Use of Extracorporeal Membrane Oxygenation (ECMO) for Management of Profound Dyspnea Caused by a Mediastinal Goiter

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Abstract

Perioperative management of a large mediastinal mass poses a substantial clinical challenge, particularly during induction of general anesthesia. Critical airway compression with inability to ventilate is a significant threat. We discuss a patient undergoing resection of a complex mediastinal mass who upon induction and attempted intubation had insufficient oxygenation and ventilation. We initiated veno-venous extracorporeal membrane oxygenation allowing for a controlled resection of the complex tumor. She recovered successfully with no complication. Our case exemplifies the importance of multidisciplinary coordination in the perioperative management of the patient with a complex mediastinal mass.

Introduction

The anesthetic management of the patient with a large mediastinal mass poses a significant clinical challenge. The induction of general anesthesia has the potential to evoke tumor-associated compression that may progress to acute respiratory decompensation and/or cardiovascular collapse [1-3]. Furthermore, airway compression can be deceptive; in an asymptomatic patient, it may manifest only during the induction period [4]. As such, a thorough, well-coordinated, multidisciplinary perioperative approach is essential, including appropriate pre-operative contingency planning.

Description of the Case Type

A 60 year old, African–American female with a history of a large thyroid goiter, containing a significant intra-thoracic component, presented with acutely worsening dyspnea, orthopnea, biphasic stridor, and hoarseness. She was evaluated in an outside hospital three months earlier, where she declined surgery and underwent a tracheal stent placement to treat her progressive dyspnea. Computed tomography (CT) imaging of the thorax revealed a very large, heterogeneously thyroid goiter with a significant posterior mediastinal component, extending distally between the trachea and esophagus, as well as inferiorly to the carina (Figure 1). The trachea was tortuous and deviated significantly to the right (Figure 2). The tracheal stent extended from the thoracic inlet to the carina, with mass effect on the posterior aspect of the stent throughout its course, resulting in stent buckling and fracture. There was tracheal luminal narrowing, greatest at the mid-aspect, with a minimum diameter of approximately 8 mm x 5 mm. There was compression and narrowing of the right and left mainstem bronchi. The distal airways were patent, although there was extensive bilateral consolidative and ground glass opacities throughout the lung fields, most significant in the right upper and lower lobes. The great vessels were noted to be normal in both course and caliber.

Surgical and anesthesia subspecialty teams developed a coordinated plan for induction, intubation, possible initiation of extracorporeal membrane oxygenation (ECMO), excision of the tumor and removal of the fractured stent. All team members agreed that cardiothoracic anesthesia, thoracic surgery, cardiovascular surgery, otolaryngology and perfusion specialists would be present for evaluation and treatment upon entry to the operating room. Supplemental helium-oxygen mixture, 6L/min, was administered via nasal cannula. The patient was hemodynamically stable and oxygen saturation by pulse oximetry was 95%. She was placed in semi-Fowler’s position, as she could not tolerate the supine position without significant hypoxia and distress. Standard monitoring cardiac monitoring, pulse oxygen measurement and blood pressure monitoring was established.
Large-bore peripheral intravenous catheters and a right radial arterial catheter were placed. In preparation for awake oral tracheal intubation, intravenous midazolam and fentanyl were carefully titrated to achieve sedation with preserved respiratory function. Oxygen saturation measured by pulse oximetry remained 98%. Topical anesthesia of the airway was achieved with administration of atomized 4% lidocaine solution. Meanwhile, the cardiothoracic surgical team prepped and draped the groin in preparation of veno-venous ECMO, but because of the semi-fowler positioning and the patient’s body habitus, access to the femoral vessels was limited. The perfusion team prepared for VV ECMO and the ECMO circuit was primed.

We loaded a flexible video bronchoscope with an 8.0-mm wire-reinforced endotracheal tube (ETT). We chose the smallest sized ETT that would accommodate our bronchoscope, keeping the option for bronchoscopic intervention including balloon dilatation or stent management available. The patient had sustained end-tidal CO₂, bilateral chest rise and breath sounds, though markedly diminished on the right. Though the bronchoscope could not traverse the distal trachea, a view of bilateral mainstem bronchi revealed profound tracheobronchial narrowing, multiple fractures of the tracheal stent eroding into the airway and a stent fragment occluding the right mainstem bronchus, none of which was amenable to flexible bronchoscopic intervention. Spontaneously breathing, the patient received inhaled induction of anesthesia with sevoflurane in 100% oxygen. However, as the anesthetic depth increased, elevations in peak airway pressure developed along with significant decreases in tidal volume. Improvements in tidal volume could not be achieved with manual-assisted synchronous ventilation. Shortly thereafter, SpO₂ began to decline.

