Bedside placement of an endobronchial valve to aid invasive ventilation and weaning from extracorporeal membrane oxygenation

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Clinical record
An 18-year-old male university student was referred to our centre for consideration of extracorporeal membrane oxygenation (ECMO) for severe, acute, hypoxic respiratory failure. He was previously fit and well, took no regular medications, and had no known allergies. He presented to his local hospital with a 3-week history of cough, dyspnoea and pleuritic chest pain which had failed to improve despite a course of amoxycillin from his general practitioner. He was febrile, had leukophila (leukocyte count, 23.05 \times 10^9/L [reference range, 4–11 \times 10^9/L]), and his chest radiograph showed right lower lobe consolidation and right-sided pleural effusion. He was diagnosed with pneumonia, and started on intravenous (IV) antibiotics (ceftriaxone 2 g once daily, clarithromycin 500 mg twice daily, and flucloxacillin 1 g four times daily).

His clinical condition subsequently deteriorated and he required intubation and ventilation within 12 hours of admission to hospital. He required significant vasoactive support, which was delivered via a right subclavian central venous catheter. He was acidotic, anuric, and despite an \textit{FiO2} of 1.0, his peripheral arterial haemoglobin oxygen saturation remained at only 84%. He was referred to our centre for ECMO.

Our retrieval team placed the patient on venovenous ECMO (VV-ECMO) before transfer back to our centre. A 29 Fr multilumen venous drainage cannula (Maquet) was placed in the left femoral vein, and a 21 Fr venous return cannula (Maquet) was placed in the right internal jugular vein, both without complication, and flows of about 8 L/min were achieved using a centrifugal pump (BPX-80 BIO Pump Plus [Medtronic]). With this intervention, the patient’s systemic haemoglobin oxygen saturation improved to about 90%.

On arrival at our centre, the patient was febrile (temperature, 38.7°C), tachycardic (heart rate, 128 beats/min) and hypotensive (blood pressure, 83/35 mmHg) and he was being supported with noradrenaline (0.37 \mu g/kg/min), arginine vasopressin (2 U/hr) and IV hydrocortisone (100 mg three times daily). With this intervention, the patient’s systemic haemoglobin oxygen saturation improved to about 90%.

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We performed transoesophageal echocardiography, which showed a structurally normal heart with vigorous biventricular function and a small, non-significant, pericardial effusion. We saw no intracardiac shunts. A chest radiograph showed complete, four-quadrant, consolidation of the patient’s lung fields. Extensive microbiological investigations were negative for a causative organism. Repeat chest radiographs showed continuing dense four-quadrant consolidation, and his inflammatory markers failed to settle.

On Day 8 of the patient’s stay, his condition deteriorated significantly, with a lactataemia developing over 24 hours, despite continued CVVH, and peaking at 17.1 mmol/L (normal range, 0.5–2.2 mmol/L). His transaminase levels became elevated and his leukocytosis became more profound. Antibiotic therapy was changed to meropenem and vancomycin. Computed tomography (CT) of his head, chest, abdomen and pelvis was performed and bilateral loculated pleural collections were seen. Bilateral intercostal drains (ICDs) were placed.
placed, using the Seldinger technique, and drained about 2500 mL of frank pus. This pus subsequently grew *Fusobacterium necrophorum*. Subsequently, a right BPF was diagnosed with persistent, vigorous bubbling into the chest drain and a large right pneumothorax (Figure 1).

A second CT scan was performed 2 days later and showed a large residual right pneumothorax with a lung adhesion to the lateral pleura. A second ICD was placed anteriorly (in the second intercostal space at the mid-clavicular line) on the right side in an attempt to drain the superior pleural air. Vigorous bubbling via the two right-sided drains continued, despite the patient being on only 10 cmH₂O of positive end expiratory pressure on the ventilator, ie, “rest settings”. The right lung failed to expand and a repeat CT scan showed a large BPF (Figure 2). Therefore on Day 10, a flexible bronchoscopy was performed and copious, purulent, malodorous secretions were found throughout the tracheobronchial tree. We localised the airleak to the anterior basal segment of the right lower lobe (RB8), and inflated an endobronchial balloon catheter (BS-2C, Olympus). This occluded all the right lower lobe except for the superior lower lobe segment and resulted in complete cessation of the airleak (Figure 3). The balloon was left inflated in situ overnight pending sourcing and placement.
of an endobronchial valve the next day. On Day 11, a 7 mm endobronchial valve (IBV-V7, Olympus) was placed, using a catheter and loader (IBV-C20, Olympus) after sizing of the bronchus with a valve-sizing kit (IBV-SK, Olympus). After deployment of this valve, bubbling from the right chest drains improved markedly and the right lung mostly re-expanded (Figure 4). A repeat flexible bronchoscopy on Day 12 revealed that the valve had moved slightly, so it was repositioned to again cover all three subsegments of the RB8 lobe.

