Anesthetic management of superior vena cava syndrome due to anterior mediastinal mass

Kapil Chaudhary^{1,2}, Anshu Gupta³, Sonia Wadhawan¹, Divya Jain⁴, Poonam Bhadoria¹

Department of Anesthesiology and Intensive Care, ¹Maulana Azad Medical College and Associated Hospitals, ² Current affliation: G B Pant Hospital & associated MAMC ³Hindu Rao Hospital, New Delhi, ⁴Postgraduate Institute of Medical Education and Research, Chandigarh, India

Abstract

Anesthetic management of superior vena cava syndrome carries a possible risk of life-threatening complications such as cardiovascular collapse and complete airway obstruction during anesthesia. Superior vena cava syndrome results from the enlargement of a mediastinal mass and consequent compression of mediastinal structures resulting in impaired blood flow from superior vena cava to the right atrium and venous congestion of face and upper extremity. We report the successful anesthetic management of a 42-year-old man with superior vena cava syndrome posted for cervical lymph node biopsy.

Key words: Anesthesia, preoperative assessment, superior vena cava syndrome

Introduction

Superior vena cava syndrome (SVCS) was first described by William Hunter in 1757. The syndrome results from the impairment of blood flow through the superior vena cava (SVC) to the right atrium. The superior vena caval obstruction (SVCO) may be caused by a mediastinal mass enlargement, which may be either malignant or nonmalignant. It poses a challenge to the anesthesiologist due to the risk of life-threatening complications such as cardiovascular collapse and complete airway obstruction during anesthesia. [1-4]

We report the successful anesthetic management of a 42-yearold man with SVCS posted for cervical lymph node biopsy. The patient had insignificant preoperative clinical and radiological findings. However, significant airway obstruction occurred after the induction of anesthesia and positioning for surgery.

Address for correspondence: Dr. Kapil Chaudhary,

Department of Anaesthesiology and Intensive Care, G B Pant Hospital and associated Maulana Azad Medical College, New Delhi 110 002, India. E-mail: kapsdr@gmail.com

Access this article online	
Quick Response Code:	Website: www.joacp.org
	DOI: 10.4103/0970-9185.94910

Case Report

A 60-kg, 42-year-man presented with complaints of swelling over upper part of the body. The swelling was initially on face and it gradually and progressively increased over 20 days to the neck, chest, and both upper arms. There was no history of swelling of lips or tongue. The patient had no complaints suggestive of dyspnea, dysphagia, hemoptysis, and chest pain.

Examination of the patient revealed swelling over face, neck, chest, and upper arms along with dilated veins over the thorax and cervical lymphadenopathy. Airway examination suggested difficult intubation due to a short neck, modified Mallampatti grade IV, and decreased submandibular space compliance with decreased sternomental and thyromental distances [Figure 1]. Mouth opening was, however, adequate.

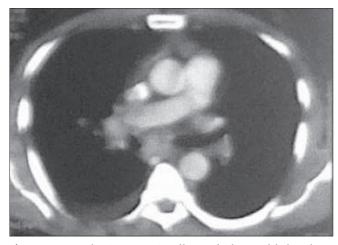
All investigations including hemogram, renal function tests, liver function tests, and serum electrolytes were normal. Contrast-enhanced computed tomography (CT) showed features suggestive of a right hilar mass causing marked attenuation of right upper lobe bronchus [Figure 2] with enlarged mediastinal lymph nodes compressing SVC with, development of few chest wall collaterals and minimal right-side pleural effusion. Another mass of approximately 1 × 1 cm size, causing slight occlusion of the tracheal lumen (less than 10%), was visualized slightly above the carina. The diagnosis of mediastinal lymphadenopathy with SVCS possibly due to lymphoma was established.

