Official Case Reports Journal of the Asian Pacific Society of Respirology
Respirology Case Reports
OPEN ACCESS

Endotracheal tube clamping and extracorporeal membrane oxygenation to resuscitate massive pulmonary haemorrhage

Chien-Feng Lee, Chun-Ta Huang & Sheng-Yuan Ruan

Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan.

Keywords
Antineutrophil cytoplasmic antibody, extracorporeal membrane oxygenation, Haemoptysis, microscopic polyangiitis, pulmonary haemorrhage.

Correspondence
Sheng-Yuan Ruan, Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, National Taiwan University Hospital, No.7 Chung-Shan South Road, Taipei 10002, Taiwan.
E-mail: syruan@ntu.edu.tw

Received: 5 March 2018; Revised: 20 March 2018; Accepted: 22 March 2018.

***Respirology Case Reports*, 6 (5), 2018, e00321**

doi: 10.1002/rcr2.321

Abstract

Massive pulmonary haemorrhage is a life-threatening and difficult-to-manage condition. In certain circumstances, traditional approaches for haemoptysis may not be effective. Here, we report a 64-year-old man presenting with dyspnoea and leg oedema. He was diagnosed with microscopic polyangiitis due to positive perinuclear anti-neutrophil cytoplasmic antibody and other supportive evidence. His hospital course was complicated with massive pulmonary haemorrhage, which led to hypoxic respiratory failure, shock, and pulseless electrical activity. Extracorporeal membrane oxygenation (ECMO) was employed during cardiopulmonary resuscitation. To control blood loss from his lungs, we clamped the endotracheal tube for tamponade therapy. The tube was clamped for 15 h till the haemorrhage subsided. ECMO and ventilator support were successfully weaned off after 5 and 10 days, respectively. Our favourable experience suggests that endotracheal tube clamping with ECMO support is a viable management option for life-threatening pulmonary haemorrhage.

Introduction

Massive pulmonary haemorrhage is a life-threatening and difficult-to-manage condition. The recommended approaches are to secure the airway, correct the bleeding tendency, and stabilize haemodynamics, followed by localizing the bleeding site through clinical history, imaging studies, and bronchoscopy [1,2]. Bronchoscopic intervention and bronchial arterial embolization can stop bleeding in most patients. However, in some cases, interventional angiography and bronchoscopy might not be safely and effectively performed.

Here, we report a case of microscopic polyangiitis (MPA) with massive pulmonary haemorrhage, which led to hypoxaemia, shock, and pulseless electrical activity (PEA). Life-threatening pulmonary haemorrhage was successfully controlled by airway tamponade therapy using endotracheal tube clamping with extracorporeal membrane oxygenation (ECMO) support.

Case Report

A 64-year-old man without a past medical history presented to the hospital because of malaise, poor appetite, shortness of breath, and bilateral leg oedema for several weeks. His shortness of breath was progressively worsening, and he had orthopnoea for a few days before admission. He also had dry cough and decreased urine output but no fever, chills, abdominal pain, skin rashes, or joint pain.

On physical examination, he was alert and acutely ill-looking. His blood pressure was 184/112 mmHg, pulse rate 100 bpm, temperature 36.2 °C, and pulse oxygen saturation 98% while breathing ambient air. His conjunctiva was pale. Crackles in both lower lung fields and bilateral legs pitting oedema were noted.

His white blood cell count was 6630 cells/μL with 82.4% of neutrophils and 0.9% of eosinophils, haemoglobin level was 7.1 g/dL, and platelet count was 305,000 cells/μL.

