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Acute myocardial infarction complicated by cardiogenic shock: an algorithm based ECMO program can improve clinical outcomes.

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Abstract

Objective: Extracorporeal membrane oxygenation (ECMO) in our institution resulted in near total mortality prior to the establishment of an algorithm-based program in July 2010. We hypothesized that an algorithm based ECMO program improves the outcome of patients with acute myocardial infarction complicated with cardiogenic shock.

Methods: Between March 2003 and July 2013, 29 patients underwent emergent catheterization for acute myocardial infarction due to left main or proximal left anterior descending artery occlusion complicated with cardiogenic shock (defined as systolic blood pressure < 90mmHg despite multiple inotropes, +- balloon pump, lactic acidosis). Of 29 patients, 15 patients were before July 2010 (Group 1, old program), 14 patients were after July 2010 (Group 2, new program).

Results: There were no significant differences in the baseline characteristics, including age, sex, coronary risk factors and left ventricular ejection fraction, between the two groups. Cardiopulmonary resuscitation prior to ECMO was performed in 2 cases (13%) in Group 1 and 4 cases (29%) in Group 2. ECMO support was performed in 1 case (6.7%) in Group 1 and 6 cases (43%) in Group 2. The 30-day survival of Group 1 vs. Group 2 was 40% vs. 79% ($p = 0.03$), and one-year survival rate was 20% vs. 56% ($p=0.01$). The survival rate for patients who underwent ECMO was 0% in Group 1 vs. 83% in Group 2 ($p = 0.09$). In Group 2, the mean duration on ECMO was 9.8 ± 5.9 days. Of the 6 patients who required ECMO in Group 2, 100% were successfully weaned off ECMO or were bridged to ventricular assist device implantation.

Conclusions: Initiation of an algorithm based ECMO program improved the outcomes in patients with acute myocardial infarction complicated by cardiogenic shock.

(Word count of abstract: 311)

Key words: cardiac surgery, extracorporeal membrane oxygenation, acute coronary syndrome

Introduction

Cardiogenic shock occurs in 5 - 10% of the patients with acute myocardial infarction (AMI), and its mortality is reported to be 40 - 50% (1-3). AMI caused by left main (LM) occlusion or proximal left anterior descending artery (pLAD) occlusion carries an increased risk of mortality, compared to mid to distal left anterior descending artery, circumflex, or right coronary artery occlusion (4, 5). The volume of the myocardium at risk is greater in LM and pLAD lesions than in the others, which can lead to hemodynamic instability with worse outcome (5, 6). Inotropes, vasopressors, and intra-aortic balloon pump (IABP) have been used as the first line support for the hemodynamics during emergent revascularization, either by percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). Recently, reports of using venoarterial extracorporeal membrane oxygenation (VA-ECMO) to stabilize the hemodynamics during the period of evolving AMI have been published (7, 8). In our institution, a new ECMO program was started in July 2010, which consists of a dedicated intensive care unit (ICU), multidisciplinary providers (intensivists, mid-level providers, nurses, and perfusionists), educational programs (didactic sessions, hands-on training, and competency tests), and the introduction of new technologies and the development of treatment algorithms. We hypothesized that our new ECMO program improved the outcome of patients with acute myocardial infarction caused by LM or pLAD lesion complicated by cardiogenic shock.

Methods

Patients: Using our catheterization laboratory database, patients with AMI due to left main or proximal left anterior descending artery occlusion complicated with cardiogenic shock were identified. AMI was defined as prolonged (> 30 minutes) chest pain or equivalent symptoms and electrocardiographic changes (9). Cardiogenic shock was defined as systolic blood pressure < 90 mm Hg for more than 30 to 60 minutes or the need for escalating inotropes, vasopressors, or IABP to maintain systolic blood pressure > 90 mm Hg. Demographics, hemodynamics and biochemical values were collected retrospectively from patients' charts and other databases after approval of internal review board. The survival status was obtained from a Social Security Death Index search. Between March 2003 and July 2013, 29 adult

patients (age > 18 years) underwent emergent catheterization for AMI due to LM or pLAD occlusion, complicated with cardiogenic shock. Patients who had coronary artery bypass surgery in the past, patients with circumflex lesion or mid to distal LAD lesion which was considered to be the culprit of the cardiogenic shock, were excluded. The patients were divided into two groups: Group 1 before July 2010 (old program) and Group 2 after July 2010 (new program).

