

Veno-Venous Extracorporeal Membrane Oxygenation for an Acute Type-B Aortic Dissection Compromised with Acute Respiratory Distress Syndrome: A Case Report

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ABSTRACT

Background: Type B aortic dissection (TBAD) is sometimes complicated by respiratory failure with the need for ventilation.

Case Presentation: A 51-year-old man who suffered from back pain was diagnosed with acute TBAD. On the third day of hospitalization, he complained of dyspnea and presented severe hypoxia and acidosis, then was transported to our hospital for intensive care. Computed tomography (CT) showed a diffuse infiltration shadow to be diagnosed with acute respiratory distress syndrome (ARDS). The mechanical ventilation could not improve hypoxia or hypercapnia. We decided on the care of veno-venous extracorporeal membrane oxygenation (VV-ECMO) and steroid pulse therapy. The pulmonary condition improved daily. VV-ECMO was discontinued on the fifth day and mechanical ventilation was removed. CT showed improvement in pulmonary infiltration shadow and non-progression in aortic dissection.

Conclusion: VV-ECMO should be regarded in patients with potentially reversible respiratory failure even if the basic illness is TBAD.

INTRODUCTION

Acute aortic dissection is itself a disease with high mortality. Some of the cases are complicated by oxygenation impairment. This respiratory failure is sometimes hard to be treated only with mechanical ventilation. We report a case of an acute type-B aortic dissection with acute respiratory distress syndrome (ARDS) that was treated by veno-venous extracorporeal membrane oxygenation (VV-ECMO).

CASE PRESENTATION

The patient was a 51-year-old man that suffered from back pain. The symptom was getting worse and worse. He was transported to the previous hospital by ambulance car. He was diagnosed with acute type-B aortic dissection by enhanced computed tomography (CT) scan and immediately admitted (Figure 1 A-3). The chest radiogram indicated pulmonary congestion, but there was no sign associated with ARDS in the CT images at admission (Figure 1 A-1, A-2). Pain control by morphine and treatment for hypertension by nitroglycerin were performed since admission. The patient complained of dyspnea on the next day of hospitalization, and the chest radiogram showed worse congestion. The additional diuretic therapy induced more urine volume but was not effective for the symptom. The infiltration shadow in the chest radiogram was getting worse, and hypoxia was not improved by non-invasive persistent pressure ventilation. On the third day of hospitalization, the patient was transported to our hospital for intensive care. At arrival at our hospital, arterial blood gas analysis indicated severe hypoxia and acidosis. Laboratory data are shown in Table 1 and

Figure 2. CRP was very high at admission to our hospital and the pulmonary CT scan showed a diffuse infiltration shadow of both lungs that was not shown in the previous CT (Figure 1 B-2). We could diagnose the patient as the ARDS from these data. Although the mechanical ventilation was started soon after the transportation, the hypoxia was not improved even with a 1.0 fraction of inspiratory oxygen (FiO₂). We decided on the care of VV-ECMO and steroid pulse therapy. The treatment course since hospitalization is shown in Figure 2.

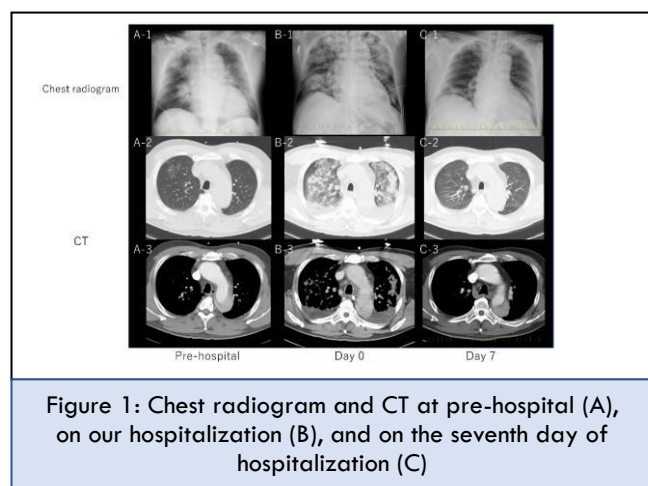


Figure 1: Chest radiogram and CT at pre-hospital (A), on our hospitalization (B), and on the seventh day of hospitalization (C)

