 75 bpm, and respiration rate of 21 breaths/min. General anesthesia was induced with an intravenous injection of 1 mg/kg lidocaine, 0.2 mg/kg etomidate, and 0.6 mg/kg rocuronium, followed by maintenance with 1.0–1.3 MAC desflurane. Conventional endotracheal intubation was performed with direct laryngoscopy, and a 7.0-mm plain tube was inserted. She had no postural variation in BP when her position changed from a supine position to a seated position. Approximately 1 h after the induction of anesthesia, sudden profound hypotension occurred (BP, 52/30 mmHg; HR, 52 bpm; BIS, 41; $EtCO_2$, 28 mmHg) with ST-segment elevation (from 1.1 to 2.5 mm). Hypotension and bradycardia were treated with a fluid challenge of crystalloid solution (300 mL), an intravenous injection of ephedrine (10 mg + 10 mg), and incremental doses of phenylephrine (100 μg + 200 μg), but these were ineffective. Intravenous atropine (0.5 mg) and incremental doses of intravenous epinephrine (10 μg + 20 μg + 50 μg + 100 μg + 200 μg) were also ineffective. Coronary artery stent thrombosis was suspected, and the ECMO team was consulted. Dopamine (10–20 μg/kg/min), dobutamine (10–20 μg/kg/min), and epinephrine (0.1 μg/kg/min) were administered via infusion at the same time to treat hypotension. However, these aggressive treatments proved ineffective; her BP decreased to 42/30 mmHg, and her HR decreased to 43 bpm. CPR was initiated 10 min after the occurrence of hypotension, and 1 mg epinephrine was administered intravenously three times alongside chest compressions; however, these interventions proved ineffective. While performing CPR, VA-ECMO was performed on the right femoral artery and vein approximately 25 min after the profound hypotension occurrence; effective circulation was subsequently confirmed, and her MAP was restored to 65 mmHg. Her BIS was >30–34, and $EtCO_2$ was 22–31 mmHg during CPR.

The patient was transferred to the CAG room. CAG showed total occlusion of the proximal LAD and LCX (Figure 2). Mechanical thrombectomy, balloon angioplasty, and stent insertion were performed successfully in

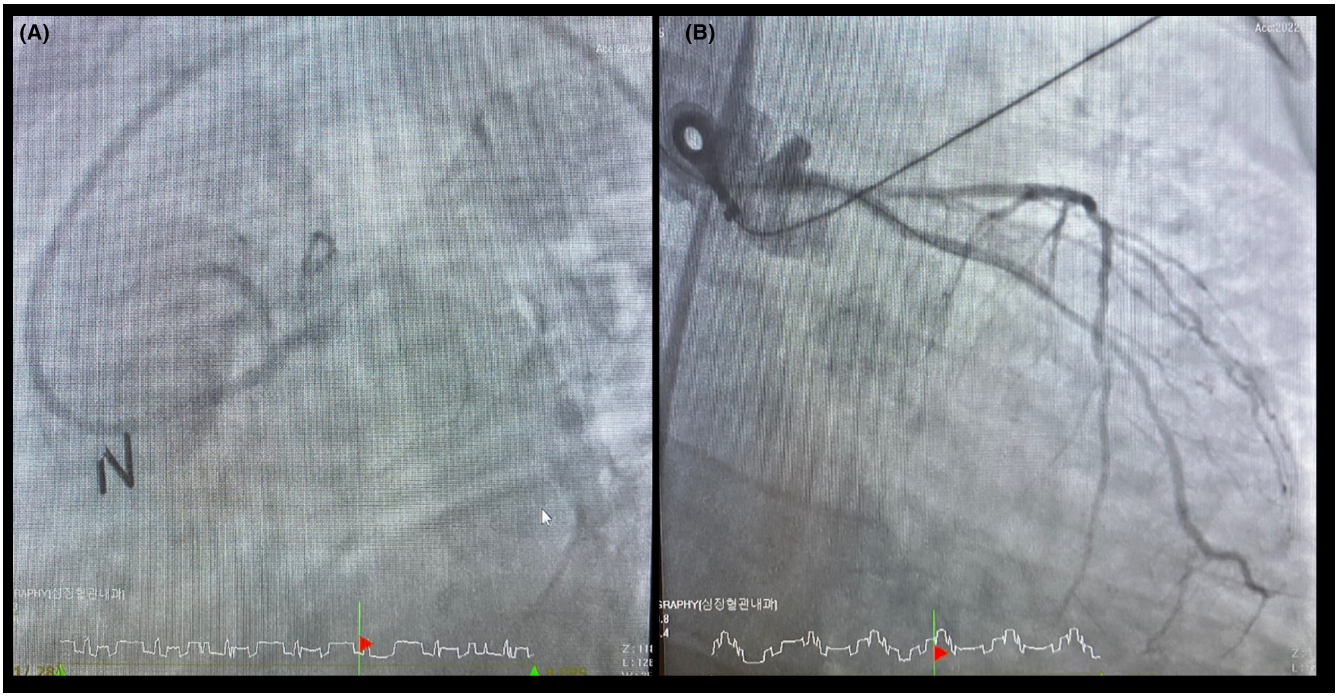

FIGURE 2 (A) Coronary artery angiogram showed total occlusion of proximal-LAD and proximal-LCX. (B) Normal blood flow after revascularization.

the proximal LAD and LCX (approximately 82 min after the profound hypotension).