The Vasoactive-inotropic score was calculated using the following formula: dopamine [ug/kg/min] + dobutamine [ug/kg/min] + 10,000 x vasopressin [U/kg/min] + 10 x milrinone [ug/kg/min] + 100 x epinephrine [ug/kg/min] + 100 x norepinephrine [ug/kg/min]) (10).

Indication for ECMO: ECMO consult was obtained if the patient had persistent profound cardiogenic shock, despite high doses of multiple intravenous inotropes and vasopressors, and IABP/Impella support, and/or ventricular tachycardia/fibrillation refractory to anti-arrhythmic medications and cardioversion. Profound cardiogenic shock was defined as systolic blood pressure < 90mmHg despite fluid resuscitation and being placed on high doses of multiple inotropes and evidence of decreased organ perfusion (increased lactate, low urine output, cool and diaphoretic extremities, and altered mental status). An Intensivist, cardiothoracic surgical fellow, or a mid-level provider rapidly assessed the patient for the indication. Inability to access the groin vessels, presence of a terminal disease process and do-not-resuscitate orders, un-witnessed cardiac arrest and downtime were contraindications for ECMO in patients with profound cardiogenic shock. Age over 75 was considered a relative contraindication. VA-ECMO was inserted either in the catheterization laboratory or at the bedside under fluoroscopy guidance.

ECMO (Equipment and cannulation technique): Since the new ECMO program was initiated (11), our ECMO system consisted of a centrifugal pump (Rota flow, Maquet, San Jose, California), PMP oxygenator (Quadrox D ,Maquet), and a heparin coated circuit with minimal access points. The portable CardioHelp system (Maquet) was also utilized to transport patients from outside facilities. Heparin coated cannulas (arterial 16 - 22 Fr, venous 18 - 24 Fr, Edwards Life Sciences, Irvine, CA) were inserted percutaneous through the femoral veno-arterial route using the Seldinger technique (12). The tip of the

arterial cannula is directed to the common iliac artery, and the tip of the venous cannula is directed into mid-right atrium. In all patients, a distal limb perfusion catheter (4 Fr or 5Fr) was inserted percutaneous in the superficial femoral artery for distal limb perfusion.

Management and Monitoring: Anticoagulation was started 6-12 hours after ECMO cannulation using heparin with a goal PTT level of 50 - 60 sec. Patients were sedated adequately using continuous infusions of midazolam and fentanyl; paralysis with vecuronium was given whenever necessary. The ECMO flow rate is set so as to achieve a goal of > 2.2 L/min/m^2. Inotropes (epinephrine, milrinone, dobutamine) were weaned off and IABP was discontinued after ECMO stabilization. Vasopressors (neosynephrine, vasopressin, norepinephrine) were used to maintain a mean arterial pressure of 60 - 80 mmHg. The Swan-Ganz catheter was removed once ECMO was initiated due to the risk of migration and possible perforation of the pulmonary artery. Monitoring of the heart was done periodically with a miniaturized hemodynamic transesophageal echocardiography probe (hTEE; ImaCor, Garden City, NY) to observe the cardiac function and the degree of distension and decompression (13). In addition, to ensure tissue perfusion, cerebral oximetry and lower limb oximetry was monitored routinely using near-infrared spectroscopy (INVOS, Covidien, Mansfield, MA or FORESIGHT, CAS Medical Systems, Branford, CT) (14). Patients who had cardiac arrest before the initiation of ECMO were actively cooled to 33°C within 3 hours, and maintained with a cooling device (Arctic Sun conductive cooling system [Medivance, Inc., Louisville, CO]). After 24 hours of cooling, patients were rewarmed 0.10-0.20° C every 1 hour. Blood products were transfused to maintain a hemoglobin of 8 - 10 g/dl, platelet count of 80,000 - 100,000 µl/ml. Ventilator setting was minimized; tidal volume of 4 - 6 mL/kg, frequency of 8 - 10 /min, positive end expiratory pressure of 5 cm H_2O, and an FiO_2 of 0.40 - 0.60. Circuit exchange was considered when arterial thrombus was seen in the circuit, or if there was unexplained hemolysis or inadequate oxygenation. Discontinuation of ECMO and withdrawal of care were discussed with the family if irreversible neurological damages were confirmed by head CT scan and neurological consultation.

