

CASE REPORT

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Our interventional lung assist experience with tracheoesophageal fistula in intensive care unit: A case report

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Abstract

A tracheoesophageal fistula (TEF) is a congenital or acquired communication between the trachea and esophagus. Acquired TEF is a rare but serious clinical entity. Here, we report the treatment of a patient with interventional lung assist (iLA) in the course of TEF -related hypercarbia and respiratory acidosis as a result of failure of protective ventilation strategy and his outcome after treatment. iLA contains a specially designed low resistance lung membrane, which uses the pressure difference between the arterial and venous circulation. This system enables the use of high airway pressures for oxygenation in combination with very low tidal volumes to avoid ventilator-induced lung injury and this gives time to patient for lung recovery.

Keywords: Tracheoesophageal fistula, interventional lung assist, hypercapnia

Introduction

A tracheoesophageal fistula (TEF) is a congenital or acquired fistula between the trachea and esophagus. Acquired TEF occur secondary to malignant disease or non-malignant conditions like infection, iatrogenic and trauma[1]. TEF from malignancy causes for more than 50% of acquired TEFs and the primary tumor is mainly located in the esophagus (78%). The other locations of the primary tumor are lung (16%), trachea, larynx, thyroid and lymph nodes. Etiologic factors of the benign TEF are trauma like postintubation injuries, infections, prior surgery, iatrogenic injuries, and AIDS[2,3].

TEF is a rare but serious clinical entity. The major complications are mediastinitis and pulmonary sepsis due to gastrointestinal secretion leakage to respiratory system. Spontaneous closure of small fistulas may ensue, but larger fistulas must be treated [1,4]. Various procedures have been tried, including surgical repair, endoprostheses, endo-bronchial gluing and tracheal stents[5,6]. Other workers have shown good long-term results of palliative treatment with covered expandable metallic stents [7]. Most effective treatment of TEF is esophageal bypass or stenting rather than surgical closure. After Song et al. reported successful experiences with the use of self-expandable metallic stents

(SEMS) different types of stents have been used widely. SEMS has a lower complication rate, mortality and morbidity compared to other palliative methods. The endoscopic methods' success rate of closure of TEF varies from 87– 100% [8]. Esophageal stent may lead to dyspnea, tracheobronchial stricture or tracheal narrowing and to overcome this problem tracheobronchial stenting may be done. But in this situation pressure against tracheobronchial wall may cause necrosis[9].

There are various techniques of blocking the TEF and the main stem bronchus simultaneously like usage of DLT (double lumen endotracheal tube) for a large acquired TEF or bronchial occluder combined with ETT(endotracheal tube) . Another easy but effective alternative technique of single-lung ventilation is to deliberately slide a general tracheal tube into the one side of main stem bronchus [10].

ILA: To prevent mechanical ventilation induced iatrogenic lung injury new strategies that include the maintenance of spontaneous breathing, avoidance of high inspiratory pressures and the application of positive end-expiratory pressure (PEEP) to open the collapsed lung tissue and to ensure oxygenation have been established [11]. But conventional treatment modalities with lung protective ventilation is often not sufficient to prevent life-threatening hypoxemia or hypercapnia. This is not only true for severe ARDS (adult respiratory distress syndrome) or less severe ALI (acute lung injury) but also in other clinical situations, such as severe asthma or lung cancer in which lung parenchymal capacity is limited.

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First established more than four decades ago, extracorporeal membrane oxygenation (ECMO) is now used in cases of cardiac and respiratory failure widely. The increased use of ECMO during the worldwide H1N1 epidemic brought this technique into broader focus. However, despite recent developments, ECMO remains a very complex and expensive procedure that requires a well-trained team and immediate technical support in any case of problem. Alternative approaches to conventional ECMO aim for the simplification and reduction of complications associated with the use of a pump. Currently, a pumpless extracorporeal lung support system was developed using an arterio-venous bypass into which a gas exchange membrane is integrated ("interventional lung assist" [ILA]). ILA has very limited effect on oxygenation but provides effective CO₂ elimination. In addition to the growing evidence from experimental studies, arterio-venous ILA has been used in different clinical situations, such as severe asthma, transport of patients affected by severe ARDS, trauma, thoracic surgery and as a bridge to lung transplantation [12].