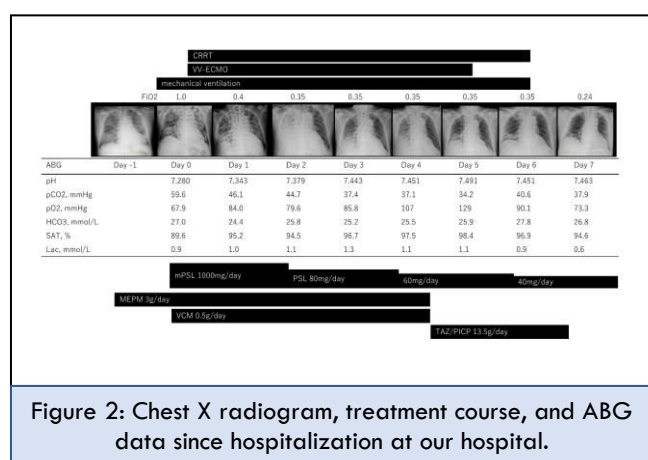


Figure 2: Chest X radiogram, treatment course, and ABG data since hospitalization at our hospital.

CRRT indicates Continuous Renal Replacement Therapy; VV-ECMO: Veno-Venous Extracorporeal Membrane Oxygenation; FiO₂: Fraction of Inspiratory Oxygen; ABG: Arterial Blood Gas; pH: Potential Hydrogen; pCO₂: Partial Pressure of Carbon Dioxide; pO₂: Partial Pressure of Oxygen; HCO₃: Hydrogen Carbonate; SAT: Saturated; Lac: Lactate; mPSL: Methylprednisolone; PSL: Prednisolone; MEPM: Meropenem, VCM: Vancomycin; TAZ/PIPC: Tazobactam/ Piperacillin

Table 1: Laboratory data at pre-hospital admission, day -1, day 0, and day 5 on hospitalization.

	Pre-hospital admission	Day -1	Day 0	Day 5
WBC, /μL	13860	15930	16400	9690
Hb, /μL	13.2	10.3	10.1	10.3
PLT, x10 ⁴ /μL	14.2	16.1	18.7	20.8
PT-INR	0.97		1.20	1.15
APTT (26.9-38.1)	28.7		30.9	39.6
D-dimer, μg/mL	71.2		5.2	4.6
FDP, μg/mL	444.5		9.7	5.2
TP, g/dL	7.7	6.7	7.2	6.1
Alb, g/dL	4.3	3.1	2.9	2.6
AST, U/L	49	17	27	145
ALT, U/L	29	13	17	446
LDH, U/L	533	317	367	513
T-Bil, mg/dL	1.12	1.11	1.9	1.7
CK, U/L	129	171	203	35
Na, mmol/L	139	137	137	144
K, mmol/L	2.6	2.8	3.1	4.2
Cl, mmol/L	98	96	97	108
BUN, mg/dL	32.4	61.2	61	79
CRE, mg/dL	3.12	3.91	3.71	3.01
UA, mg/dL	9.7	8.0	8.5	5.7
CRP, mg/dL	0.84	33.12	39.22	6.98
NT-proBNP, pg/mL	7068		11275	1858

WBC indicates White Blood Cell Count; Hb: Hemoglobin; PLT: Platelet Count; PT-INR: Prothrombin Time; APTT: Activated Partial Thromboplastin Time; FDP: Fibrinogen Degradation Products; TP: Total Protein; Alb: Albumin; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; LDH: Lactate Dehydrogenase; T-Bil: Total Bilirubin; CK: Creatine Kinase; Na: Sodium; K: Potassium; Cl: Chloride; BUN: Blood Urea Nitrogen; CRE: Creatinine; UA: Uric Acid; CRP: C-Reactive Protein; NT-proBNP: N-Terminal Pro-Brain Natriuretic Peptide

Methylprednisolone 1000 mg/day was given for two days, later prednisolone 80 mg/day was given and gradually decreased every two days. Although we were concerned that anti-coagulation for VV-ECMO might affect the state of the dissection, we maintained the activated partial thromboplastin time (APTT) level as usual, twice the normal level. The chest radiogram showed that the lung condition improved day by day

(Figure 1 B-1, C-1). VV-ECMO was discontinued on the fifth day of our hospitalization and mechanical ventilation was successfully weaned on the day after VV-ECMO removal. Pulmonary CT scan indicated that the infiltration shadows in the lung disappeared (Figure 1 C-2). In addition, aortic dissection did not progress more than the original CT images (Figure 1 A-3, B-3, C-3).

