Successful Rescue of Sustained Ventricular Tachycardia/Ventricular Fibrillation after Coronary Artery Bypass Grafting by Extracorporeal Membrane Oxygenation

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Abstract: Extracorporeal membrane oxygenation (ECMO) can be set up quickly at the bedside and provides reliable temporary mechanical circulatory support for severe heart failure. We report the case of a 56-year-old female with circulatory collapse due to sustained ventricular tachycardia and ventricular fibrillation (VT/Vf) after coronary artery bypass grafting (CABG) who was successfully resuscitated using ECMO. The sustained VT/Vf might have been secondary to myocardial stunning, ischemia, infarction, or reperfusion. There were 40 cardioversions within the first 5 postoperative days. The patient improved after 8 days of ECMO in addition to use of an intraaortic balloon pump and administration of inotropic agents for profound heart failure. Left ventricular ejection fraction improved from 28% preoperatively to 54.5% on the 20th postoperative day. Cardiogenic shock due to sustained VT/Vf after CABG may be an indication for ECMO support. Immediate establishment of circulatory support using ECMO provides valuable time for spontaneous and interventional correction of reversible causes of sustained VT/Vf.

Key words: extracorporeal membrane oxygenation coronary artery bypass grafting ventricular tachycardia ventricular fibrillation

Simple ventricular arrhythmias are not uncommon after coronary artery bypass grafting (CABG) and may not considerably affect the patient’s prognosis [1]. However, sustained ventricular tachycardia and ventricular fibrillation (VT/Vf), defined as those lasting more than 30 seconds, are among the most dreadful events after CABG [1, 2], and carry an in-hospital mortality rate of around 25 to 44% [1, 3]. In post-CABG patients, the estimated incidence of new-onset sustained VT/Vf is around 0.41 to 3.1% [1, 2]. Associated factors include myocardial ischemia, post-infarction scarring, and profound heart failure [1-4].

Extracorporeal membrane oxygenation (ECMO) should be considered in patients with drug-resistant sustained VT/Vf [5]. For reversible heart failure, ECMO is the mechanical circulatory support of choice after intraaortic balloon pumping (IABP) [6]. ECMO might save more than half of patients with cardiogenic shock after coronary artery surgery, allowing them to survive to the point of hospital discharge [7, 8].

Rescue of sustained VT/Vf using ECMO or a ventricular assist device (VAD) has been reported before [5, 9, 10]. These cases included post-CABG adults [5, 10] and non-CABG children [9]. The duration of ECMO support ranged from 6 hours to 3 days [5, 10]. Survival after recurrent, sustained, and refractory VT/Vf after a relatively long duration of ECMO support is unusual. We report the case of an adult patient who was successfully rescued using ECMO support for 8 days. She had 40 cardioversions for recurrent sustained VT/Vf within the first 5 days after CABG.
Case Report

A 56-year-old woman was sent to the emergency room due to worsening chest pain and dyspnea for 6 months. She had undergone percutaneous transluminal coronary angioplasty (PTCA) of the left anterior descending artery (LAD) and right coronary artery (RCA) at another medical center 2 months previously.

Physical examination showed regular heartbeat, fine lung crackles, and mild leg edema. Laboratory data were unremarkable except for hyperglycemia (283 mg/dL). Electrocardiogram (ECG) showed sinus rhythm, Q waves at inferior limb leads, ST elevation at anterior precordial leads, a corrected QT interval of 370 ms, and a QRS duration of 80 ms. Coronary angiography found 95% stenoses over the proximal LAD, left circumflex artery, and RCA. Echocardiography showed a left ventricular ejection fraction (LVEF) of 28%, global hypokinesia, and no significant valvular dysfunction. In the cardiac surgery ward, she received infusion of nitroglycerin but no catecholamine.

