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Clinical Research

Conversion to Veno-arteriovenous Extracorporeal Membrane Oxygenation for Differential Hypoxia

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Background: Patients who require initial venoarterial extracorporeal membrane oxygenation (VA ECMO) support may need to undergo veno-arteriovenous ECMO (VAV ECMO) conversion. However, there are no definitive criteria for conversion to VAV ECMO. We report 9 cases of VAV ECMO at Gyeongsang National University Changwon Hospital, Gyeongsang National University College of Medicine.
Methods: Of 158 patients who received ECMO support between January 2017 and June 2019, 82 were supported by initial VA ECMO. We retrospectively reviewed the medical records of 9 patients (7 men and 2 women; age, 53.1±19.4 years) who had differential hypoxia and required VAV ECMO support. Percutaneous transaortic catheter venting was used to detect the differential hypoxia.
Results: Among the 82 patients who received VA ECMO support, 9 (10.9%) had differential hypoxia and required conversion to VAV ECMO support. The mean time from VA ECMO support to VAV ECMO support and the mean duration of the VAV support were 2.1±2.2 days and 1.9±1.5 days, respectively. The average peak inspiratory pressure before and after VAV ECMO application was 23.89±3.95 cmH_2O and 20.67±5.72 cmH_2O, respectively, decreasing by an average of 3.2±3.5 cmH_2O (p=0.040). The PaO_2/FiO_2 ratio was kept below 100 mm Hg in survivors and non-survivors for 116±65.4 and 250±124.9 minutes, respectively (p=0.016). Six patients underwent extracorporeal cardiopulmonary resuscitation, of whom 4 survived (67%). The overall survival rate of patients who underwent conversion from VA ECMO to VAV ECMO was approximately 56%.
Conclusion: Rapid detection of differential hypoxia is required when VA ECMO is applied, and efficient conversion to VAV ECMO may be critical for patient survival.

Keywords: Veno-arteriovenous, Extracorporeal membrane oxygenation, Differential hypoxia

Introduction

Venovenous (VV) and venoarterial (VA) extracorporeal membrane oxygenation (ECMO) is applied to patients with an increased risk of death from cardiopulmonary disease [1,2]. The principle of ECMO is to withdraw a portion of the patient's blood from the body, oxygenate it, and then return it into circulation. There are 2 types of ECMO: VA ECMO, which is commonly used for patients with various etiologies of cardiogenic shock, and VV ECMO, which is commonly used for patients with respiratory failure [3].

Peripheral VA ECMO is typically performed using the femoral vessels [4]. Using femoral-femoral VA ECMO in patients with respiratory failure accompanied by circulatory collapse can lead to upper body hypoxemia (i.e., differential hypoxia or Harlequin syndrome) [5]. When patients receiving peripheral VA ECMO are in respiratory failure, the aortic valve opens (if the heart contraction is sufficient) and allows poorly oxygenated blood from the native heart to compete with the retrograde saturated blood flow from the ECMO pump. In this situation, poorly oxygenated blood may be preferentially delivered to the myocardium and brain, leading to hypoxic injury [6]. Understanding the physiology of differential hypoxia during VA ECMO

and its clinical implications is important. If differential hypoxia is suspected, most clinicians choose an alternative cannulation strategy [7].

At our center, if differential hypoxia was suspected in a patient while using VA ECMO, we tried to resolve the patient's upper body hypoxia by converting from VA ECMO to veno-arteriovenous (VAV) ECMO. Although our patient sample was small, we report the results of our initial experience.

Methods

Study design and patient characteristics

This retrospective study was reviewed and approved by the ethics committee of Gyeongsang National University Changwon Hospital (approval no., GNUCH 2022-12-0001). The requirement for informed consent was waived because of the retrospective nature of the study.

The baseline patient characteristics are shown in Table 1. The records of 9 consecutive patients who underwent conversion to VAV ECMO after initial VA ECMO (82 patients) between January 2017 and June 2019 were reviewed. Two of the 9 patients were female. The underlying pathologies included ST elevation myocardial infarction, non-ST elevation myocardial infarction, heart failure, and acute kidney injury. Five patients had a medical history of chronic obstructive pulmonary disease. All patients experienced acute cardiogenic shock and pulmonary edema.