Weaning from ECMO and removal: We considered weaning from ECMO when (1) the oxygenation was adequate on 50% FiO2 from ECMO and ventilator, (2) volume status is optimized, (3) resolution of

pre-ECMO organ dysfunction, and (4) afebrile (15). The anticoagulation was titrated to a PTT level of 60 to 70 seconds to avoid thrombotic complications while decreasing ECMO flow during the weaning trial (15). The weaning trials were done over a 4-6 hour period, which consists of 4 stages; the heart function and volume status was monitored continuously with the hTEE probe as described previously. ECMO was removed in the operating room with an open repair. Thrombectomy and/or a patch repair of the artery with a saphenous vein are done if required. A layer of fascia was closed over the vessels, the skin was left open, and a vacuum assisted closure device (VAC) was placed, as the wound will not be completely sterile. If there was no recovery of the myocardium 7 to 10 days after ECMO placement, and the patient was not a candidate for ventricular assist device, total artificial heart, or a transplant, an end of life discussion was held with palliative care support.

Statistical analysis: Statistical analyses were conducted using R-environment (R version 3.0.2, R Foundation for Statistical Computing, Vienna, Austria). Continuous variables were expressed as mean ± standard deviation (SD), and compared with standard t-test. Categorical variables were expressed as counts and percentages, and Chi-square test was used for comparisons**.** Kaplan-Meier survival curves were generated to assess survival, and the survival was compared between the groups using the log-rank test. The end-point for the survival analysis was death for any reason. P value less than 0.05 was considered significant.

Results

Baseline characteristics: There were 15 patients were before July 2010 (Group 1, old program) and 14 patients after July 2010 (Group 2, new program). There were no significant differences in the baseline characteristics, including age, sex, body-mass index, or coronary risk factors between the two groups (Table 1). Left main disease was the culprit lesion in 2 patients in Group 1 and 3 patients in Group 2. The remainder of patients had a proximal LAD lesion. 6 patients in Group 1 and 5 patients in Group 2 received at least one episode of CPR prior to or within the catheterization laboratory. ECMO support was performed in 1 case (6.7%) in Group 1 and 6 cases (43%) in Group 2.

Initial treatment: Treatments for the two groups are summarized in Table 2. 11 patients in Group 1 and

9 patients in Group 2 underwent emergency PCI, and 4 patients in Group 1 and 3 patients in Group 2 underwent emergency CABG. One patient in Group1 underwent mitral valve replacement and VSD repair at the time of CABG. Of the 7 patients that required ECMO support, ECMO was initiated just before revascularization in 2 patients, just after revascularization in 2 patients. 3 patients were placed on ECMO more than 24 hours after revascularization.

ECMO data: ECMO support was performed in 1 case (6.7%) in Group 1 and 6 cases (43%) in Group 2 ($p < 0.05$). The indications for ECMO were E-CPR in 3, unstable hemodynamics/high dose inotropes in 3, and recurrent ventricular tachyarrhythmia in 1. The patients who were placed on ECMO in Group 2 had a higher vasoactive-inotropic score compared to the non-ECMO patients (104.4 ± 122.0 vs. 31.9 ± 44.7 $p = 0.14$). Although not statistically significant, lab values showed a trend towards improvement within 24 hours after initiation of ECMO (Table 3). In Group 2, the mean duration on ECMO was 9.8 ± 5.9 days (1 - 18 days). There were no deaths among the patients who underwent ECMO in Group 2. The complications that occurred during ECMO support are summarized in Table 4.