With decreasing oxygenation and difficulty with ventilation, cardiovascular collapse was eminent. At this point, the collaborative decision was made to proceed with initiation of VV ECMO. The patient was placed in the supine position, bilateral femoral venous catheters were placed and VV ECMO was initiated at flow rate of 4 L/min. The SpO₂ improved rapidly. After ensuring hemodynamic and respiratory stability, anesthetic depth was increased with sevoflurane. Vecuronium was administered for neuromuscular blockade. Despite elevated peak inspiratory pressures and decreased tidal volumes, the patient had adequate arterial oxygen saturation.

The otolaryngology team subsequently performed a transverse cervical incision, left paratracheal dissection, and left hemithyroidectomy. Attempts to deliver the mediastinal component of the mediastinal tumor through the cervical incision resulted in complete obstruction of the airway. Despite VV ECMO, the patient was not able to tolerate periods of apnea. A median sternotomy allowed for removal of the 8 cm multilobulated mass extending into the posterior mediastinum. An additional 8 cm subcarinal mass was dissected, and removed in its entirety.

Unfortunately, the fractured and buckled stent continued to pose obstruction to ventilation requiring a plan for removal. The patient underwent extubation, and the thoracic surgeon performed rigid bronchoscopy. A grasper was advanced through the bronchoscope and the proximal edge of the stent was secured. The stent was extracted piecemeal with minimal force. With removal of the mediastinal component of the goiter and the fractured stent, the trachea assumed a normal size and shape immediately. The rigid bronchoscope was removed and the trachea was reintubated by direct laryngoscopy without difficulty. Subsequent flexible bronchoscopy revealed diffuse injury to the bronchial mucosa of the right mainstem bronchus as well as to the trachea from carina to vocal cords (Figure 3). However, there was no evidence of bleeding or perforation. Given the possibility of tracheal or mainstem bronchial inflammation and airway laxity, a deliberate and measured plan for weaning of the ventilator and ECMO was initiated. Again this was a discussion between all the invested surgical and anesthesiology teams.

On the morning of post-operative day 1, the patient was
successfully weaned from VV ECMO and the femoral vein cannulae were removed. Following decannulation, the patient was extubated with good respiratory function post-extubation. The remainder of her hospital course was uneventful. She was discharged on post-operative day 7, with instructions for follow-up with both ENT and thoracic surgery. One year later, the patient is alive and well without any lasting symptoms.

Pathology for the thyroid specimen was reported as multinodular goiter with scarring/hyalinization, calcification, and focal Hurthle cell metaplasia. The mediastinal component was described as multinodular goiter with scarring, hyalinization and calcification.

Discussion

The preoperative assessment is critical in identifying the potential for perioperative respiratory complications. Signs and symptoms elicited on history and physical exam that are predictive of these complications include positional dyspnea/orthopnea, hoarseness, stridor, wheezing, and cyanosis [5]. Bechard et al. [6] demonstrated a positive correlation between signs and symptoms and perioperative respiratory decompression. CT imaging is a standard in preoperative evaluation and is invaluable in gauging tumor location and degree of tracheobronchial compression [7]. A cross-sectional tracheal area of less than 50% of normal diameter can also be predictive of postoperative pulmonary complications [7]. Finally, flow-volume loops obtained by spirometry provide an estimation of dynamic airway flows and obstruction [8]. Specifically, the presence of a mid-expiratory plateau when repositioning from upright to supine, is seen with extrinsic intrathoracic airway obstruction [1]. However, studies of flow-volume loops have shown poor correlation with the degree of airway obstruction [9] and have suggested that upright and supine spirometry may not offer any advantage over identifying signs or symptoms and CT imaging [10]. In our case, the progression of airway obstruction led to atelectasis and infilrates of bilateral lung fields, which would confound spirometry. Additionally, our patient’s acute dyspnea would have prohibited accurate spirometry.

Traditionally, general anesthesia is avoided in patients at significant risk for perioperative respiratory compromise. The induction of general anesthesia escalates extrinsic airway compression by several mechanisms. First, increased abdominal tone in conjunction with decreased inspiratory muscle tone contributes to reductions in lung volume of approximately 500 – 1500 mL. In addition, there is relaxation of the bronchial smooth muscle, increasing the extent of large airway compressibility and thereby the potential for extrinsic compression. Finally, as spontaneous ventilation is diminished, there is blunting of the negative intrathoracic pressure and transpulmonary gradient that is generated by chest wall expansion and caudal excursion of the diaphragm. Airway caliber will be comprised and the effects of extrinsic compression amplified [1].