Installation and management of extracorporeal membrane oxygenation

VA ECMO (MAQUET Cardiopulmonary AG, Hirrlingen, Germany) was established peripherally through the femoral vessels using an 18F–20F Fem-FlexII femoral arterial cannula (Edwards Lifesciences LLC, Irvine, CA, USA) and an 18F–24F Fem-FlexII femoral venous cannula (Edwards Lifesciences LLC). All procedures were performed using the Seldinger technique. Distal perfusion using a 7F. Radifocus Introducer II (Terumo Corp., Tokyo, Japan) was also performed routinely in all patients to prevent limb ischemia. The target ECMO flow was 2.5 L/min/m^2 of body surface area.

The indications for percutaneous transaortic catheter venting (TACV) using a 5F–6F pigtail catheter (PIG Performa; Merit Medical, South Jordan, UT, USA) for left ventricular (LV) decompression were: (1) severe LV dysfunction (LV ejection fraction [LVEF], <25%), with persistent

Table 1. Characteristics of patients who underwent conversion to VAV ECMO

Characteristic	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9
Age (yr)	62	55	50	54	63	41	53	77	70
Sex	Male	Male	Male	Male	Male	Male	Male	Female	Female
Body surface area (kg/m^2)	1.95	1.95	1.74	1.75	1.72	2.14	1.77	1.82	1.88
Diagnosis	STEMI	AKI	STEMI	NSTEMI	NSTEMI	HF (grade IV TR)	STEMI	HF	STEMI
Comorbidities	Hypertension	CVA, COPD	Hypertension	DM, COPD	DM, COPD	Hypertension, DM, CRF	Hypertension	Hypertension, DM, COPD, CRF	Hypertension, DM, COPD
Reason for VAV ECMO	Pulmonary edema	Pulmonary edema, aspiration pneumonia	Pulmonary edema	Pulmonary edema	Pulmonary edema, pneumothorax	Pulmonary edema	Pulmonary edema	Pulmonary edema	Pulmonary edema

VAV ECMO, veno-arteriovenous extracorporeal membrane oxygenation; STEMI, ST-segment elevation myocardial infarction; AKI, acute kidney injury; NSTEMI, non-ST-segment elevation myocardial infarction; HF, heart failure; TR, tricuspid valve regurgitation; CVA, cerebrovascular accident; COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; CRF, chronic renal failure.

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pulmonary edema on chest radiography or (2) LV asystole with/without mitral insufficiency on transthoracic echocardiography (TTE). The TACV catheter was connected to the venous limb of the ECMO circuit.

The criteria for applying VAV ECMO and the target protective ventilator settings are shown in Fig. 1. First, if the ratio of arterial oxygen tension (PaO_2) to fraction of inspired O_2 concentration (FiO_2) (i.e., PaO_2/FiO_2) was <100 mm Hg, VAV conversion was performed so that protective ventilator settings could be applied even if the patient's blood gas analysis showed no hypoxia. In addition, blood gas analysis using the TACV catheter is an important criterion for VAV ECMO. VAV ECMO was applied to patients whose LV blood gas analysis showed hypoxia (PaO_2 <70 mm Hg) despite full ventilator settings. If patients did not undergo TACV, and their blood gas analysis via the right radial artery showed hypoxia, VAV ECMO was applied. Excessive position changes and prone positioning were avoided due to the risk of kinking the catheters or ECMO tubing line and altering the ECMO flow rates.

