Case Report

# Rigid Bronchoscopy for Malignant Central Airway Obstruction from Small Cell Lung Cancer Complicated by SVC Syndrome

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Central airway obstruction (CAO) and superior vena cava (SVC) syndrome are potentially life-threatening complications in locally advanced lung cancer. Therapeutic rigid bronchoscopy has become an critical component in the treatment of the lung cancer patients with CAO who are not surgical candidates. However, the technique may pose significant risks in patients with coexisting SVC syndrome, especially, and even more so perhaps in patients over the age of eighty. In this case report, we address the potential risks and known benefits of therapeutic bronchoscopic intervention in an 85-year-old man with small cell lung cancer who presented with acute dyspnea secondary to advanced SVC syndrome and CAO involving the lower trachea and right main bronchus. Emergent therapeutic rigid bronchoscopy resulted in a marked improvement, in dyspnea, atelectasis, and postobstructive pneumonia, allowing rapid administration of systemic chemotherapy.

Key words: bronchoscopy, airway obstruction, superior vena cava syndrome, lung cancer, elderly patients

## Introduction

Patients with advanced stage lung cancer commonly present with a variety of loco-regional complications such as Central airway obstruction (CAO), superior vena cava (SVC), hemoptysis and post obstructive pneumonia. Patients with CAO are usually not candidates for surgical resection, and chemotherapy and/or radiotherapy in the setting of post-obstructive pneumonia may exacerbate the infection. Prognosis is guarded and response to external beam radiation alone in the presence of critical airway narrowing is relatively poor.  $^{1\!\mathrm{)}}$ 

Therapeutic rigid bronchoscopy often provides immediate relief from malignant CAO,<sup>2, 3)</sup> but the risks of intervention warrant careful consideration when patients have significant comorbidities such as a large mediastinal mass, SVC syndrome or very advanced age.<sup>4, 5)</sup> While reports on the coexistence of the SVC syndrome and CAO and their anesthesia-related complications, including periprocedural death, have been previously published,<sup>5)</sup> a case of a patient with clinically overt SVC syndrome and critical CAO who underwent a rigid bronchoscopy has not been previously reported, to our knowledge. We also address the risks and benefits of performing an emergent rigid bronchoscopy in this patient population.

#### **Case Report**

An 85-year-old male who has smoked his entire adult life presented with dyspnea, cough, 20 kg weight loss, facial and neck edema for several months. Vital signs

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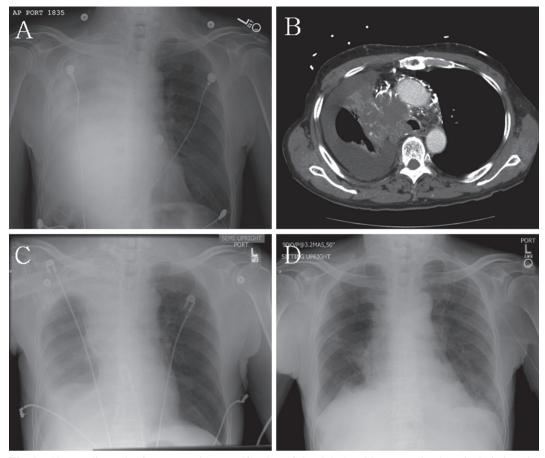


Fig. 1 Chest radiograph of near complete opacification of the right hemithorax on the day of admission (A). Chest computed tomography with intravenous contrast of large mediastinal mass occluding SVC (B). Improved atelectasis of right lung immediately after therapeutic bronchoscopy (C). Chest radiograph of near complete expansion of previous atelectatic right lung, 1 month after therapeutic bronchoscopy (D).