DISCUSSIONS

It was reported that the incidence of oxygenation impairment in patients with type-B aortic dissection was 49% [1]. The systemic inflammatory reaction and distension of aortic dissection are associated with oxygenation impairment [2]. There are activated neutrophils in inflammatory lesions. The activated neutrophils induce toxic mediators, so the pulmonary capillary endothelium is destroyed, and vascular permeability increases [3]. Oxygenation impairment in type-B aortic dissection is correlated with younger age and a higher peak CRP level [4]. Likewise, our case was a young type-B aortic dissection patient with a high CRP level.

Mortality for ARDS remains high level. Some studies reported greater than 30% in-hospital mortality and one large trial showed that moderate to severe ARDS had 43% in-hospital mortality at 90 days [5,6]. The therapy for ARDS remains controversial. The use of the high-dose corticosteroid in every ARDS patient is not recommended [7], but we used methylprednisolone 1000 mg/day for two days because we anticipated that the heavy inflammation due to aortic dissection was the cause of ARDS in this case. It is hard to determine whether the high-dose corticosteroid was effective. Regarding ECMO, one study recommends ECMO to significantly improve survival for patients with severe ARDS [8]. On the other hand, there is a study that ECMO does not indicate a significant difference in 60-day mortality for patients with very severe ARDS compared with a strategy of conventional mechanical ventilation that included ECMO as rescue therapy [9]. Moreover, the ECMO increases the frequency of bleeding events leading to transfusion. The decision-making about initiating ECMO is controversial. Especially, if the basic illness had a bleeding risk or the possibility of worsening itself, the decision-making is a very tough question. We should deeply consider the risk and benefits of ECMO in each case.

CONCLUSION

This patient smoothly recovered from ARDS and could remove an ECMO without bleeding events and worsening aortic dissection. ECMO could be considered in patients with potentially reversible respiratory failure even if complicated by aortic dissection.

CONFLICT OF INTEREST

None declared.

ETHICS STATEMENT

Approval of the research protocol: N/A.

Informed Consent: Written informed consent was obtained from the patient for the publication of this case report.

Registry and the Registration No. of the study/Trial: N/A.

Animal Studies: N/A.

REFERENCES

1. Tomita K, Hata N, Kobayashi N, Shinada T, Shirakabe A. (2014). Predicting the occurrence of oxygenation impairment in patients with type-B acute aortic dissection. The International journal of angiology: official publication of the International College of Angiology, Inc. 23:53-60.
2. Hasegawa Y, Ishikawa S, Ohtaki A, Otani Y, Takahashi T, et al. (1999). Impaired lung oxygenation in acute aortic dissection. The Journal of cardiovascular surgery. 40: 191-195.
3. Piantadosi CA, Schwartz DA. (2004). The acute respiratory distress syndrome. Annals of internal medicine. 141: 460-470.
4. Kashiwagi Y, Komukai K, Suzuki K, Oi Y, Tominaga M, et al. (2018). Predictors of oxygenation impairment in medical treatment for type B acute aortic dissection. Heart and vessels. 33: 1463-1470.
5. Bellani G, Laffey JG, Pham T, Fan E, Brochard L, et al. (2016). Epidemiology, Patterns of Care, and Mortality for Patients with Acute Respiratory Distress Syndrome in Intensive Care Units in 50 Countries. Jama. 315: 788-800.
6. Moss M, Huang DT, Brower RG, Ferguson ND, Ginde AA, et al. (2019). Early Neuromuscular Blockade in the Acute Respiratory Distress Syndrome. The New England journal of medicine. 380: 1997-2008.
7. Tasaka S, Ohshimo S, Takeuchi M, Yasuda H, Ichikado K, et al. (2022). ARDS clinical practice guideline 2021. Respiratory investigation. 60: 446-495.

8. Peek GJ, Mugford M, Tiruvoipati R, Wilson A, Allen E, et al. (2009). Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. *Lancet (London, England)*. 374: 1351-1363.
9. Combes A, Hajage D, Capellier G, Demoule A, Lavoué S, et al. (2018). Extracorporeal Membrane Oxygenation for Severe Acute Respiratory Distress Syndrome. *The New England journal of medicine*. 378: 1965-1975.