Korean J Crit Care Med
2013 August 28(3):225-229 / http://dx.doi.org/10.4266/kjccm.2013.28.3.225

Case Report

Extracorporeal Membrane Oxygenation as a Rescue Therapy in a Patient with Non-Iatrogenic Massive Hemoptysis

Jong Hoo Lee, M.D., **Su Wan Kim,** M.D.* and **Yee Hyung Kim**, M.D.†

Departments of Pulmonary and Critical Care Medicine, *Thoracic and Cardiovascular Surgery, Jeju National University Hospital, Jeju National University School of Medicine, Jeju, †Department of Pulmonary and Critical Care Medicine, Kyung Hee University Hospital at Gangdong, Kyung Hee University School of Medicine, Seoul, Korea

Despite the advanced technologies of intensive care, massive hemoptysis can still cause death in a small subset of patients. Extracorporeal membrane oxygenation (ECMO) is expected to provide adequate gas exchange, to reduce ventilator-induced lung injuries, and to eventually improve outcomes in these patients. Also, the instability of vital signs due to hemoptysis makes it impossible to perform immediate interventional procedures such as embolization and resectional surgery. In these cases, ECMO may be instituted as a bridge therapy. Herein, we describe the detailed course of our case, with the hopes of helping physicians to decide when to initiate ECMO in patients with massive hemoptysis.

Key Words: anoxia, extracorporeal membrane oxygenation, hemoptysis.

Massive hemoptysis should always be considered as a life-threatening condition that demands effective assessment and management.[1] Cases of massive hemoptysis that show hemodynamic instability and unstable respiratory changes generally require bronchoscopic treatment, bronchial artery embolization or surgical resection. However, unstable hemodynamics and severe hypoxemia often make it difficult or impossible to perform these emergent procedures.

Extracorporeal membrane oxygenation (ECMO) may be considered for use in patients with acute respiratory failure if one or more of the following are present: severe hypoxemia, uncompensated hypercapnia, or excessively high end-inspiratory plateau pressures despite adequate management with a mechanical ventilator.[2] However, there are a few cases describing ECMO in patients with massive hemoptysis.[3-5] Therefore, it is not clear whether it is beneficial to apply ECMO as a rescue therapy to unstable patients with massive hemoptysis prior to definite therapeutic procedures. We report a successful application of ECMO as a rescue therapy prior to ultimate intervention for severe non-iatrogenic hemoptysis.

Received on May 27, 2013, Revised on July 2, 2013, Accepted on July 18, 2013

Correspondence to : Yee Hyung Kim, Department of Pulmonary and Critical Care Medicine, Kyung Hee University Hospital at Gangdong, 149, Gangdong-gu, Sangil-dong, Seoul 134-727, Korea
Tel: 02-440-6281, Fax: 02-440-8150
E-mail: lovlet@paran.com

This research was supported by the 2013 scientific promotion program funded by Jeju National University.

CASE REPORT

A 68-year-old man visited emergency department after experiencing aggravated dyspnea for 10 days. He had a past medical history of pulmonary tuberculosis. His vital signs were as follows: blood pressure (BP) 103/56 mmHg, pulse rate (PR) 112 beats/min, respiratory rate (RR) 36/min and body temperature (BT) 38.1°C. Auscultation revealed crackles in both lower lungs. A complete blood count showed WBC count of 7,500/㎕, hemoglobin of 11.7 g/dl and platelet count of 245,000/㎕. His activated partial thromboplastin time (aPTT) and prothrombin time (PT) were within normal limits. Hepatic and renal function test were also normal. Arterial blood gas analysis (ABGA) at room air showed severe hypoxia: oxygen partial pressure (PaO_2) of 42.3 mmHg, carbon dioxide partial pressure ($PaCO_2$) of 46.9 mmHg, pH of 7.45, and oxygen saturation (SaO_2) of 76.5%. C-reactive protein was 20.51 mg/dl. The cardiac specific enzymes including troponin-T were within normal limits.