The patient was scheduled for cervical lymph node biopsy. The surgeons anticipated difficulty in lymph node biopsy under local anesthesia as there was swelling over the upper part of body and the lymph nodes were deeply seated. General anesthesia with awake nasal fiberoptic intubation was planned. The patient was premedicated with oral ranitidine 150 mg and dexamethasone 8 mg intravenously (IV) at night prior to surgery and on the morning of surgery as well as oral metoclopramide 10 mg on the morning of surgery. Preparation for awake fiberoptic intubation was done with glycopyrrolate 0.2 mg intramuscularly 45 min prior to surgery, 0.5% xylometazoline nasal drops, nebulization with 4 ml of 4% lignocaine, and gargling twice with 6 ml of 2% viscous lignocaine. The patient was shifted to the operating room in the semi-upright position. The baseline heart rate (HR), non-invasive blood pressure (NIBP), and oxygen saturation (SpO₂) were within normal limits. A 16-G IV cannula was placed in the lower extremity. The patient was preloaded with 500 ml of crystalloid, and fentanyl 100 mcg (50 + 50 mcg) was given IV prior to the procedure. Fiberoptic visualization revealed edematous epiglottis and vocal cords and narrowed glottic opening [Figure 3], but the trachea appeared normal. The trachea was intubated with an armored 7-mm-internal diameter endotracheal tube, which was placed approximately 3 cm above the carina. Anesthesia was induced with propofol 100 mg IV, and after ensuring adequate ventilation, rocuronium 40 mg IV was given to facilitate endotracheal intubation with a cuffed tube. The endotracheal tube was fixed at the 25-cm mark, and bilaterally equal air entry was ensured. Anesthesia was maintained on oxygen (33%), nitrous oxide (67%), and isoflurane on controlled ventilation. The patient was positioned head-up, with slight extension of neck and head turned to the left side for surgery. On turning the head, the oxygen saturation suddenly decreased to 88%. This was associated with a sudden decrease in end-tidal CO₂ (18 mm Hg), increase in airway pressures (38 cm H₂O), and decrease in compliance of breathing bag. The circuit and connections were checked immediately, and the patient was simultaneously auscultated. Auscultation revealed a marked decrease in air entry bilaterally. The inspired oxygen fraction was increased to 1.0, and the patient's head was immediately placed in the neutral position without neck extension. This immediately increased the oxygen saturation to 96% on pulse oximetry, improved bag compliance, normalized airway pressures (22 cm H₂O), and increased end tidal CO₂ to 30 mm Hg. Repeat fiberoptic visualization revealed an apparently normal tracheal lumen in the neutral position. However, when the patient was again positioned for surgery, compression of the tracheal lumen distal to the tip of the endotracheal tube was visualized. The tube was passed further down under vision beyond the obstruction. The tube was fixed at the 26-cm mark, and surgery proceeded uneventfully in the desired surgical position.

The intraoperative course was uneventful with minimal blood loss. After antagonism of neuromuscular blockade, the patient was extubated over the Frova[™] intubating stylet (Cook Critical Care, Bloomington, IN) when fully awake with adequate respiratory efforts and stable vitals. The patient was observed in the operating room in propped up position for half an hour. Oxygen was given via a facemask, and the



Figure 1: Patient with short neck, facial edema, and decreased submental and thyromental distances



 $\textbf{Figure 2:} \ \mathsf{CT} \ \mathsf{scan} \ \mathsf{showing} \ \mathsf{attenuation} \ \mathsf{of} \ \mathsf{lumen} \ \mathsf{of} \ \mathsf{right} \ \mathsf{upper} \ \mathsf{lobe} \ \mathsf{bronchus}$

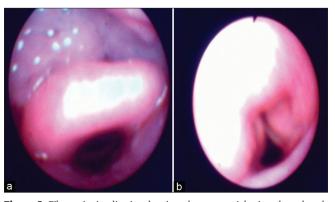


Figure 3: Fiberoptic visualization showing edematous epiglottis and vocal cords and narrowed glottic opening

patient was nebulized with adrenaline to reduce the airway edema. He was then shifted to intensive care unit where he was nursed in the sitting position, given supplemental oxygen through the facemask, nebulized with adrenaline, and given supplemental steroids. The patient remained comfortable and maintained vitals and was shifted to the ward after 24 hours. The postoperative course in the ward was uneventful. The biopsy of the cervical lymph node confirmed the diagnosis of lymphoma, and the patient was planned for chemotherapy.

Discussion

SVCS results from impaired blood flow through the SVC to the right atrium. The most common causes are malignancy (85%), mainly bronchial carcinoma (68%) and malignant lymphoma (6%). The nonmalignant causes (15%) are attributed to idiopathic fibrosing mediastinitis, SVC thrombosis, tubercular mediastinitis, histoplasmosis, and thrombosis associated with intracaval catheters, pacemaker wires, or implantable cardioverter-defibrillators. [5-7] The common signs and symptoms are dyspnea, facial and neck swelling, venous distention of the neck and chest wall, wheezing, stridor, nasal stuffiness, and dry nonproductive cough. Less commonly, patients may complain of chest pain, dysphagia, hoarseness, headache, and other features suggestive of increased intracranial tension. The symptoms get worse with leaning forward, coughing, or lying down. The severity of the syndrome depends on the rapidity of the onset of obstruction and its location.^[8] When the obstruction is slow and progressive, collateral pathways develop and symptoms are usually mild or absent as in this case.