Survival results: The 30-day survival rate of Group 1 vs. Group 2 was 40% vs. 79% ($p = 0.03$, Figure 1). The cause of death in each group is listed on Table 5. All of the 6 patients who required ECMO in Group 2 were successfully weaned off ECMO; 2 patients were bridged to implantation of left ventricular assist device, and 4 patients had total cardiac function recovery. The survival rate of Group 1 vs. Group 2 who underwent ECMO was 0% vs. 83% ($p = 0.09$, Figure 1). In Group 2, among the 5 patients who had CPR, 3 patients were placed on ECMO, 1 patient had withdrawal of care because of age (87 years old), and another patient had withdrawal of care because of the prolonged CPR before arrival to our institution. Kaplan-Meier survival and log-rank analysis showed significantly improved mid-term survival in Group 2 with a mean follow-up period of 11.8 ± 16.1 months ($p = 0.01$, Figure 2). The 1-year survival of Group 1 and Group 2 was, 20.0% and 56%, respectively.

Discussion

The main findings of the present study are (1) that an algorithm based ECMO program with dedicated personnel improved short term and mid term survival in patients who had cardiogenic shock due

to left main or proximal LAD, (2) initiation of ECMO was able to improve the hemodynamics and the organ dysfunction due to cardiogenic shock, and (3) ECMO was performed and managed with a relatively low complication rate.

Even though the target vessel is revascularized with PCI or CABG, stunned or hibernating myocardium may develop and it takes 7 - 10 days to recover. Reperfusion injury may further deteriorate the cardiac function. Poor hemodynamics during this period will reduce coronary perfusion with resultant poor cardiac function and pulmonary edema. The subsequent result is poor oxygen supply to not only the myocardium but also to other vital organs, resulting in multiple organ failure. Adequate hemodynamic support with ECMO will improve oxygen and blood supply and theoretically improve survival. In addition, ECMO will reduce the myocardial work-load and reduce oxygen consumption by unloading the heart. Intervention to the LM or pLAD may be difficult due to arrhythmias and hypotension; however, supporting the hemodynamics with ECMO will enable the cardiologist to achieve complete revascularization. Inotropes and vasopressors may cause vasoconstriction, tissue ischemia and potentially fatal arrhythmias, but in the majority of times these medications can be discontinued or decreased within 24 hours of the initiation of ECMO. We also discontinue IABP after the initiation of ECMO, as we believe there are no benefits in the use of IABP during ECMO. Park et al. reported that the combined use of IABP with ECMO did not improve in-hospital survival (16). IABP may increase coronary blood flow during diastole, but will cause intermittent aortic occlusion, which competes with the flow from ECMO.

AMI with cardiogenic shock caused by LM, pLAD lesion carries a high mortality. Trzeciak et al. reported that the in-hospital mortality when the infarct related artery was LM, LAD, LCx, RCA was 64.7%, 41.0%, 36.0%, and 30.8%, respectively. The 12-month mortality was 77.7%, 58.2%, 55.1%, and 45.0%, respectively (4). Although in their report, the LAD itself was not significantly related to increased mortality, cardiac arrest during hospitalization was most commonly noted in the LM and LAD groups (4). Brener et al. reported that patients who had AMI caused by proximal LAD had higher frequency of having a Killip class > 1 heart failure, ejection fraction < 40%, and mortality, compared to

patients who had AMI due to a mid LAD lesion (5). This result was thought to be due to larger infarcts in patients with proximal LAD occlusion.

Initiation of an algorithm based ECMO program in our institution resulted in an improved outcome in patients with acute myocardial infarction complicated by cardiogenic shock due to LM or pLAD lesions. We believe the patients who were placed on ECMO would not have survived traditional treatment. Although the numbers of our patients are low, the 30-day survival, hospital discharge rate, 1 year survival were significantly better in the new program; 40% vs. 79% ($p = 0.03$) and 20.0% vs. 56% ($p = 0.01$).