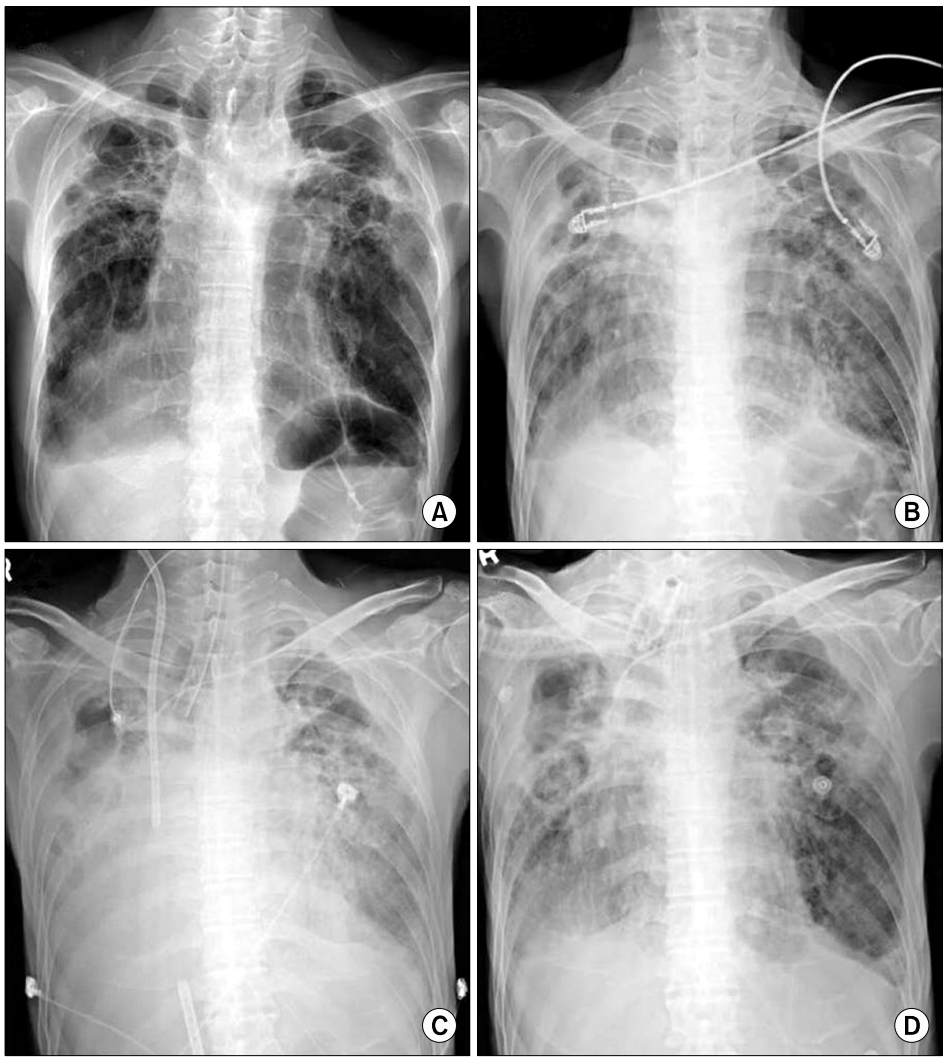

Fig. 1. (A) Chest radiography obtained two months prior to admission showed pulmonary lesions that were destroyed by pulmonary tuberculosis. (B) On admission, the patient's chest radiography revealed increased bilateral pulmonary infiltrates. (C) Along with collapse of right middle and lower lung due to massive hemoptysis, a right femoral drainage cannula and a right carotid return cannula were inserted for ECMO. (D) At ECMO removal, chest radiography showed loss of atelectasis on the right lower lung field with decreased pneumonic infiltration.

Chest radiography revealed increased bilateral pulmonary infiltration with underlying severe post-infectious sequelae, compared with previous images (Fig. 1A, 1B). The patient was diagnosed with community acquired pneumonia and started on a course of piperacillin/tazobactam and levofloxacin. ABGA while breathing at oxygen content of 40% via venturi mask showed severe respiratory acidosis and hypoxemia: pH of 7.11, $PaCO_2$ of 93.3 mmHg, PaO_2 of 60.7 mmHg and SaO_2 of 81.7%. We started the patient on mechanical ventilation. After endotracheal intubation and bronchial toileting, his ABGA showed improvements of gas exchange and hypoxemia. The mechanical ventilator was set as follows in pressure control mode: rate of 10 breaths/min, PEEP of 8 cmH_2O, pressure support of 20 mmHg and FiO_2 of 0.35.

On the second day, the patient's condition changed abruptly. He developed a large amount of bleeding through the endotracheal tube. Vital signs were as follows: BP of 54/27 mmHg, PR of 130 beats/min, RR of 32/min and BT of 36.8°C. Despite maximal ventilatory support (pressure control mode, pressure support of 25 mmHg, rate of 28 breath/min, PEEP of 12 cmH_2O and FiO_2 of 1.0), the patient remained severely hypoxemic and had respiratory acidosis (pH of 7.25, $PaCO_2$ of 67.9 mmHg, PaO_2 of 51.4 mmHg and SaO_2 of 82.4%). Chest radiography and CT showed newly developed atelectasis on the right lower lobe and ground glass opacities in left lower lobe (Fig. 1C, Fig. 2). Flexible bronchoscopy revealed a hematoma on the right main bronchus and fresh blood on whole bronchi. We cleared the blood clots and secretions out from the airways to prevent asphyxia and to preserve ventilation. We administered topical epinephrine and cold saline lavage. In addition, unilateral intubation through the left main bronchus was performed to isolate the non-affected lung.