revealed a blood pressure of 150/70 mmHg; heart rate, 115 /min; temperature, 37.2°C; and respiratory rate, 22 / min. Examination showed prominent edema of the face, neck and upper extremities, neck vein distension and multiple engorged dilated vessels over the anterior aspect of the chest. Cervical range of motion and thyromental distance were normal, mouth opening was 2 fingerbreadths, and Mallampati classification was III. No breath sounds were heard over the right hemithorax and expiratory wheezing was heard on the left. Laboratory test results were normal, except for a WBC count of 20, 200 (neutrophils 88%). The initial chest radiograph revealed a near complete opacification of the right hemithorax (Fig. 1A). Chest computed tomography revealed a  $7.3 \times 5.7$  cm large mediastinal and right hilar mass with near complete occlusion of SVC, erosion into the right main stem bronchus and lower trachea with near complete collapse of the right lung (Fig. 1B). Emergent radiation therapy was not recommended because of concerns about worsening the tracheal obstruction by radiation induced edema and because of impending sepsis from post obstructive pneumonia. Emergent rigid bronchoscopy was performed to reestablish airway patency and to prevent respiratory failure in this patient with comorbidities of a large mediastinal mass, SVC syndrome and advanced age. When general anesthesia was induced with intravenous propofol and ramifentanyl, the patient was difficult to ventilate with the bag mask due to severe edema in the oropharynx and larynx. The patient was, therefore, promptly intubated using a 12 mm ventilating rigid bronchoscope (Bryan Corp, Woburn, MA). On bronchoscopic examination, the tumor involved the lower third of the trachea starting at 3 cm above the

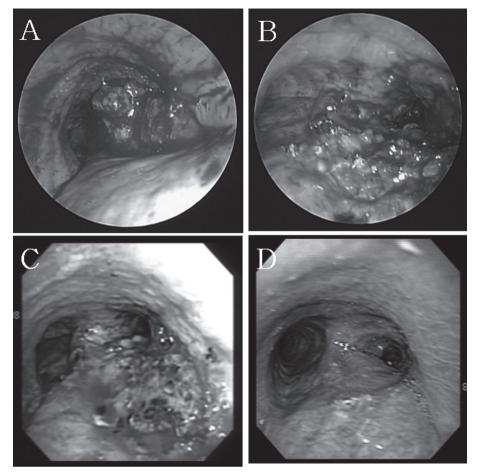


Fig. 2 Serial bronchoscopic findings of malignant central airway obstruction. The bleeding tumor occludes the lower third of trachea by 60%, completely occluding the right main bronchus (A). Immediately after the intervention, the lower tracheal and mainstem bronchi show improved airway patency on rigid bronchoscopy (B) and flexible bronchoscopy views (C). Follow-up flexible bronchoscopy, four weeks after therapeutic bronchoscopy reveals maintained airway patency (D).

main carina and completely occluded the entrance to the right main bronchus (**Fig. 2A**). The carina was infiltrated but the left bronchial tree was normal. Nd:YAG laser coagulation and tumor vaporization using a total of 7120 joules were followed by dilatation and tumor debulking. The patency to the right lower lobe and right middle lobe was established but the right upper lobe was completely occluded by the tumor with no evidence of functional distal airways. Abundant thick secretions were suctioned from the previously obstructed right middle and lower lobes and the bleeding areas were cauterized using an Nd:YAG laser (**Fig. 2B and 2C**). At extubation, there was evidence of laryngeal edema so we intubated the patient with a 7.5 mm endotracheal tube to avoid postoperative respiratory failure. Corticosteroids and ventilatory support were provided overnight in the intensive care unit, and the patient was successfully extubated the next morning. His chest radiograph showed improved aeration with re-expansion of significant portion of the atelectatic right lung (**Fig. 1C**). The patient initiated chemotherapy for small cell lung cancer (SCLC) confirmed on biopsy. Follow-up bronchoscopy showed patent airways (**Fig. 2D**), and one month later, the chest radiograph showed marked improvement in the right lung atelectasis (**Fig. 1D**). A second cycle of systemic chemotherapy consisting of Etoposide and Carboplatin improved symptoms, quality of life and SVC-related clinical findings further. Unfortunately, seventy-five days after the bronchoscopic intervention, the patient died suddenly at home from a suspected a cardiovascular event.

### Discussion

Many patients with lung cancer are diagnosed at advanced stages and need multimodal therapy including palliative interventions for symptoms caused by distal or loco-regional disease. When CAO is present, as in 20–30 percent of patients with lung cancer, patients may develop severe dyspnea which may progress to respiratory failure or sepsis from post obstructive pneumonia.<sup>6)</sup> In many instances, pneumonia is a contraindication of chemotherapy and is an exclusion criterion in clinical trials.<sup>7)</sup> Emergent rigid bronchoscopy in these instances, however, remains a therapeutic option, even in patients older than age eighty<sup>4)</sup> because it improves functional status, allows initiation of systemic therapy and prolongs survival.<sup>2, 3)</sup>