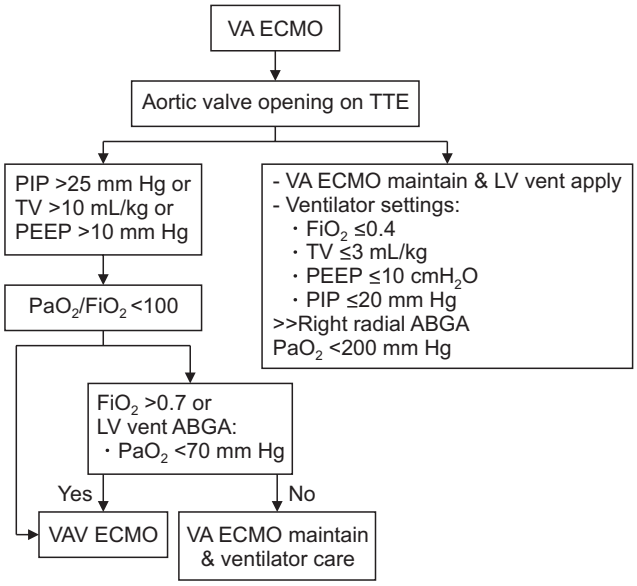

Fig. 1. Proposed algorithm for the conversion from veno-arterial extracorporeal membrane oxygenation (VA ECMO) to veno-arteriovenous extracorporeal membrane oxygenation (VAV ECMO) in our department. TTE, transthoracic echocardiogram; TV, tidal volume; PIP, peak inspiratory pressure; PEEP, positive end-expiratory pressure; FiO_2, fraction of inspired O_2 concentration; PaO_2, arterial oxygen tension; LV, left ventricular; ABGA, arterial blood gas analysis.

An additional venous return catheter was inserted through the right jugular vein in 7 patients and into the femoral vein in the remaining 2 patients using the Seldinger technique.

After application of VAV ECMO, the mean blood flow ratio of the supplying venous cannula was maintained at 91.8%±47.3% by partial clamping of the arterial line using a Transonic ELSA Monitor (Transonic Systems Inc., Ithaca, NY, USA).

The weaning criteria for VAV ECMO are shown in Fig. 2. During VAV ECMO, the differential hypoxia should be corrected by using protective ventilator settings and by the formation of an arterial pulse pressure.

Statistical analysis

Data were collected from all enrolled patients. The baseline characteristics of the participants are presented as

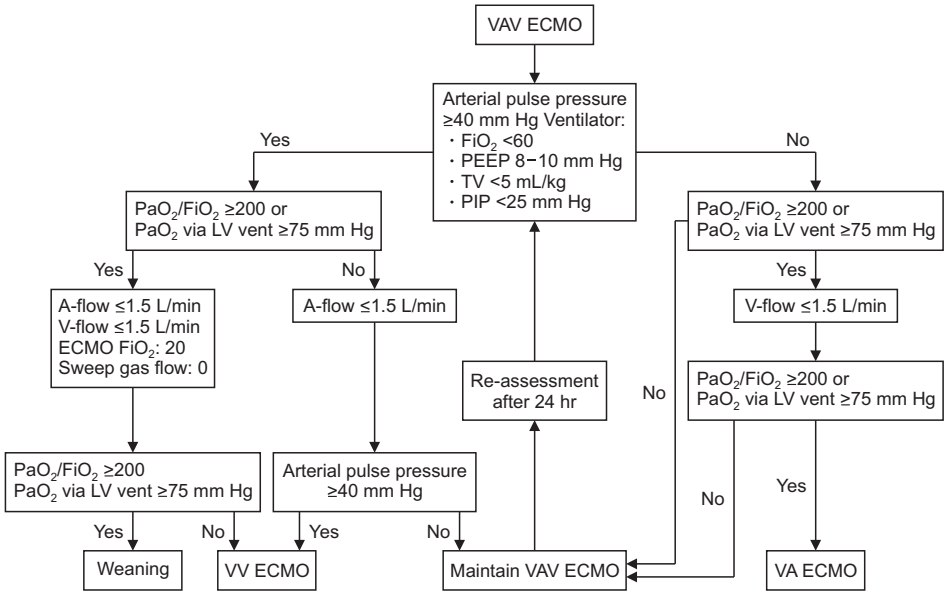

Fig. 2. Proposed weaning algorithm for veno-arteriovenous extracorporeal membrane oxygenation (VAV ECMO) in our department. VV, venovenous; VA, venoarterial; PaO_2, arterial oxygen tension (mm Hg); FiO_2, fraction of inspired O_2 concentration; LV, left ventricular; PEEP, positive end-expiratory pressure (cmH_2O); TV, tidal volume (mL/kg); PIP, peak inspiratory pressure (mm Hg); A-flow, arterial flow (L/min); V-flow, venous flow (L/min).

mean±standard deviation values for continuous variables. The Mann-Whitney U test was used to analyze continuous variables. IBM SPSS ver. 24.0 (IBM Corp., Armonk, NY, USA) was used for all statistical analyses, and p-values <0.05 were considered statistically significant.