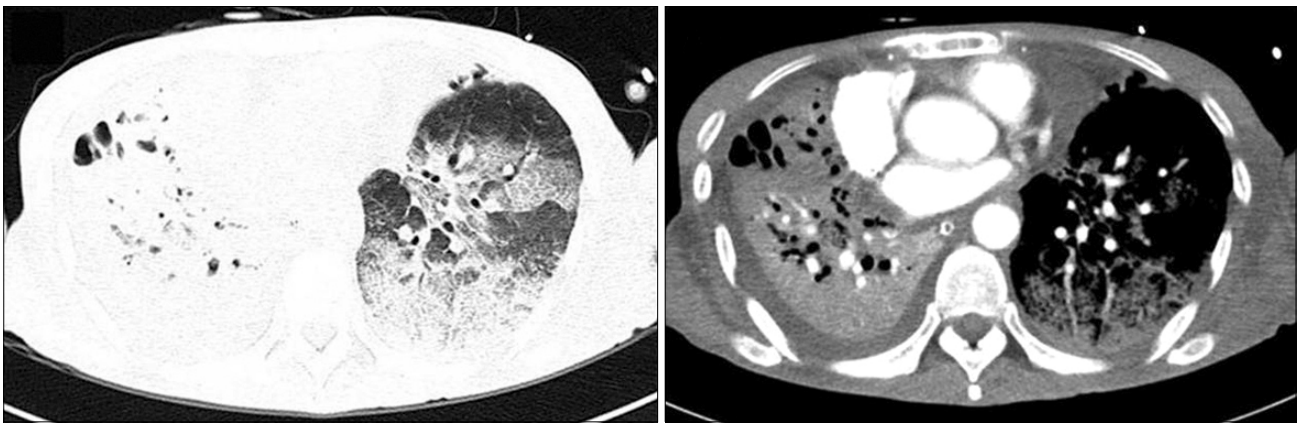

Fig. 2. A chest CT scan obtained on second hospital day showed extensive consolidation and ground glass opacities on left lower lobes and atelectasis on right lower lobe.

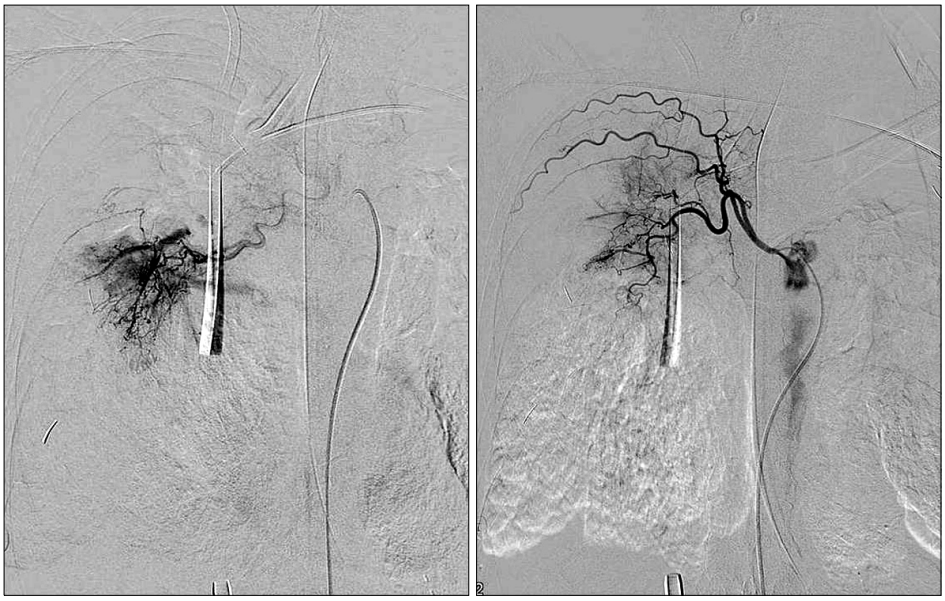

Fig. 3. Bronchial arteriography showed hypertrophic right bronchial arteries and chronic inflammatory hyperemic lesions with bronchial artery-pulmonary artery shunts, suggesting the cause of hemoptysis.