SVC obstruction by lymph node metastasis into the right paratracheal or precarinal stations or by direct invasion of lung cancer can cause SVC syndrome in up to 10% of newly diagnosed SCLC.<sup>8)</sup> Tumor growth in most cases is gradual, allowing sufficient time to develop adequate collateral circulation; however, many patients eventually develop headache, swelling of the face and neck and sometimes even enter a coma. SVC syndrome is, however, no longer considered an emergency, and intravascular stents are only recommended for relapsed or persistent SVC obstruction after chemotherapy or radiation therapy for SCLC.<sup>9)</sup>

Despite the recognized benefits of therapeutic bronchoscopy, elderly patients with severe SVC syndrome and CAO might be denied palliative bronchoscopic treatment because of lack of physician awareness or concerns about high perioperative risks. General anesthesia in patients with various forms of CAO can result in the failure to maintain airway patency caused by the loss of airway tone and subsequent worsening obstruction.<sup>6)</sup> In patients with SVC syndrome, airway management can be difficult because of upper airway edema, hemodynamic instability during general anesthesia and procedure-related bleeding.<sup>10)</sup> Furthermore, the majority of patients with malignant CAO are very ill and has an ASA grade 3 or 4.<sup>11)</sup>

Premedication with opiates or benzodiazepines in the preoperative period raises concerns about airway obstruction; however, anxiety, pain and periprocedural agitation should be avoided because they can lead to tachypnea and high airflow velocity during breathing, which increases the already turbulent flow in the narrowed airways, exacerbates the pressure drop along the stenosis, resulting in labored breathing. When possible, it is best to transport patients in a seated position to the operating room to avoid worsening preexistent airway and vascular obstruction.<sup>12)</sup> This simple maneuver could reduce the likelihood of precipitating severe cough, which in turn can lead to sudden airway obstruction.<sup>11)</sup>

Rigid bronchoscopic intubation can be difficult in patients with a large mediastinal mass SVC symdrome and CAO. The same degree of edema that is seen externally in the face and neck, for example, can be present in the mouth, oropharynx, hypopharynx and larynx.<sup>13)</sup> Prompt and atraumatic insertion of the rigid bronchoscope is critical to prevent further worsening of preexisting upper airway edema related to severe SVC symdrome. By standing at the head of the patient's bed during the induction, the bronchoscopist can be ready and equipped to assure airway control and bypass the obstruction, especially in patients with tracheal lesions.<sup>14)</sup>

Anesthesia of a patient in a supine position can lead to a smaller thoracic cage, a cephalad displacement of the dome of the diaphragm and a reduction in thoracic volume. Patients may be asymptomatic while awake and yet develop critical airway obstruction during anesthesia caused by a reduction in the dimensions of the chest wall, which limits the available space in the airways. Anesthetic agents acting on chest wall muscle tone reduce tracheal distension pressure, thus promoting central airway collapse.<sup>11)</sup> The supine position required for interventions also causes an increase in central blood volume, which can further increase tumor blood volume and size, thus worsening both the SVC syndrome and the CAO. Airway obstruction might be ameliorated by placing patients into a lateral decubitus or sitting position in case adequate ventilation cannot be assured during induction. Loss of airway control has been reported during induction using intravenous or inhalation anesthesia for rigid bronchoscopy.<sup>11, 14, 15)</sup> Muscle relaxants or a dose of intravenous hypnotic such as propofol sufficient to produce apnea can also be disastrous in a patient whose lungs cannot be ventilated because of CAO.<sup>5)</sup>

The induction of anesthesia, especially with drugs that have a marked tendency for hemodynamic depression, is the most dangerous step during a procedure because it may cause cardiovascular instability. Careful hemodynamic monitoring should be performed since these patients are prone to decreased venous return, reduced cardiac output and refractory hypotension.<sup>4, 10)</sup> Acute worsening of symptoms has also been reported to occur as a result of generous fluid administration, and some authors recommend diuresis in patients with clinically overt findings, assuming that diuresis will also decrease tumor volume.<sup>12)</sup> Diuresis, however, may decrease cardiac preload leading to hypotension, worsening a situation already complicated by a compromised venous return.