After dilation with Ikazuchi-Rev balloon catheter (2.0×15 mm, 14 atm and 2.5×15 mm, 14 atm), a 3.5×38 mm SYNERGY™ stent was implanted in the p-LAD, and a 2.75×32 mm SYNERGY™ XD (Boston Scientific) stent was implanted in the p-LCX.

The patient was transferred to the intensive care unit. Postoperative echocardiography showed severe left ventricular dysfunction (akinesia in the inferoseptal, anteroseptal, anterior, lateral wall from mid to apex, whole apex, and whole LV hypokinesia-LVEF: 26%). On POD 1, ventricular fibrillation was detected several times but was successfully controlled with DC cardioversion. Her cardiac contractility gradually recovered (LVEF: 38%), and ECMO weaning was attempted from POD 3 but failed until POD 6 due to intermittent ventricular fibrillation. The patient was transferred to another hospital under VA-ECMO treatment for trying other treatment options.

3 | DISCUSSION

The optimal management of perioperative DAPT in patients previously treated with PCI is still controversial. Favorable results with second-generation DESs have been reported, which led to the updating of the 2016 ACC/AHA guidelines.[2,3] Several studies on DAPT duration in patients with second-generation DESs showed a similar risk of stent thrombosis among patients treated for at least 3–6 months with DAPT.[2] Thus, the guidelines recommend the cessation of DAPT 6 months after PCI and proceeding with noncardiac surgery. However, Smith et al.[4] reported a 3.1% incidence rate of MACEs after DAPT treatment for longer than 1 year. Late-stent thrombosis has been reported up to 5.5 years after PCI. This means that even with DAPT treatment for a sufficient period, the occurrence of MACEs cannot be entirely prevented. The 2016 ACC/AHA guidelines recommend continuing aspirin therapy during surgery, if possible, but this is based primarily on expert opinion.[2] However, there is still disagreement over the protective effect of continuing perioperative aspirin to decrease MACE occurrence.[1,6,7] Howell et al.[6] reported that patients who continued with DAPT during the perioperative period did not show significant MACE reduction and had an increased risk of perioperative bleeding. The mechanism underlying the occurrence of perioperative MACEs is still unclear.[4] Wąsowicz et al.[7] reported that there was no difference in the degree of occurrence with, either aspirin or clopidogrel-mediated platelet inhibition, based on a platelet mapping assay between subjects with or without MACE. They also reported that the degree of platelet inhibition did not change significantly through the first 24 postoperative hours. Thus, it is suggested that perioperative MIs likely result from myocardial oxygen supply–demand imbalance (type II MI) or lesion progression.[7,8]

In the cases reported herein, the first patient was on DAPT for 21 months after PCI, which was discontinued

5 days before surgery. The second patient was on DAPT for 6 months after PCI and continued DAPT perioperatively but presented with MACEs. In this regard, a long (>6 months) period of DAPT and perioperative antiplatelet therapy may not guarantee patient safety. Bridging therapy with various parenteral antiplatelets or anticoagulants has not yielded convincing evidence yet, but can still be considered if DAPT has to be discontinued perioperatively, especially within 1 month of PCI.[2,8] Rossini et al.[8] recommended bridging therapy with the intravenous P2Y12 inhibitor (cangrelor) or small molecule glycoprotein IIb/IIIa receptor inhibitors (tirofiban, eptifibatide) while continuing small-dose of aspirin perioperatively in patients at high risk of stent thrombosis and perioperative bleeding.

A prospective study evaluated risk factors that may increase the incidence of MACEs and concluded that preoperative anemia, severe renal failure, urgent surgery, high-risk surgery, and interruption of antiplatelet agents for more than 5 days may increase the risk of MACEs.[4] Rossini et al.[8] described clinical risk factors (acute coronary syndrome at the time of index PCI, multiple previous MIs, LVEF <35%, CKD, and DM), and angiographic features (long and multiple stenoses, overlapping stents, small stent diameter, bifurcation lesions, extensive coronary artery disease, incomplete revascularization, and treatment of chronic total occlusion) that will increase the risk of MACE.