After the concept of a pumpless arterio-venous ECMO was first described by Ohtake et al. [13] new alternative treatment modalities were introduced like ILA (NovaLung GmbH, Talheim, Germany) which uses a low-resistance gas exchange membrane (lung assist device, LAD) interposed between two cannulas that are connected via short tubing that establish an

arterio-venous shunt. The heparin-coated cannulas are basically inserted by Seldinger's technique into the femoral vessels and the gas exchange unit consists of heparin-coated polymethyl pentene (PMP) hollow-fiber mats. The blood passes outside of the hollow-fiber system while the gas phase is located inside. By this way the gas exchange takes place alongside the semipermeable membrane. It is connected to an oxygen supply (10-12 L/min) and gas exchange is driven by the partial pressure gradient of carbon dioxide and oxygen between blood and the gas phase. And the blood flow through the tubing and the gas exchange membrane is merely determined by the difference between arterial and venous blood pressure. An ultrasound flow meter that is connected to the tubing measures blood flow through the device. Flow rates require an average driving pressure difference between 60-80 mmHg to be effective and sufficient decarboxylation should be possible at flow rates of about 1-1,5 L/min.

In comparison to standard ECMO, the main advantages of ILA are the avoidance of pump-related side effects and technical complications, the reduced artificial surface contact and the reduced operating expense. Bein et al. reported an overall incidence of serious (indirect and direct) complications of 24,4% (14) and the direct complications of ILA are mostly associated with arterial and venous cannulation (Table 1). Contraindications for ILA based on complications and working system are low cardiac output, hypotension, and serious peripheral artery disease.

The LAD system should be inspected routinely for thrombotic debris that may occlude blood flow and prevent gas exchange. If blood flow is reduced over time while hemodynamics are stable, an occlusion should be suspected and exchange of the LAD system should be considered. With the excellent follow up the usage time of an ILA device is only approved for use for up to 29 days and a convenient approach for weaning from ILA has been described

by Bein et al. [14]. This approach includes a reduction in the gas supply to the system for 30 minutes, if the ventilator settings are kept well within the limits of a lung-protective approach. Unless gas exchange deteriorates, the extracorporeal circuit can be stopped, and the cannulas are removed. Immediate manual compression of the cannulation site for at least 30 minutes, followed by placement of a pressure bandage for at least 24 hours. Vascular closure with surgical support is rarely needed.

In this case report, we present our ILA experience that was easier than ECMO to use and effective in gas exchange. It becomes particularly important in the ECMO era with lung protection strategy.

Table 1. Direct complications associated with the usage of pumpless interventional lung assist (ILA).

- Bleeding during cannulation
- Hematoma or aneurysm at insertion site
- Ischemia of a lower limb after cannulation
- Compartment syndrome of a lower limb
- Cannula thrombosis
- Hemolysis

Adapted from Bein et al. [14]

Case presentation

A 59 years old man [weight 55 kg, height 165cm and ideal body weight (IBW) 62 kg] had been intubated in another medical center and was referred to our intensive care unit with the diagnosis of tracheoesophageal fistula unknown aetiology. Endoscopic stenting had been tried in this medical center, but after that on bronchoscopy, stent migration and failure of stent had been noted. On thorax CT it was reported that stent displaced left bronchus and active infection signs -milier pneumonia- in both lung lobes.