Spontaneous assisted ventilation shows a relatively good cardiac safety profile in patients undergoing rigid bronchoscopy under general anesthesia.<sup>15)</sup> Although a deeper level of anesthesia is occasionally required to avoid excessive coughing and bucking, secondary to the stimuli produced by maneuver of the bronchoscope in the tracheobronchial tree, light anesthesia allows spontaneous ventilation, helping to maintain the hemodynamic stability and improve the oxygenation.<sup>15)</sup> Neuromuscular blocking agents should be avoided because their use often eliminates airway muscular tone that helps maintain airway patency, and may result in prolonged weakness leading to respiratory failure post operatively.<sup>14)</sup> Positive pressure ventilation and deep anesthesia should also be avoided, if possible, in order to preserve a normal transpulmonary pressure gradient and maintain airway patency during spontaneous inspiration.<sup>5)</sup> When the distending pressure gradient is abolished and the muscles of the chest wall and airways are relaxed, the weight of a mediastinal mass and intraluminal tumor may cause refractory airway obstruction. Positive pressure ventilation will increase the flow velocity and turbulent flow past the region of stenosis. Subsequently, the laminar flow pattern cannot be resumed, resulting in ineffective ventilation of the distal airways and loss of effective gas exchange.13)

In conclusion, coexisting SVC syndrome and CAO can cause life-threatening complications at any time during anesthesia in elderly patients with lung cancer undergoing rigid bronchoscopy. Working together as a team, anesthesiologists and bronchoscopists can anticipate these complications and institute precautionary measures to assure a safe and effective intervention.

## References

- Slawson RG, Scott RM. Radiation therapy in bronchogenic carcinoma. Radiology 1979; 132: 175–6.
- 2) Colt HG, Harrell JH. Therapeutic rigid bronchoscopy allows level of care changes in patients with acute respiratory failure from central airway obstruction. Chest 1997; **112**: 202–20.
- 3) Jeon K, Kim H, Yu CM, Koh WJ, Suh GY, et al. Rigid bronchoscopic intervention in patients with respiratory failure caused by malignant central airway obstruction. J Thorac Oncol 2006; 1: 319–23.
- Davoudi M, Shakkottai S, Colt HG. Safety of therapeutic rigid bronchoscopy in people aged 80 and older: a retrospective cohort analysis. J Am Geriatr Soc 2008; 56: 943–4.
- Mackie AM, Watson CB. Anaesthesia and mediastinal masses. A case report and review of the literature. Anaesthesia 1984; 39: 899–903.
- 6) Ernst A, Feller-Kopman D, Becker HD, Mehta AC. Central airway obstruction. Am J Respir Crit Care Med 2004; 169: 1278–97.
- 7) Thomas P, Robinet G, Gouva S, Fournel P, Lena H, et al. Randomized multicentric phase II study of carboplatin/gemcitabine and cisplatin/vinorelbine in advanced non-small cell lung cancer GFPC 99-01 study. Lung Cancer 2006; **51**: 105–14.
- 8) Baker GL, Barnes HJ. Superior vena cava syndrome: etiology, diagnosis, and treatment. Am J Crit Care 1992; 1: 54–64.
- 9) Rowell NP, Gleeson FV. Steroids, radiotherapy, chemotherapy and stents for superior vena caval obstruction in carcinoma of the bronchus: a systematic review. Clin Oncol (R Coll Radiol) 2002; 14: 338–51.
- Narang S, Harte BH, Body SC. Anesthesia for patients with a mediastinal mass. Anesthesiol Clin North America 2001; 19: 559–79.
- Conacher ID. Anaesthesia and tracheobronchial stenting for central airway obstruction in adults. Br J Anaesth 2003; 90: 367–74.
- Pullerits J, Holzman R. Anesthesia for patients with mediastinal masses. Can J Anaesth 1989; 36: 681–8.
- Sibert KS, Biondi JW, Hirsch NP. Spontaneous respiration during thoracotomy in a patient with a mediastinal mass. Anesth Analg 1987; 66: 904–7.
- 14) McMahon CC, Rainey L, Fulton B, Conacher ID. Central airway compression: Anaesthetic and intensive care consequences. Anaesthesia 1997; 52: 158–62.
- 15) Perrin G, Colt HG, Martin C, Mak MA, Dumon JF, et al. Safety of interventional rigid bronchoscopy using intravenous anesthesia and spontaneous assisted ventilation. A prospective study. Chest 1992; 102: 1526–30.