Increased referrals, standardized insertion and decannulation techniques, protocol-based management and weaning of ECMO, were the keys to improved patient outcome. Except for two patients in Group 2, all of the patients who had profound cardiogenic shock or an episode of cardiac arrest were placed on ECMO. These 2 patients were referred and evaluated for ECMO, but due to family wishes and due to the likelihood of having irreversible brain injury, it was decided to withdraw care. Continuous efforts to notify cardiologists about the ECMO program and keeping a good relationship, rapid response to 'code blue' enabled the increase in referrals. 100% of patients who were placed on ECMO were successfully weaned off and were either bridged to a ventricular assist device or recovered. This was possible because of the relatively low rate of major complications observed during ECMO. Previous studies reported a leg ischemia of 4 - 34%, renal failure requiring dialysis of 40%, bleeding complications in 10%, neurological injury of 10 - 46% (Table 6). None of our patients included in the study had a limb ischemia, neurological injury or required hemodialysis during ECMO support. Routine use of the distal perfusion catheter, monitoring the lower extremity saturation, open repair of the vessels, and thrombectomy if needed enabled us to eliminate limb ischemia. We do not recommend the use of smaller femoral arterial cannulas (< 16 Fr) (17), as it may lead to increased shear stress and hemolysis, leading to possibly both liver and kidney failure. In addition, insufficient flow will result in inadequate decompression of the heart, which will decrease the chance of the heart to recover. We have not had minor or major infection at the insertion site after decannulation, since the use of routine VAC dressing on

the groin incision. Previous studies regarding treatment of cardiogenic shock due to AMI reported ECMO weaning rate of 24 - 80%, hospital discharge rate of 34 - 76% (Table 5). Although the number of patients included in our study is low, and the fact that our study included the patients who were supported with ECMO for pLAD and LM lesions, the results were better than most reports.

Limitations of this study

This study was performed at a single center with retrospective data collection, and the number of cases was relatively small. There were new physicians that were introduced into our practice and the medical treatment besides the ECMO program may have had a substantial difference between the two groups that could have resulted in the differences in the outcomes. To further elucidate the benefit of ECMO in the treatment of AMI complicated with cardiogenic shock, a randomized, multicenter, controlled, large-scale studies are needed to investigate the benefit of ECMO.

Conclusion

The new ECMO program significantly improved patient survival that developed cardiogenic shock due to AMI caused by left main or proximal LAD lesion. The initiation of ECMO enabled to improve the hemodynamics and organ perfusion with relatively small number of complications.

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Table 1: Patients' baseline characteristics. Data are expressed with mean ± standard deviation or number (percentage).

□	Group 1 (n=15)	Group 2 (n=14)	p-value
Age (y)	63.5 ± 13.9	63.4 ± 18.4	0.99
Sex (male)	12 (80%)	9 (64%)	0.34
Body mass index (kg/m_2)	27.7 ± 6.2	29.3 ± 7.0	0.52
Diabetes	8 (53%)	5 (36%)	0.34
Family history of coronary disease	6 (40%)	3 (21%)	0.28
Hyperlipidemia	4 (27%)	5 (36%)	0.60
Smoking	11 (73%)	9 (64%)	0.60
Hypertension	7 (47%)	7 (50%)	0.86
Previous MI	5 (33%)	2 (14%)	0.23
Renal injury (creatinine >1.5 g/dl)	6 (40%)	2 (14%)	0.12
Left main disease	2 (13%)	3 (21%)	0.56
Intra-aortic balloon pump support	14 (93%)	10 (71%)	0.12
Cardiopulmonary resuscitation	6 (40%)	5 (38%)	0.81
Ejection fraction (%)	30.4 ± 17.5	30.4 ± 19.0	1.00

Table2: Treatment for the two groups

	Group 1	Group 2	p-value
Percutaneous coronary intervention	11 (73%)	9 (64%)	0.60
Coronary artery bypass graft	2 (13%)	4 (29%)	0.56
ECMO support	1 (6.7%)	6 (42%)	0.02
PCI with ECMO support	1 (6.7%)	0	
CABG after ECMO	0	1 (7.1%)	

Table 3. Laboratory data of patient on ECMO. Data are expressed mean ± standard deviation.