On admission, he was on mechanical ventilation with the settings of pressure support Synchronized Intermittent Mandatory Ventilation (P-SIMV) over PEEP (PEEP') 24 cmH₂O, frequency 15 1/min., fiO₂ 60%, Positive End Expiratory Pressure (PEEP) 8 cmH₂O, pressure tidal volume (VT) 560 mL and his arterial blood gasses (ABG) analysis revealed respiratory acidosis and hypercapnia (pH 7.23, PCO₂ 99,2 mmHg, PO₂ 148 mmHg, BE 11,4, saturation 98,4%). His blood pressure was 132/66 mmHg, heart rate was 121 1/min. and his body temperature was 37,5°C. Auscultation of the lungs revealed basillary inspiratory and expiratory crackles with bronchospasm bilaterally. His biochemical blood analysis revealed hyperlactatemia and leucocytosis (lactate 2,3 mmol/L, leucocyte count 18500 cells/mm³). Antimicrobial treatment that had been started in another medical center continued and new specimens of blood, urine and deep tracheal aspiration collected for microbiological examinations. After admission ventilation settings were immediately changed as pressure controlled with fiO₂ 50%, PEEP 12 cmH₂O, PEEP' 15 cmH₂O, fr 22, and VT 300 mL, 8 hours after the new ventilatory settings, ABG analysis improved; pH 7.40, PCO₂ 63 mmHg, PO₂ 78,7 mmHg, BE 15,4, sat. 99,3%. As the patient was severely symptomatic and the situation could have progressed to complete obstruction of the trachea, endoscopic tracheal stenting was done 12 hours after admission. 2 days after endoscopic stenting hypercapnia and respiratory acidosis recurred

and bronchoscopy was done. There was a huge gap in the right bronchial wall next to the carina with esophageal stent visible and protruding into the trachea and narrowing the lumen (Figure 1). A new endoscopic expandable metallic stent was inserted covering the TEF.



Figure 1. There was a huge gap in the right bronchial wall next to the carina with esophageal stent visible and protruding into the trachea and narrowing the lumen

However, on admission day 4, the patient again entered hypercapnic and hypoxic state. Despite vigorous ventilatory treatment, his clinical status reached a deadlock and in order to give him a chance for treatment as a last choice we decided to implement the pumpless interventional lung assist device (ILA, NovaLung, Hechingen, Germany). We used the right femoral vein and the left femoral artery, respectively for insertion of a 15 French arterial and 17 French venous cannula by the Seldinger technique (Figure 2).



Figure 2. We used the right femoral vein and the left femoral artery, respectively for insertion of a 15 French arterial and 17 French venous cannula by the Seldinger technique

After insertion, ILA pre-filled with 0,9% NaCl was connected; initial passive blood flow was 1 L 1/min and gas flow (oxygen) in the membrane lung was 12 L 1/min. Because of the heparin-bonded system, systemic anticoagulation with heparin was targeted only to an activated clotting time of 120-150 s. Blood flow through the extracorporeal system was measured by ultrasonography (Blood Flow Monitoring System, NovaLung®, Hechingen, Germany). The targeted mean arterial pressure was 70 mmHg and the minimal cardiac index ensure sufficient blood flow through the membrane lung was 1,5 L/min/m². Norepinephrine infusion (maximum dosage 1,5 µg/ kg/ min) was only administered for 24 hrs after starting ILA to reach this index.

In the first few hours there was a marked decrease in hypercapnia, as well as an advanced hypoxia; therefore, we had to increase both the FiO₂ and PEEP at the beginning. After the second day following ILA implementation we were able first to reduce FiO₂ stepwise, and then PEEP and mean airway pressure over several days. The changes in the arterial blood gases were shown in Table 2.

Table 2. Course of time and changes in respiratory mechanics of the patient on Interventional

	preILA	1h	2h	4h	6h	8h	12	24h	2.gün	3.gün	Post İLA
FiO₂	100	80	70	60	45	40	40	40	40	40	60
PEEP	5	5	5	6	6	6	5	5	5	5	8
PH	6.99	7.06	7.19	7.28	7.35	7.41	7.40	7.39	7.40	7.35	7.23
PaO₂	97	95.5	119	52.2	62.3	62.9	55	75.4	84.8	65.6	74.6
PCO₂	147	127	89.7	75.7	62.5	53.1	53.9	55.2	49.8	58.2	68.4
HCO₃	33.8	34.6	33.5	35	34.1	33.5	33.9	33	30.9	30.3	28
BE	2.4	2.7	4.8	7.7	7.7	8	8.4	7.4	8.3	7.9	6.5
SPO₂	94.2	93.8	97	84.7	93.4	93.3	86.3	94.5	94.6	90.5	92.5

Lung Assist device (ILA).