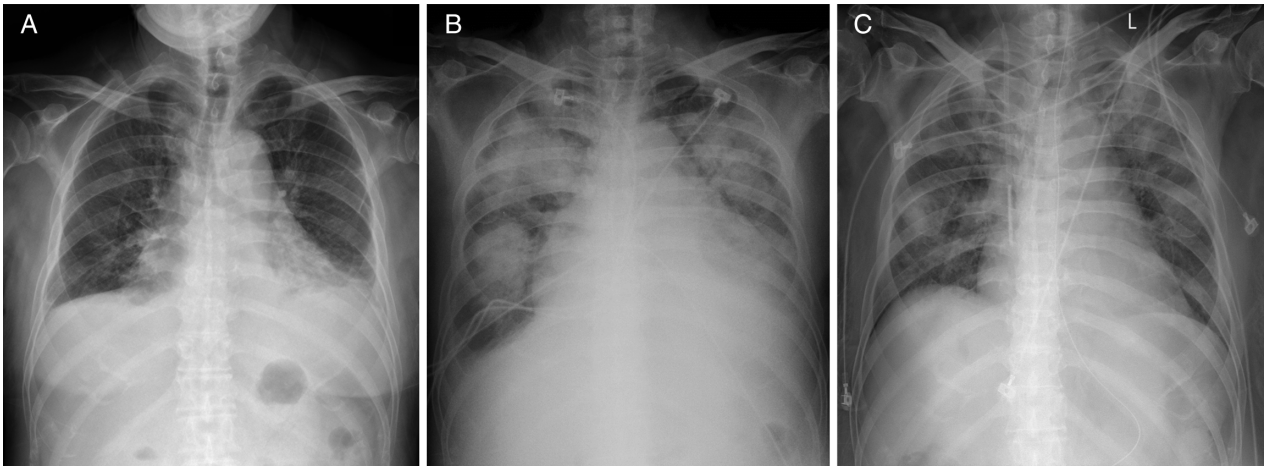

Figure 1. (A) The chest X-ray on admission showed cardiomegaly and increased bilateral lower infiltration. (B) The chest X-ray before intubation revealed increased heart size and new bilateral patchy consolidation. (C) The chest X-ray on the day of general ward transferal showed resolution of bilateral patches and a right-sided double lumen catheter.

Blood biochemistry testing revealed blood urea nitrogen of 189.9 mg/dL, creatinine of 17.3 mg/dL, total bilirubin of 0.21 mg/dL, and albumin of 2.8 g/dL. A venous blood gas showed pH: 7.226, pCO_2: 26.8 mmHg, pO_2: 32.6 mmHg, and HCO_3: 11.2 mmol/L. A chest X-ray showed cardiomegaly with increased bilateral lower infiltrates (Fig. 1A). Renal sonography showed bilateral parenchymal disease with normal-size organs (right kidney: 10 cm; left kidney: 9.7 cm).

Intermittent haemodialysis was initiated for uremic symptoms and fluid overload. Unfractionated heparin was used only for priming the circuits (1200 IU) before each haemodialysis session. Serial examinations, including autoimmune profile, urinalysis, and renal biopsy, were performed to assess the aetiology of renal failure. He was diagnosed with MPA due to a strongly positive perinuclear anti-neutrophil cytoplasmic antibody (P-ANCA) (>134 IU/mL; reference, < 5 IU/mL) and a pathology finding of pauci-immune-type crescentic glomerulonephritis on renal biopsy. However, the patient developed haemoptysis before starting MPA treatment on the 10th hospital day. A chest X-ray showed a further increase in cardiac silhouette and new bilateral patchy consolidation (Fig. 1B). His haemoptysis rapidly worsened, and he was intubated on the 11th hospital day because of hypoxic respiratory failure and shock. After intubation, he was transferred to the ICU immediately. Unfortunately, PEA developed 2 h after the intubation due to persistent hypoxaemia and haemorrhagic shock. Cardiopulmonary resuscitation was started for PEA. During the resuscitation, more fresh blood continuously flooded from his endotracheal tube and caused airway obstruction (Fig. 2A and Video S1, Supporting information). Due to inability to maintain adequate ventilation and sustained PEA, veno-arterial (V-A) ECMO was used, followed by an add-on veno-venous (V-V) ECMO. To control blood loss from the airway, we clamped his endotracheal tube and suspended mechanical ventilation (Fig. 2B). Anticoagulants were not administered during ECMO due to active bleeding. The levels of activated clotting time (ACT) during ECMO are shown in Figure 2C. Transfusion requirement dramatically reduced after clamping the endotracheal tube. Meanwhile, cardiac sonography showed a moderate amount of pericardial effusion, and pericardiocentesis drained 500 mL of bloody fluids.