Both our cases were high-risk patients with multiple risk factors (history of CKD, multiple coronary artery lesions, multiple previous MIs, multiple stenosis, CKD, and DM). When proceeding with anesthesia and noncardiac surgery in high-risk patients with PCI, following 2016 ACC/AHA guidelines alone may be insufficient for the patient's safety. Anesthesiologists should be prepared for the worst-case scenario, such as MI with stent thrombosis and profound hypotension, which do not respond to conservative treatments and conventional CPR.

Early PCI for revascularization of the coronary artery lesion is crucial but may be challenging to perform when the patient's status is extremely unstable.[9] Stent thrombosis or total occlusion in the proximal coronary artery can result in severe cardiac dysfunction and conventional conservative management may not be effective. In this regard, an early decision to apply mechanical circulatory support devices (MCSD) is crucial.[10]

Temporary MCSD can help stabilize patients and allow time for decisions regarding the appropriateness of transitions to definitive management, such as use of durable MCSDs as a bridge or destination therapy, stabilization until cardiac transplantation, or in the case of improvement and recovery, suitability for device removal.[11]

Temporary MCSDs, including an intra-aortic balloon pump (IABP), percutaneous ventricular assist devices (p-VAD), and VA-ECMO, have been used to treat severe cardiogenic shock.[12]

An IABP is most often used as the first-line support in patients with various indications, including acute MI with or without shock, high-risk PCI, and cardiogenic shock.[12] However, according to previous randomized controlled trials, IABP use did not significantly improve hospital mortality.[9,12] P-VAD use induced a significantly higher MAP and a faster decrease in lactate levels than those observed when using IABP. However, there was no significant difference in 30-day mortality, and p-VAD use caused more bleeding complications.[12]

VA-ECMO involves the use of a modified heart-lung bypass machine that performs life support for a longer time (days or weeks) to treat life-threatening cardiopulmonary failure.[13] It consists of a pump capable of propelling up to 8 L/min of blood via venous drainage and arterial return cannulas.[10] A hollow fiber membrane oxygenator that ensures blood oxygenation and carbon dioxide clearance is spliced into the circuit.[10] Full VA-ECMO support has several advantages over other MCSDs. First, VA-ECMO can maintain systemic circulation with oxygenated blood regardless of residual cardiopulmonary function. The native right ventricular (RV) function is not overly critical for providing systemic perfusion with VA-ECMO (proper RV function is needed in IABP and some p-VADs).[10] Second, diagnostic and therapeutic interventions can be performed in a relatively stable status while maintaining appropriate hemodynamics, gas exchange, and potential organ recovery time. Third, VA-ECMO significantly decreases RV preload, transpulmonary blood flow, left ventricular end-diastolic pressure and volume, and the use of inotropic drugs and vasopressors that decrease cardiac workload. Fourth, fluid removal and venous congestion relief can be enhanced by splicing a continuous hemodialysis machine.[10]

It is recommended that VA-ECMO should be initiated within 60 min of diagnosing refractory cardiogenic shock after the failure of fluid resuscitation and pharmacologic therapies.[10] Sheu et al.[13] reported that VA-ECMO application in patients with profound cardiogenic shock showed a 45.8% reduction in 30-day mortality. The 2021 European Resuscitation Council Guidelines recommend extracorporeal CPR as rescue therapy for selected patients with cardiac arrest when conventional advanced life support measures are failing or to facilitate interventions like CAG and PCI.[14]

VA-ECMO is a promising strategy but is associated with potentially devastating complications, including bleeding, vascular injury, limb ischemia, infections, patient immobility, and hyperbilirubinemia.[10] Close monitoring, smaller-sized cannulas, and regular laboratory tests can decrease these complications.

4 | CONCLUSION

In conclusion, we treated two patients with PCI based on the 2016 ACC/AHA guidelines, but the patients developed MI and profound hypotension caused by stent thrombosis. Despite conventional CPR being ineffective, we successfully revascularized the patients using VA-ECMO while maintaining a stable condition. Anesthesiologists should be prepared for the worst scenario when performing non-cardiac surgery in patients with PCI. Early diagnosis and revascularization are crucial to maintain a stable condition. VA-ECMO is an advantageous treatment method in patients with stent thrombosis and profound hypotension. The ECMO team should be available during the perioperative period.

AUTHOR CONTRIBUTIONS

Sungman Hong: Writing – original draft. **Jaegyok Song:** Supervision; writing – review and editing. **Sung mi Ji:** Writing – review and editing. **Wonkyu Lee:** Data curation.

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CONFLICT OF INTEREST STATEMENT

The authors declare that no conflict of interest exists.

DATA AVAILABILITY STATEMENT

All data generated or analyzed during this study are included in this article and its supplementary information files. Further information can be obtained from the corresponding author upon reasonable request.

ETHICS STATEMENT

Institutional review board (IRB) approval was obtained from Dankook university hospital IRB.

CONSENT

Written informed consent was obtained from the patients for the publication of this study.

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