On day 3 ILA membrane had thrombosis on the upper part (figure 3) and this thrombotic debris enlargement was prevented by the way of heparin infusion. On day 4 arterial catheter is displaced and ILA device was removed. After removal of ILA device ETT

with DLT to occlude right bronchial tree and blocking TEF. Before ILA treatment this attempt to reduce pCO₂ is unsuccessful, but after ILA hypercapnia was reduced efficiently via DLT. No buffer used for treatment of hypercapnia at any time. He was not given

any steroids either as his hemodynamics were not compromised to lead to septic shock any time throughout his treatment.

Discussion

In the last few years, there has been a consensus on the ventilation strategies of lung failure conditions (like ARDS, severe asthma, lung cancer, etc) and on the context of “lung protective ventilation” through which we may face with inevitable hypercapnia [15]. Similarly, in our patient, as a result of this lung protective ventilation strategy, we could not manage to overcome the sustained hypercapnia and we decided to establish the ILA for carbon dioxide removal to offer less aggressive ventilation.

The aim of ILA insertion in this reported case was to allow lung-protective ventilation and to improve gas exchange. This allows the native lung function to be supported and the diseased lung may recover better as artificial ventilation can be downgraded. Accordingly, additional iatrogenic lung injury such as barotrauma and volutrauma caused by mechanical ventilation with high tidal volumes and high peak inspiratory pressures can be reduced. Our case demonstrates that moderate transfer of oxygen and efficient elimination of carbondioxide is well achieved with ILA.

The ILA system is characterized by a novel membrane gas exchange device with optimized blood flow integrated into an arteriovenous heparin-coated bypass. A passive shunt flow established by cannulation of the femoral artery and vein allows effective carbon dioxide extraction and moderate improvement in arterial oxygenation. The oxygen transfer capacity of the ILA is limited mainly by the fact that well oxygenated arterial blood, is fed into the device and therefore only a small additional amount of oxygen can be bound to haemoglobin. In our patient, ILA enabled a safe application of lung protective tidal volume less than 6 ml. kg⁻¹ without provoking severe acidosis, by profound carbondioxide elimination. The combination of very low VT with high PEEP allowed the limitation of plateau pressure at 30 cmH₂O or lower and thus the avoidance of barotrauma. The use of extracorporeal technology to accomplish gas exchange, with/without cardiac support, is based on the premise that ‘lung rest’ facilitates repair and avoids the barotrauma/volutrauma of ventilator management. Interventional, extracorporeal pump-free pulmonary support opens up new possibilities for pulmonary protection. Despite the lack of randomized controlled studies and the possibility of significant risks, results are encouraging.

Conclusion

Using the interventional lung assist (ILA) membrane ventilator as a pumpless extracorporeal carbondioxide elimination method is a modern concept for the treatment of hypercapnia due to respiratory failure which cannot be sufficiently treated by conventional strategies [16]. Today, low-tidal-volume ventilation is a common practice in the treatment of acute lung failure. However, it is often accompanied by hypercapnia and respiratory acidosis. In this context, extracorporeal carbon dioxide elimination could offer an opportunity to avoid potentially dangerous decreases in pH. ILA may provide a sufficient improvement at arterial blood gas parameters in patients with persistent hypoxia and hypercapnia. In patients where sufficient oxygenation by the patient's lungs is still possible but removal of carbon dioxide is not feasible by lung-

protective ventilatory settings, ILA may give an opportunity to ensure even ‘ultraprotective’ ventilation.

Declaration of Conflicting Interests

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Ethical approval

This article does not contain any studies with human participants performed by any of the authors. This is an case report and for the interventions and the procedures informed consent was obtained from the patient or his relatives during the treatment period.

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