Unfortunately, these procedures did not improve the patient's oxygenation. We were not able to immediately perform bronchial artery embolization or surgical resection because of the patient's unstable vital signs and severe hypoxemia. Prior to performing definite interventional procedures, we decided to use ECMO to temporarily maintain arterial oxygenation. A venovenous ECMO system (EBS, Capiox® Emergency Bypass System, Terumo Inc., Tokyo, Japan) was set up the third day of hospitalization, with a 21-Fr drainage cannula (DLP®, Medtronic Inc., MN) from the right femoral vein and a 17-Fr return cannula into the right internal jugular vein. Heparin was not used due to hemoptysis. The ECMO settings were as follows: blood flow of 4.0 L/min, revolutions per minute of 2,050 times, and FiO_2 of 1.0. The mechanical ventilator care was minimally maintained (pressure control mode, pressure support of 10 mmHg, rate of 10 breath/min, PEEP of 5 cmH_2O, and FiO_2 of 0.3). Within 1 hour after the application of ECMO, the patient's ABGA showed improved oxygenation and gas exchange (pH 7.479, $PaCO_2$ of 38.3 mmHg, PaO_2 of 64.5 mmHg and SaO_2 of 97.0%). At the same time, his BP increased to 105/70 mmHg.

As the patient began to show stable vital signs, we initiated bronchial artery embolization. On the bronchial arteriography, hypertrophic right bronchial arteries and chronic inflammatory hyperemic lesions were noted in the right lung with bronchial artery-pulmonary artery shunts, suggesting the cause of hemoptysis (Fig. 3). After contour embolization through super-selection of these arteries, no active bleeding was observed.

In order to wean the patient from ECMO without any anticoagulant, only gas flows were gradually decreased and the blood flow was maintained unchanged. During ECMO, an activated clotting time (ACT) and aPTT of the patient were maintained within normal limits. Fortunately, there were no complications associated with the ECMO application. The ECMO was withdrawn on day 8, when the ventilator mode was pressure support. The total duration of the ECMO application was 70 hours. Chest radiography showed loss of atelectasis on the right lower lung field with decreased pneumonic infiltration (Fig. 1D). The patient was weaned off the mechanical ventilator on day 10 and discharged from ICU on day 20.

DISCUSSION

Although massive hemoptysis is a potentially fatal condition, there is no clear consensus on its precise definition. A more relevant definition relies on the main clinical consequences of hemoptysis such as airway obstruction and hemodynamic instability. Other definitions of hemoptysis have included the absence or presence of factors such as transfusion, hospitalization, intubation, aspiration, airway obstruction, hypoxemia (PaO_2 < 60 mmHg) or death.[1] In our case, we observed airway obstruction and hypoxemia leading to respiratory failure that required transfusion and volume resuscitation. These findings indicate that our patient had massive hemoptysis complicated with pneumonia and structural lung disease.

The initial approach for management of massive hemoptysis includes protection of the airways and volume resuscitation.[6] If hemodynamic or respiratory status is compromised, urgent rigid bronchoscopy should be attempted by a skilled physician, as it is the most efficient means of clearing the airways of blood clots and secretions. However, a trained bronchoscopist who can perform rigid bronchoscopy is not always readily available in the event of massive hemoptysis. In this case, the patient should be intubated with a large-caliber endotracheal tube, and flexible bronchoscopy should be performed immediately, mainly to remove blood clots and secretions. In addition, once the airways are cleared, unilateral intubation can be performed to protect the non-bleeding lung from aspiration and to allow effective ventilation while awaiting definitive treatment strategies. In this situation, urgent interventional procedures such as bronchial artery embolization and surgical resection should be considered. However, it is sometimes impossible to perform these interventions immediately because of hemodynamic instability and severe hypoxemia. If an urgent interventional procedure cannot be performed, ECMO might be an effective rescue therapy to maintain arterial oxygenation, even in the case of massive hemoptysis. In our case, even though we performed bronchoscopic clearing and a unilateral intubation, the patient's oxygenation was not improved because of his massive hemoptysis, pneumonia, and decreased lung function due to post-infectious sequelae. Hypotension also developed. Abrupt desaturation and arterial hypotension made it impossible to perform an embolization or resectional surgery, even though these procedures can save lives. We finally decided to start ECMO as a rescue therapy to maintain oxygenation.

ECMO is an extracorporeal circuit that directly oxygenates and removes carbon dioxide from the blood. Though previous clinical trials did not show improved survival in patients with ARDS,[7,8] a recent CESAR study using modern ECMO technology reported favorable outcomes with ECMO.[9] Now, ECMO is considered as a rescue therapy when positive- pressure ventilation cannot maintain adequate oxygenation or carbon dioxide removal. As results of clinical studies and experiences accumulate, the range of ECMO applications can be extended.