Results

The patient characteristics are presented in Table 1. Seven patients were male, and all were converted from VA ECMO to VAV ECMO due to pulmonary edema.

There was a statistically significant difference in the ventilator FiO_2 levels between pre-VAV ECMO and post-VAV ECMO (0.87±0.19 versus 0.42±0.11, p<0.001). The average peak inspiratory pressure (PIP) before and after VAV ECMO application was 23.89±3.95 cmH_2O and 20.67±5.72 cmH_2O, respectively, and decreased by an average of 3.2±3.5 cmH_2O (p=0.040) (Table 2).

The effect of TACV on ECMO is shown in Table 3. The pre- and post-VAV ECMO mean arterial pressures were 45.2±14.2 mm Hg and 83.1±13.2 mm Hg, respectively (p< 0.001). There were significant increases in PaO_2 (64.2±5.9 mm Hg versus 166.1±51.1 mm Hg, p<0.001), PaO_2/FiO_2 (75.22±11.7 versus 435.3±150.6, p<0.001), and LVEF (9.6%±12.0% versus 26.7%±10.8%, p=0.014) after VAV ECMO application.

The characteristics of survivors and non-survivors are presented in Table 4. The length of time that the PaO_2/FiO_2 ratio was kept below 100 mm Hg was 116±65.4 minutes and 250±124.9 minutes for survivors and non-survivors, respectively (p=0.016). The PIP was lower in survivors than in non-survivors (18.8±4.4 cmH_2O versus 23±2.9 cmH_2O, p=0.032). Six patients underwent extracorporeal cardiopulmonary resuscitation (ECPR), and 4 of those patients sur-

Table 2. Comparison of mechanical ventilator settings for pre-VAV ECMO and post-VAV ECMO

	Mechanical ventilator settings of pre-VAV ECMO				Mechanical ventilator settings of post-VAV ECMO			
	FiO_2	PEEP (cmH_2O)	Tidal volume (mL/kg)	PIP (cmH_2O)	FiO_2	PEEP (cmH_2O)	Tidal volume (mL/kg)	PIP (cmH_2O)
Patient 1	0.75	8	5	27	0.3	8	5	28
Patient 2	0.8	8	5.7	28	0.4	10	5.6	22
Patient 3	1.0	10	10	24	0.45	8	6.1	20
Patient 4	1.0	10	0.9	26	0.3	10	1	26
Patient 5	0.45	5	6.7	15	0.3	5	2.8	10
Patient 6	0.8	10	5	26	0.6	10	3.5	26
Patient 7	1.0	10	7.8	25	0.5	10	4.5	15
Patient 8	1.0	7	6.3	23	0.4	10	4.4	20
Patient 9	1.0	8	3.6	21	0.55	8	2.9	19
Mean±SD	0.87±0.19	8.44±1.74	5.67±2.57	23.89±3.95	0.42±0.11	8.78±1.72	3.98±1.59	20.67±5.72
p-value					<0.001	0.666	0.077	0.040
Mean change in ventilator settings					-0.45±0.18		-1.69±1.65	-3.2±3.5

Values are presented as mean or mean±standard deviation, unless otherwise stated.
VAV ECMO, veno-arteriovenous extracorporeal membrane oxygenation; FiO_2, fraction of inspired O_2 concentration; PEEP, positive end-expiratory pressure; PIP, peak inspiratory pressure.