She underwent elective CABG under cardiopulmonary bypass. The left internal mammary artery was grafted to the obtuse marginal artery. Two greater saphenous veins were grafted to the LAD and the RCA. However, immediately after decannulation, a sustained attack of VT/Vf developed unexpectedly. Intravenous lidocaine (1 mg/kg) and magnesium sulfate (64 mEq) failed to reverse the arrhythmias. Ten cardioversions were given to restore the sinus rhythm. Proven cardiogenic shock followed. Amiodarone (6 mg/kg loading), dopamine (13.25 µg·kg⁻¹·min⁻¹), epinephrine (0.085 µg·kg⁻¹·min⁻¹), norepinephrine (0.26 µg·kg⁻¹·min⁻¹), milrinone (25 µg/kg loading), and emergency IABP all failed to control the cardiogenic shock. Veno-arterial ECMO was then urgently set up via the right femoral route. ECMO rapidly stabilized the circulation. In the intensive care unit, the patient’s hemodynamic status under ECMO was maintained by the infusion of amiodarone (12 µg·kg⁻¹·min⁻¹), milrinone (0.5 µg·kg⁻¹·min⁻¹), norepinephrine (0.052 µg·kg⁻¹·min⁻¹), nitroglycerin (3.4 µg·kg⁻¹·min⁻¹), and dopamine (6.6 µg·kg⁻¹·min⁻¹). Within the first 4 hours postoperatively after ECMO, sustained VT/Vf relapsed and she received eight cardioversions. During this time, electrolytes and hemoglobin were intensively monitored and interventions were given to maintain them within normal ranges. During the fifth postoperative hour, emergency surgery was performed to control bleeding. A 2-mm tear in the middle portion of the greater saphenous vein grafted to the RCA was found and repaired. Lidocaine infusion was started (50 mg loading, then 2 mg/min) but there were still four more attacks of sustained VT/Vf that required cardioversions.

Two additional cardioversions for VT/Vf were required on postoperative day 1 (POD1) and one on POD2. There was no sustained VT/Vf on POD3. On POD4, discontinuation of lidocaine infusion was attempted, but a further sustained VT/Vf attack required 15 cardioversions, so lidocaine infusion was resumed. Later on that day, ECMO flow declined and marked hemoglobinuria was also noted. Plasma leakage and thrombi were found inside the ECMO oxygenator. ECMO support had to be continued because of the low LVEF (30%) and the high risk of sustained VT/Vf. We replaced the oxygenator immediately with a flow interruption of less than 30 seconds. The ECMO flow resumed and became stable, with no more hemoglobinuria.

On POD5, further surgery for cardiac tamponade was performed. Pericardial hematoma was evacuated and meticulous hemostasis was done at the aorta and the split sternum. On inspection, all coronary grafts were patent and there was a myocardial infarct of 2 cm in diameter over the territory of the distal diagonal artery (LAD branch). Electrolytes and hemoglobin were kept within normal limits. Over the next 3 days, there were no more sustained VT/Vf attacks. The patient was weaned from ECMO on POD8. Her hemodynamics remained stable under support with IABP and infusions of dopamine (3.3 µg·kg⁻¹·min⁻¹), lidocaine (3 mg/min), and nitroglycerin (1.7 µg·kg⁻¹·min⁻¹).

The patient was weaned from IABP on POD12, but pharmacologic support with dopamine (3.3 µg·kg⁻¹·min⁻¹), nitroglycerin (2 µg·kg⁻¹·min⁻¹), and lidocaine (1.6 mg/min) was maintained. These medications were all tapered off on POD26. She was weaned from mechanical ventilation and transferred to the cardiac surgery ward on POD30. She recovered and was discharged 3 months later after a rehabilitation program. During outpatient follow-up over 15 months, she had New York Heart Association function class I, with no more chest pain, and was free from the need for antiarrhythmia drugs. Her LVEF remained around 55%. Echocardiographic LVEF was 28% preoperatively, 28.9% on POD1, 33.6% on POD5, 48.6% on POD6, and 54.5% on POD20.

The patient received 40 cardioversions for sustained VT/Vf during the first 5 days postoperatively. Her pre- and postoperative ECGs were similar except for anterior ST elevation that disappeared on POD12. There were two clusters of sustained VT/Vfs (Figure). The first occurred within the first few postoperative hours, and the second was on POD4. Peak concentration of myocardial-bound creatine kinase (CK-MB; 1,309 U/L) followed the first cluster (Figure).