His pulmonary haemorrhage was stabilized after endotracheal tube clamping; transfusion; immunosuppressant therapy with cyclophosphamide, rituximab, and methylprednisolone; and plasmapheresis. The endotracheal tube was clamped for 15 h and then reconnected to the ventilator after removing endotracheal blood using suction. Bronchoscopy revealed blood clots coating on airways but no bleeders or ulcers. The ECMO support was terminated in 5 days. The patient was successfully extubated on the 10th ICU day and was then transferred to the general ward without significant neurological deficits. The chest X-ray on the day of general ward transferal showed that bilateral patchy infiltrates were significantly resolved (Fig. 1C).

Discussion

Pulmonary haemorrhage due to ANCA-associated vasculitis is a critical condition associated with high mortality [3]. The recommended management, including bronchoscopy, double-lumen endotracheal tube placement, and bronchial arterial embolization, might not be promptly and

effectively performed due to unstable vital signs and diffuse bleeding in both lungs. In this condition, ECMO can be used to provide cardiopulmonary support to enable definitive therapies for vasculitis to take effect. To prevent further haemorrhage, anticoagulant therapy could be withheld during ECMO by introducing biocompatible circuits [4]. Under ECMO, the endotracheal tube was clamping for a tamponade effect to decrease blood loss from the airway and reduce the need for transfusion in our case. This successful experience suggests that endotracheal tube clamping with ECMO support is a feasible approach to control life-threatening pulmonary haemorrhage.

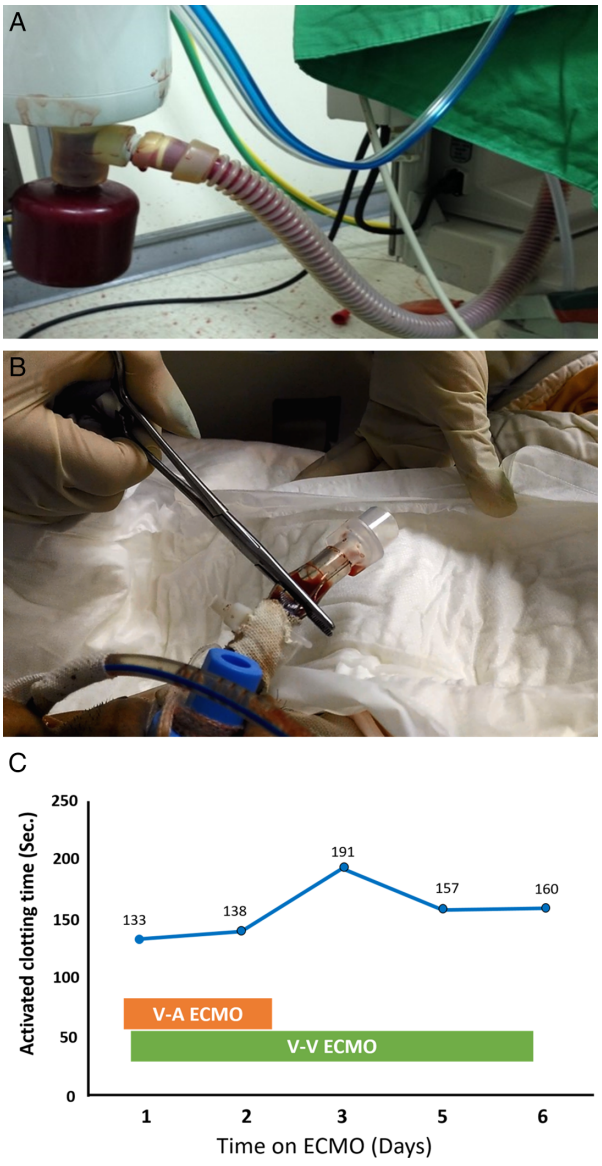

Figure 2. (A) Ventilator tubing was fully filled with the blood flooding from the endotracheal tube. (B) The endotracheal tube was clamped for airway tamponade therapy. (C) The levels of activated clotting time (ACT) and during extracorporeal membrane oxygenation (ECMO). V-A: Veno-arterial, V-V: Veno-venous.

The literature regarding the combination of endotracheal tube clamping and ECMO for the management of severe airway bleeding is limited. Only a few cases describing endotracheal tube clamping as airway tamponade therapy under ECMO support for massive bleeding in the lower airways were reported [5]. This is the first case of vasculitis-related massive pulmonary haemorrhage treated with endotracheal tube clamping and anticoagulant-free ECMO support. The requirement for transfusion dramatically reduced after clamping the endotracheal tube. The risk for transfusion-related lung injury may consequently be reduced. The airway tamponade therapy via endotracheal tube clamping did not introduce significant respiratory complications or infections in this case. Ventilator support was successfully discontinued in 10 days. Significant adverse effects related to endotracheal tube clamping were not observed.