If general anesthesia is necessary, it is prudent to proceed with a slow induction using short-acting, titratable agents, while maintaining spontaneous ventilation and avoiding the use of neuromuscular blockade [7]. Intravenous anesthetics such as dexmedetomidine and ketamine may have utility in this context, as they have both analgesic and sedative properties with the advantage of producing minimal respiratory depression [11,12]. In addition, helium-oxygen mixtures may be employed in order to promote laminar gas flow distal to points of significant obstruction [8]. Neuromuscular blockade should be avoided until the ability to ventilate with positive pressure is confirmed. However, cardiopulmonary collapse despite maintenance of spontaneous ventilation techniques has been reported in the literature [13].

Airway management is a precarious task in patients with a large mediastinal mass. Traditionally, the ideal modality for securing the airway is awake tracheal intubation [7], using local anesthesia and carefully titrated sedation. The location and degree of tracheobronchial tumor burden dictate the position of the ET. A lesion located in the proximal airway may allow the safe passage of a reinforced ETT distal to the obstruction. However, lesions involving the distal trachea through the mainstem bronchi may hinder passage of an ETT beyond the obstruction and necessitate the use of rigid bronchoscopy [8]. The rigid bronchoscope can be advanced beyond the obstruction and ventilation achieved. If the obstruction extends beyond the mainstem bronchi, conventional intubation may not provide adequate oxygenation or ventilation. Jet ventilation can be a temporizing maneuver. However, jet ventilation can cause increased airway pressure, overexpansion of the lungs, and decreased blood return to the right ventricle thus exacerbating cardiovascular collapse.

We elected to employ VV ECMO as the patient began to demonstrate progressive hypoxia. Case reports describe cannulation for CPB prior to induction of general anesthesia [14,15]. Preinduction cannulation in preparation for ECMO was considered, but not performed for multiple reasons. First, we determined that the optimal location for cannulation would be the femoral veins. Because of our patient’s body habitus, the femoral vein anatomy was not easily visible or palpable in the semi-Fowler position. The patient could not tolerate any flattening at the waist to expose the groin. Positioning the patient in any position beside semi-Fowler position to place femoral vein cannulae would likely have caused tumor compression of the airway, leading to acute airway obstruction and rapid hypoxia before ECMO could be instituted. Safe and accurate placement of the femoral cannulae was achievable after increasing anesthetic depth and gradually repositioning the patient to the supine position. Second, awake cannulation can be painful and anxiety provoking. Though awake intubation can also cause anxiety, our judgment was that awake cannulation would cause more pain, anxiety, hypoxia and rapid demise than proceeding with awake intubation. Slinger et al. [16] contest the utility of CPB as a ‘standby’ measure during the induction of general anesthesia, as cannulation and institution of CPB in an emergent circumstance is not without potential obstacles and may require a substantial duration of time prior to achieving adequate circulation and oxygenation. However, the institution of ECMO has its own potential complications including but not limited to vessel injury, bleeding and thromboembolism. As mentioned previously, the patient’s body habitus and inability to tolerate any position except the semi-Fowler position made approach of cannulation prohibitive if not inevitably complicated or ineffectual. We continuously evaluated the need for ECMO and the optimal timing. The patient was successfully intubated and initially successfully oxygenating and ventilating. We subsequently learned that this was not sustainable. The rate of arterial oxygen desaturation in our patient was not rapid and never reached dangerous levels. The combination of partial ventilation and oxygenation from the airway and the subsequent addition of ECMO allowed for optimal management of the airway, thus averting cardiovascular collapse.

For this clinical scenario the institution ECMO after intubation was the safest plan. In addition, the cardiothoracic surgical service at our institution is very experienced in the placement of ECMO and was
able to cannulate the femoral veins in a timely fashion. Nonetheless, in the setting of a mediastinal mass compressing the airway, femoral vein cannulation or percutaneous placement of guidewires for ECMO prior to induction should be considered, as it was in this case.

In summary, anesthetic management of the patient with a mediastinal mass must be managed on an individualized basis. As exemplified by our case, the clinical worth of a thorough, well-coordinated, multidisciplinary perioperative approach is invaluable. Inadequate preoperative risk assessment and preparation, a poorly executed anesthetic plan or lack of contingencies may ultimately result in critical complications.

References