Discussion

Sustained VT/Vf after coronary artery surgery may be treated using electric cardioversion [3, 11], antiarrhythmic drugs [3, 11], elimination of etiologies [3, 11], electrophysiologic study (EPS)-guided antiarrhythmic (AA) therapy [3], or implantable cardioverter-defibrillators (ICD) [3, 11]. The use of a VAD [10] and ECMO [5, 9] to stabilize the hemodynamics may be required. ECMO is usually used for temporary cardiogenic shock such as myocardial stunning and favorable results have been shown in post-CABG patients [7–9]. In this case of unanticipated sustained VT/Vf, the major reason for choosing ECMO was its easy set-up at the bedside during an emergency [8]. ECMO stabilizes the hemodynamics to allow the search for difficult
Extracorporeal Membrane Oxygenation for Ventricular Tachyarrhythmia

Etiologies under emergency conditions and also provides a safe bridge to another treatment modality such as EPS-guided AA therapy, VAD, ICD, or heart transplantation [5, 8]. Under ECMO-stabilized hemodynamics, we had time to investigate the etiology of sustained VT/Vf that might have included perioperative myocardial ischemia, focal infarction scarring, reperfusion effect, and hemodynamic instability [1–4].

The first cluster of VT/Vf attacks could be explained as the effects of ischemia, infarction, and reperfusion. Post-CABG VT/Vf might be a sign of myocardial ischemia [2, 3]. Cardiac tamponade and graft defects might cause and aggravate myocardial ischemia. The focal infarct at the diagonal artery could serve as a fixed anatomic substrate for arrhythmogenesis [3]. Patients with new-onset sustained VT are more likely to have prior myocardial infarction [1, 3]. When infarction is extensive, sustained VT/Vf might result from electrical instability secondary to LV remodeling or aneurysm formation [12]. However, when the infarct is focal, as in this case, ventricular arrhythmia develops via reperfusion [1, 3, 4]. Steinberg et al found that reperfusion was crucial in the pathogenesis of new-onset sustained VT in a prospective study that enrolled 382 patients [1]. They also found that the risk of sustained VT increased significantly when an infarct zone was revascularized, and that when an infarct was not reperfused, VT was very unlikely to develop during the in-hospital period [1]. Reperfusion by CABG might restore electrical function in the hibernating myocardium around an infarct and, thus, create reentrant circuits for the genesis of VT [1, 4].

The second cluster on POD4 in this patient might have resulted from LV dysfunction or premature withdrawal of lidocaine. Preoperative low LVEF and hemodynamic instability (the need for IABP) are associated with the development of sustained VT/Vf after CABG [2]. This patient needed IABP for low LVEF. LV dysfunction led to coronary hypoperfusion and then aggravated the myocardial ischemia. Ischemia-associated ventricular arrhythmia may have caused LV dysfunction again and, thus, created a vicious cycle. Lidocaine re-infusion and ECMO replacement broke this vicious cycle and the cluster of VT/Vf attacks was diminished.

The peak CK-MB concentration (1,309 U/L) on POD2 could be explained by the repeated electric cardioversions [13]. This high CK-MB concentration did not necessarily indicate extensive myocardial infarction. The infarct, which was visualized during the repeated surgery, was focal with a diameter of 2 cm. The reversible nature of the heart failure also suggested an absence of extensive post-infarction scarring. The baseline ECG Q-waves might imply that the infarct existed before the CABG. The repaired graft defect was in the territory of the RCA that had little anatomical relationship with the infarct at the diagonal artery (LAD branch). The focal infarct probably contributed to generate sustained VT/Vf [1–3].

ECMO played a pivotal role in the treatment of this patient with sustained VT/Vf after CABG. Lidocaine and amiodarone, as the standard treatment for post-

Figure. Myocardial-bound creatine kinase (CK-MB) concentration and ventricular tachycardia/ventricular fibrillation (VT/Vf) frequency in the postoperative period. ECMO = extracorporeal membrane oxygenation.
CABG VT/ Vf [3, 11], did not produce an immediate response, so we decided to set up ECMO promptly at bedside for cardiac rescue. Under ECMO support and with removal of pro-arrhythmic factors, lidocaine and amiodarone successfully suppressed sustained VT/ Vf in this case. If they had not suppressed the arrhythmia, procainamide could have been added next [11].

This patient developed unanticipated sustained VT/ Vf attacks during the post-cardiopulmonary bypass period. After repeated defibrillation, high doses of inotropic agents and IABP failed to restore hemodynamics. ECMO was needed for emergency cardiac support. The LVEF improved and VT/ Vf became less frequent under ECMO support. The ECMO fulfilled its role as a transient support and was weaned off on POD8.

In conclusion, this patient had profound heart failure due to sustained VT/ Vf. The possible etiologies included myocardial stunning, ischemia, infarction, and reperfusion. To stabilize the hemodynamics, ECMO was chosen for its simplicity and rapidity of set-up. It bridged this patient successfully to recovery.

References