Table 3. Comparison of parameters before and after conversion to VAV ECMO

Variable	Pre-VAV ECMO	Post-VAV ECMO	p-value
Mean arterial pressure (mm Hg)	45.2±14.2	83.1±13.2	<0.001
Serum lactate (mmol/L)	8.8±7.9	5.1±4.7	0.297
PaO_2 (mm Hg)	64.2±5.9	166.1±51.1	<0.001
PaO_2/FiO_2	75.22±11.7	435.3±150.6	<0.001
LV ejection fraction (%)	9.6±12.0	26.7±10.8	0.014
PIP (cmH_2O)	23.89±3.9	20.67±5.7	0.040

Values are presented as mean±standard deviation.
VAV ECMO, veno-arteriovenous extracorporeal membrane oxygenation; PaO_2, arterial oxygen tension; FiO_2, fraction of inspired O_2 concentration; LV, left ventricular; PIP, peak inspiratory pressure.

Table 4. Characteristics of the survivors and non-survivors after conversion from VA ECMO to VAV ECMO

Variable	Age (yr)	Sex	Time from VA to VAV (hr)	LV vent (yes/no)	Duration of PaO_2/FiO_2 <100 (min)	PaO_2/FiO_2 (pre-VAV)	PaO_2/FiO_2 (post-VAV)	PIP (post-VAV)	ECPR (yes/no)	PCI (yes/no)	Duration of ECMO (day)	FiO_2 (post-VAV)	Tidal volume (mL/kg) (post-VAV)
Survivors													
Patient 2	62	M	16	Yes	40	76	420	23	Yes	No	4	0.4	5.6
Patient 3	50	M	68	Yes	60	100	200	22	Yes	Yes	14	0.45	6.1
Patient 5	63	M	26	Yes	180	133	760	12	No	Yes	14	0.3	2.8
Patient 7	54	M	16	No	120	61	360	17	Yes	Yes	4	0.5	4.5
Patient 9	72	F	38	Yes	180	69	472	20	Yes	Yes	24	0.55	2.9
Mean±SD or no.	60.2±8.6		32.8±21.7	4	116±65.4	90.7±32.8	442.4±204.8	18.8±4.4	4	4	12±8.4	0.44±0.1	4.38±1.5
Non-survivors													
Patient 1	62	M	18	Yes	240	101	500	26	Yes	Yes	4	0.3	5
Patient 4	54	M	20	Yes	120	62	410	24	Yes	Yes	3	0.3	1
Patient 6	41	M	120	No	420	76	226	23	No	No	10	0.6	3.5
Patient 8	78	F	145	Yes	220	68	298	19	No	No	14	0.4	4.4
Mean±SD or no.	58.8±15.5		75.8±66.3	3	250±124.9	76.8±17.2	358.5±120.9	23±2.9	2	2	7.8±5.2	0.4±0.1	3.48±1.8
p-value	0.905		0.413		0.016	0.556	0.286	0.032			0.413	0.556	0.556

Values are presented as number or mean±SD, unless otherwise stated.
VA, venoarterial; VAV, veno-arteriovenous; ECMO, extracorporeal membrane oxygenation; LV, left ventricular; PaO2, arterial oxygen tension (mm Hg); FiO_2, fraction of inspired O_2 concentration; PIP, peak inspiratory pressure (cmH_2O); ECPR, extracorporeal cardiopulmonary resuscitation; PCI, percutaneous coronary intervention; SD, standard deviation.

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vived (67%). The overall survival rate of patients who underwent conversion from VA ECMO to VAV ECMO was approximately 56% (5 of 9 patients).

Discussion

In this study, we investigated the benefit to patients of the effective and rapid detection of differential hypoxia during VA ECMO and the subsequent conversion to VAV ECMO to resolve the differential hypoxia. Because the number of patients who underwent VAV ECMO was small, multivariate regression analysis could not be used to remove confounding variables that might have affected the effectiveness of VAV ECMO. However, by converting to VAV ECMO, it was possible to apply protective ventilator settings and correct the differential hypoxia in patients.