Proper anticoagulation is usually required to prevent circuit thrombosis during ECMO support. However, heparin was not infused in this case due to severe pulmonary haemorrhage. A high ECMO blood flow rate was maintained, and the inflow and outflow pressures were closely monitored. During the 5 days of ECMO support, we did not encounter any circuit or pump failure due to clot formation. Due to the advances in ECMO equipment, successful experiences with heparin-free ECMO support have been increasingly reported [6,7]. Miniaturized ECMO systems have reduced the blood contact surface and heparin coating, and increased biocompatible circuits also minimize the activation of blood coagulation cascades.

In cases with pulmonary vasculitis, the anticoagulation strategy during haemodialysis is challenging. Regional citrate anticoagulation (RCA) may be a good alternative treatment for these patients because they are highly at risk of bleeding due to vasculitis. RCA restricts the anticoagulatory effect to the extracorporeal circuits and is associated with lower risk of bleeding compared to heparin [8]. However, it has not yet been widely available in clinical practice due to higher costs and more complicated management protocols. In our case, the patient's pulmonary haemorrhage was considered to be associated with the disease activity of MPA rather than haemodialysis. In addition, he did not undergo haemodialysis on the index day of haemoptysis.

ECMO can oxygenate red blood cells and eliminate carbon dioxide. In this case, ECMO was used to allow time for recovery from severe pulmonary haemorrhage. During the ECMO support, the protective ventilation strategy is important to prevent ventilator-induced lung injury [9]. Ventilation settings during ECMO support were a pressure

control mode with an inspiratory pressure of 14 cm H_2O and a PEEP of 10 cm H_2O. The tidal volume under the settings was 150–200 mL, which is equal to 2–3 mL per predicted body weight (kg). Respiratory compliance rapidly improved after the pulmonary haemorrhage subsided. ECMO was terminated in 5 days, and the ventilator was discontinued in 10 days.

Disclosure statement

Appropriate written informed consent was obtained for publication of this case report and accompanying images and video.

References

1. Radchenko C, Alraiyes AH, and Shojaee S. 2017. A systematic approach to the management of massive hemoptysis. J. Thorac. Dis. 9:S1069–S1086.
2. Yendamuri S. 2015. Massive airway hemorrhage. Thorac. Surg. Clin. 25:255–260.
3. Lauque D, Cadranel J, Lazor R, et al. 2000. Microscopic polyangiitis with alveolar hemorrhage. A study of 29 cases and review of the literature. Groupe d'Etudes et de Recherche sur les maladies "Orphelines" Pulmonaires (GERM"O"P). Medicine 79:222–233.
4. Combes A, Bacchetta M, Brodie D, et al. 2012. Extracorporeal membrane oxygenation for respiratory failure in adults. Curr. Opin. Crit. Care 18:99–104.
5. Vobruba V, Grus T, Mlejnsky F, et al. 2018. Management of severe pulmonary hemorrhage in a neonate on veno-arterial ECMO by the temporary clamping of the endotracheal tube—a case report. Perfusion 33:77–80.
6. Zonies D, and Merkel M. 2016. Advanced extracorporeal therapy in trauma. Curr. Opin. Crit. Care 22:578–583.
7. Arlt M, Philipp A, Voelkel S, et al. 2010. Extracorporeal membrane oxygenation in severe trauma patients with bleeding shock. Resuscitation 81:804–809.
8. Zhang Z, and Hongying N. 2012. Efficacy and safety of regional citrate anticoagulation in critically ill patients undergoing continuous renal replacement therapy. Intensive Care Med. 38:20–28.
9. Zhang Z, Gu WJ, Chen K, et al. 2017. Mechanical ventilation during extracorporeal membrane oxygenation in patients with acute severe respiratory failure. Can. Respir. J. 2017:1783857.

Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site: http://onlinelibrary.wiley.com/doi//suppinfo.

Video S1. During the resuscitation, more fresh blood continuously flooded from the patient's endotracheal tube and caused airway obstruction.