The use of ECMO for treatment of massive hemoptysis has been described in only a few cases.[3-5] Most previous reports were about patients with iatrogenic hemoptysis that developed as complications during cardiopulmonary bypass.[5] Otherwise, cases stabilized by ECMO in non-iatrogenic massive hemoptysis are very scarce.[3] In the case of Korea, Yoo et al. reported on a case of successful application with using both veno-venous and veno-arterial ECMO for a patient with pulmonary tuberculosis who suffered from repeated life threatening hemoptysis.[4]

During ECMO, there is continuous contact between the circulating blood and the foreign surface of the circuit. As the hemostatic balance is shifted to hypercoagulability with patients, systemic anticoagulation with unfractionated heparin is required during ECMO to avoid thrombus formation in the circuit.[2] But, because systemic anticoagulation could cause life-threatening bleeding, we did not use any anticoagulants for our patient with massive hemoptysis. Lappa et al. reported on a case weaning veno-venous ECMO without anticoagulation due to severe bleeding from thoracic and mediastinal drainages.[10] To avoid thrombotic events, a high blood flow was maintained and gas flows were slowly reduced. We used the same strategy and our patient underwent a successful weaning from veno-venous ECMO without thrombotic complications and the change of membrane, fortunately. But, the standard strategy

to wean the patient from ECMO without anticoagulation is necessary further studies. In addition, this strategy is not recommended for veno-arterial ECMO requiring blood flow reduction to assess heart recovery.[10]

In our case, ECMO was used as a rescue therapy in a patient with hypoxemia and hypotension due to non-iatrogenic massive hemoptysis. These findings indicate that ECMO can be used as a rescue therapy in patients with massive hemoptysis until therapeutic procedures such as urgent angiographic embolization and/or surgical resection can be safely performed.

In summary, we present a case of successful ECMO application without anticoagulation as a rescue therapy for life-threatening, non-iatrogenic, massive hemoptysis. Therefore, ECMO seems to allow patients with desaturation due to massive hemoptysis to be stabilized and to more safely undergo subsequent therapies.

REFERENCES

1) Sakr L, Dutau H: Massive hemoptysis: an update on the role of bronchoscopy in diagnosis and management. Respiration 2010; 80: 38-58.
2) Brodie D, Bacchetta M: Extracorporeal membrane oxygenation for ARDS in adults. N Engl J Med 2011; 365: 1905-14.
3) Hsu SJ, Luo YH, Lee YC, Yang KY: Life-threatening hemoptysis due to left inferior phrenic artery to pulmonary artery fistula rescued by extracorporeal membrane oxygenation therapy. Interact Cardiovasc Thorac Surg 2011; 12: 337-8.
4) Yoo JS, Chung ES, Choi JH, Lim C, Jheon S, Park KH: Venovenous and venoarterial extracorporeal membrane oxygenation for repeated life threatening hemoptysis: a case report. Korean J Thorac Cardiovasc Surg 2008; 41: 764-7.
5) Bianchini R, Melina G, Benedetto U, Rossi M, Fiorani B, Iasenzaniro M, et al: Extracorporeal membrane oxygenation for Swan-Ganz induced intraoperative hemorrhage. Ann Thorac Surg 2007; 83: 2213-4.
6) Dweik RA, Stoller JK: Role of bronchoscopy in massive hemoptysis. Clin Chest Med 1999; 20: 89-105.
7) Zapol WM, Snider MT, Hill JD, Fallat RJ, Bartlett RH, Edmunds LH, et al: Extracorporeal membrane oxygenation in severe acute respiratory failure. A randomized prospective study. JAMA 1979; 242: 2193-6.
8) Morris AH, Wallace CJ, Menlove RL, Clemmer TP, Orme JF Jr, Weaver LK, et al: Randomized clinical trial of pressure-controlled inverse ratio ventilation and extracorporeal CO2 removal for adult respiratory distress syndrome. Am J Respir Crit Care Med 1994; 149: 295-305.
9) Peek GJ, Mugford M, Tiruvoipati R, Wilson A, Allen E, Thalanany MM, et al; CESAR trial collaboration: Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. Lancet 2009; 374: 1351-63.
10) Lappa A, Donfrancesco S, Contento C, Vitalini E, Pisani P, Menichetti A, et al: Weaning from venovenous extracorporeal membrane oxygenation without anticoagulation: is it possible? Ann Thorac Surg 2012; 94: e1-3.