Femoral-to-femoral VA ECMO does not always provide sufficient oxygenation to all internal organs. Blood from the native heart is combined with the retrograde flow of ECMO in a "mixing zone" [8,9]. Because it is difficult to determine the exact location of the mixing zone and the oxygen concentration of the blood around the mixing zone, there is a risk of heart and brain hypoxia and missed correction timing. Although the extent of the differential hypoxia is determined by the location of the mixing zone, blood gas analysis through a TACV catheter and a right radial artery is helpful in estimating the extent of the differential hypoxia. We performed TACV in 7 of the 9 patients who underwent VAV ECMO. At our center, the indications for TACV for LV unloading were: (1) narrow pulse pressure and/or severe LV dysfunction (LVEF <25%) with persistent pulmonary edema or (2) LV asystole with/without mitral insufficiency on TTE. Insufficient LV unloading is a main cause of unsuccessful LV recovery during VA ECMO. It is debatable whether atrial or ventricular unloading is more effective, especially in cases of LV asystole. Although atrial drainage can improve pulmonary edema, LV unloading is unlikely to be effective in the absence of mitral insufficiency in cases of LV asystole [10].

We used TACV and blood gas analysis through a right radial artery catheter for blood pressure monitoring and for the detection of differential hypoxia. Since the oxygen concentration of blood entering the left atrium and the left ventricle from the native lung can be directly measured by blood gas analysis via TACV, it is possible to estimate the oxygen concentration of the ejected blood from the native heart to the ascending aorta. Notably, if the mixing zone is near the sinotubular junction, brain hypoxia does not occur, but hypoxia of the cardiac muscle does take place, making it difficult for clinicians to detect differential hypoxia when using right radial artery blood gas analysis alone. In our center, TACV was not applied in patients with an ejection fraction >25%; therefore, the diagnosis of differential hypoxia was based on right radial artery blood gas analysis in most clinical situations.

The time between the recognition of differential hypoxia and the conversion to VAV ECMO averaged 176 minutes, with an average of 116 minutes for survivor cases and an average of 250 minutes for non-survivor cases (p=0.016). Once differential hypoxia was recognized, it was important to correct it as soon as possible. After conversion to VAV ECMO, return flow to the arterial and venous system was adjusted using a Transonic ELSA Monitor (Transonic Systems Inc.) based on heart function according to TTE results and PaO_2 levels (ventilator settings). At our center, the protective ventilator settings were: PaO_2 >200 mm Hg, tidal volume <5 mL/kg, positive end-expiratory pressure: 8–10 mm Hg, PIP <25 mm Hg, PaO_2 on LV vent arterial blood gas analysis >80 mm Hg.

Some studies have investigated methods to improve the differential hypoxia that can develop during VA ECMO. Stöhr et al. [11] found that patients undergoing ECMO therapy for acute respiratory distress syndrome showed a greater survival benefit with a conversion to VAV ECMO from VV or VA ECMO versus VV or VA ECMO alone. Moravec et al. [12] reported that 2 patients with cerebral hypoxemia during VA ECMO were successfully treated after conversion to VAV ECMO. Although the risk of hemorrhage is a disadvantage of VAV ECMO [13-15], no increased bleeding tendency was found in our patients.

In our study, several parameters were improved by identifying the differential hypoxia caused by VA ECMO and converting to VAV ECMO. After VAV ECMO conversion, the mean arterial pressure (p<0.001), PaO_2/FiO_2 (p<0.001), and LVEF (p=0.014) showed improvement. However, since the number of patients in our study was small, confounding variables may have been involved. A larger sample size is needed to clarify the statistical significance of our results.

In addition, 6 patients underwent ECPR, and 4 of those patients survived (67%). Although the number of patients was small, this was considered a significant result. Marinacci et al. [16] reported that the rate of ECPR patient survival to hospital discharge was 25.5% (n=12) in a retrospective review of patients who underwent ECPR at a quaternary care center over a 7-year period.

This study had several limitations. The sample size of this single-center retrospective study was small. Therefore,

it is difficult to generalize the effect of VAV ECMO. Although the role of oxygenation in VAV ECMO is widely accepted, confounding variables may affect the improvement of various parameters or the survival rate benefit. A large-scale study is needed to obtain further scientific evidence and overcome the limitations of the present study.

In conclusion, despite the limitations of this study, the rapid correction of VA ECMO-related differential hypoxia played an important role in patient recovery. The effective conversion to VAV ECMO helped in the rapid correction of differential hypoxia in our patients.

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Conflict of interest

No potential conflict of interest relevant to this article was reported.

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