	Pre ECMO	24hr	Pre DC	P-value (pre vs. 24hr)	P-value (pre vs. DC)
AST (IU/L)	189 ± 301	216 ± 268	127 ± 169	0.89	0.33
ALT (IU/L)	341 ± 623	142 ± 239	53 ± 28	0.31	0.35
Total bilirubin (mg/dl)	0.8 ± 0.4	1.2 ± 0.7	1.2 ± 0.4	0.40	0.14
Creatinine (g/dl)	1.2 ± 0.4	1.0 ± 0.2	0.9 ± 0.2	0.13	0.13
Lactate (mmol/L)	6.2 ± 5.0	1.6 ± 0.8	2.4 ± 2.2	0.13	0.25
Vasoactive-inotropic score (median, 25th – 75th percentile)	104 ±121 (42, 17.5 - 234)	1.3 ± 0.1 (0, 0 – 4)		0.09	

ALT: alanine aminotransferase; AST: aspartate aminotransferase, DC: decannulation.

Table 4: Outcome of patients on ECMO (Group 2)

ECMO length	9.8 ± 5.8 (1 - 18 days)
Bridge to ventricular assist device	2 (33%)
Bridge to recovery	4 (66%)
Death on ECMO	0 (0%)
Sepsis	1 (17%)
Limb ischemia	0 (0%)
Neurological injury	0 (0%)
Gastrointestinal bleed	1 (17%)
Other major bleeding	1 (17%)
Acute renal failure	0 (0%)

Table 5: Cause of Death

	Group 1	Group 2
cardiogenic shock	4	0
arrhythmia	3	1
respiratory failure	1	0
bleeding	1	1
neurological injury	0	1

Table 6: Previous studies regarding ECMO support for cardiogenic shock due to AMI.

Study	Year	N	Wean off ECMO*	30 day survival	Hospital discharge rate	1 yr. survival	Dialysis	Bleeding	Neuro**	Infection	Sepsis	Leg ischemia (distal limb perfusion)
Chen (18)	2006	36	69%		48%		53% ***					
Sheu (19)	2010	46	78%	61%				17%				7% (when ischemic)
Chung (20)	2011	20	70%		50%		40%	40%	20%		20%	
Bermudez (21)	2011	33		64%		48%	39%		12%	24%		15% (when ischemic)
Kim (22)	2012	27	82%	63%	59%		37%	15%				4% (routinely placed)
Tsao (23)	2012	33	82%		67%	64%			0%			0% (n/a)******
Sakamoto (24)	2012	98	55%		33%				3%			7% (when ischemic)
Wu (7)	2013	35	63%		40%	29%	46%		46%			9% (n/a)
Tang (25)	2013	21		76%	76%		5% ***	10%	15%		14%	
Demondion (26)	2014	77	48%	39%	34%		46%	21%	3%			9% (routinely placed)
Aoyama (27)	2014	38			37%		37%	89%		50%		34% (n/a)
Current	2014	6	100%	83%	67%	56%	0%	33%	0%	17%	17%	0% (routinely placed)
Total		470	64%	56%	44%	47%	41%*****	31%	10%	36%	17%	10%

* includes patients who were bridged to transplant or mechanical assist device

** neurological injury

*** percentage of “renal failure”, **** percentage of “dialysis”, ******information not available

Figure Legends

Figure 1. Summary of the outcome of the patients with cardiogenic shock due to AMI

Figure 2. Kaplan Meier survival curve of the patients with cardiogenic shock due to AMI

Author Contributions

Shinya Unai, MD: concept/design, data collection, data analysis/interpretation, drafting article

Daizo Tanaka, MD: concept/design, critical revision of the article, approval of article

Nicholas Ruggiero, MD: concept/design, critical revision of the article, approval of article

Hitoshi Hirose, MD: concept/design, data collection, data analysis/interpretation, critical revision of the article, drafting article, approval of article

Nicholas C. Cavarocchi, MD: concept/design, critical revision of the article, approval of article

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