This patient’s stay in the intensive care unit was further complicated on Day 17 by a pericardial effusion, seen on a CT scan of the chest, which appeared on transoesophageal echocardiogram to be causing tamponade. Pericardiocentesis was unsuccessful, so a thoracotomy was performed and a pericardial window created.

The chest drains were noted to bubble again, and a repeat flexible bronchoscopy on Day 25 showed that the valve had become damaged and displaced, so it was removed. On Day 26, after testing each RB8 lobe subsegment with a balloon and undertaking repeat sizing, another 7 mm endobronchial valve was inserted into the most lateral of the three subsegments of RB8. This again led to a significant reduction in the airleak. Balloon occlusion of the right upper and middle lobe bronchi did not alter the residual leak, so it was assumed that there may be some collateral ventilation between the RB8 subsegments or other segments of the right lower lobe. Over time, the residual leak settled and the chest drains were removed without incident.

Thereafter, the patient made a gradual recovery. VV-ECMO support was withdrawn on Day 23. A percutaneous tracheostomy was performed on Day 25 and the patient was slowly weaned from respiratory support. He was discharged from the ICU on Day 55. The tracheostomy tube was removed on Day 57, and on Day 62 he was discharged from our hospital and transferred to his local tertiary hospital for ongoing rehabilitation. A chest radiograph before discharge showed a pneumatocele laterally in the right hemithorax and improving left lower lobe atelectasis. This small pneumatocele was still present on a CT scan 4 months later, so the valve was left in situ (Figure 5). Since then, the patient has made a good recovery and returned full-time to university. At follow-up, the patient had an FEV1 of 3.83 L (73% of predicted) and an FVC of 4.06 L (64% of predicted). His DLCO was 9.2 mmol/kPa.min (68% of predicted), and his DLCO/VA was 97% of predicted. A formal cardiopulmonary exercise test performed 6 months after his initial presentation showed a VO2 max of 40.7 mL/Kg/min (60% of predicted).

Discussion

The management of BPF is complex and largely guided by expert opinion, in the absence of randomised controlled trials or specific management guidelines. Management should be tailored to each individual situation.1 Initial management is aimed at minimising the transpulmonary pressure gradient by minimising the mean airway pressure and, provided the lung remains inflated, minimising the suction on pleural drains if present. Bronchoscopy has a diagnostic and a therapeutic role. Multiple compounds have been used to attempt to seal BPFs bronchoscopically,1 and vascular coils, with or without the use of other sealants, have also been used, as have specially designed spigots.2 More recently, one-way endobronchial valves, originally designed for the treatment of emphysema, have been used.3-8 Surgical treatment of BPF is successful in most cases in which it is attempted, but is not always possible because of a patient’s comorbidities and physical condition (such as in this case).

This case shows that it is feasible to place an endobronchial valve via flexible bronchoscopy to treat a BPF at the bedside in a patient on VV-ECMO who was unfit for surgical treatment. Other groups have described the use of an endobronchial valve to treat a BPF2-8 but this is only the second time this has been performed in a patient on ECMO.7 Our patient differed from the single previous report in that the procedure was performed using a flexible bronchoscope, the valve was placed more distally in the tracheobronchial tree, and a test occlusion was performed with an endobronchial balloon for some hours in order to select the site to implant the endobronchial valve. An anaerobic infection in the patient’s lung parenchyma made it impossible to define, with high-resolution CT, which bronchial subsegment was responsible for feeding the BPF.

Figure 5. Computed tomography scan of the chest, transverse section

A residual pneumatocele in the right chest (arrow) and resolution of the lung consolidation can be seen.
Our patient was not fit enough for the BPF to be treated surgically and we were concerned that any such attempted treatment would result in the loss of too much lung tissue and/or exsanguination. We believe that, in addition to the use of ECMO, this procedure was life-saving as it allowed his right lung to re-expand and ultimately facilitated his weaning from VV-ECMO, and later from invasive ventilation.

This case shows that placement of an endobronchial valve using flexible bronchoscopy to treat BPF is possible, that it can be performed at the bedside in even the sickest patients, and that such treatment may facilitate successful weaning from extracorporeal and invasive ventilatory support.

Competing interests
None declared.

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