Awake fiberoptic intubation, with an armored tube, is the technique of choice as the tracheal lumen can get compressed on induction of anesthesia. [9] Fiberoptic visualization helps in defining the site and degree of obstruction and tracheal narrowing. The cause of airway obstruction after the administration of muscle relaxant can be easily identified, and the final positioning for surgery can be done under its guidance. The airway edema and the possibility of airway collapse necessitate avoidance of heavy sedation in these patients and premedication is limited to antisialagogues. [7] The head-up position is helpful in reducing airway edema, and so it was maintained perioperatively in our patient. [7]

An IV line in the upper extremity is contraindicated in view of long and unpredictable circulation time and as blood return from the upper body can be interrupted by SVCO. [10] Preload augmentation should be considered in these patients as the mediastinal mass may cause a significant compression of SVC, reducing preload and cardiac output. [8] A 16-G IV cannula was placed in lower extremity in this patient. It is

recommended that in all patients with more than 50% reduction in caliber of the airway preoperatively, femoral vessels should be cannulated in the preparation of cardiopulmonary bypass before the induction of general anesthesia. [11] The preoperative radiological findings of <10% narrowing of tracheal lumen in this patient did not warrant such a preparation.

The obstructed venous drainage of the upper body may cause an increase in intracranial pressure compromising cerebral perfusion pressure. To prevent this, hypotension was avoided and mean arterial blood pressure maintained close to the preinduction levels. The additional protective effect of inhaled anesthetic agents (isoflurane in this case) in reducing cerebral metabolic rate^[8] was used along with the head-up position to prevent such an increase in intracranial pressure.

Positioning for surgery (slight extension and leftward tilt of the neck) resulted in a sudden decrease in oxygen saturation with features suggestive of airway obstruction. Immediate fiberscope visualization revealed compression of tracheal lumen distal to the tip of the tube. The passage of tube beyond the obstruction improved the oxygen saturation and end-tidal CO₂. The neck tilt may have possibly caused the swelling over the trachea to compress the tracheal lumen. The distortion of the trachea by excessive rotation or compression by the enlarged lymph nodes was a possibility in our case. Induction of general anesthesia has been found to exacerbate the extrinsic airway compression by decreasing lung volumes and relaxing bronchial smooth muscles.^[12] It could have been further exacerbated by neuromuscular blockade and positive pressure ventilation, which reduces normal transpleural gradients, causing narrowing of large caliber airways. [13]

General anesthesia in patients with SVCS is associated with high morbidity and mortality rates. [3,11,14,15] Increased risks of difficult intubation along with airway edema, obstruction, and vocal cord paralysis have been associated with induction and maintenance of general anesthesia in patients with SVCO.[3,16] Severe hemodynamic compromise, secondary to compression of the heart and great vessels, may be there in patients with anterior mediastinal mass with SVCO, especially parturients. [16,17] The hemodynamic instability may be further exacerbated by positive pressure ventilation by an increase in intrathoracic pressure and a rapid decrease in venous return. [16,17] Avoidance of general anesthesia and maintenance of spontaneous ventilation should be favored, wherever feasible. The surgeons anticipated difficulty in biopsy under local or regional anesthesia without adequate muscle relaxation, as the lymph nodes were deep seated and there was edema over the upper body, and thus requested for general anesthesia. The increased risk of bleeding due to venous engorgement, a longer surgical duration, and higher chances of an inconclusive biopsy report were other considerations. The cervical plexus block required for open biopsy has its associated complications such as phrenic nerve block and spinal injection, which can be catastrophic in patients with airway edema, and anticipated airway obstruction when the airway is not secure. Endotracheal intubation was the preferred and safest airway management technique as airway compromise can be better tackled. Maintenance of spontaneous ventilation in intubated cases with airway edema and resultant significant increase in airway resistance can be detrimental, especially when anticipating prolonged duration of surgery.

Perioperative steroids have been used to decrease airway edema. [5,18] Although their role in SVCO is inconclusive, [19] Wan and Bezjak [20] have described steroids to be effective in symptomatic patients of SVCS in whom airway edema is suspected to contribute to the symptoms. Steroids are very useful in decreasing the inflammatory response to tumor invasion and also in decreasing the edema around the tumor, especially in cases of lymphoma. [5] The provisional diagnosis of lymphoma also favored the use of perioperative steroids. Inhalational agents such as sevoflurane and intravenous agents such as etomidate, with minimal effect on hemodynamics should be preferred. In cases with confirmed diagnosis of malignancy, preoperative radiotherapy or chemotherapy should be given to decrease the size of mediastinal mass.

The CT scan of the patient revealed small mass with insignificant tracheal attenuation (<10%). However, the possibility of an increase in size of the mass near carina during the 20 days' time from the CT scan to the surgery cannot be ruled out. Although the obstruction of airway following induction of anesthesia in patients with mediastinal mass is a likely possibility, we could not find any such report in patients with insignificant CT findings.

An integrated team approach with the involvement of anesthesiologist and surgeon to check for any respiratory/cardiovascular compromise in the desired position for surgery during an immediate examination before surgery may be beneficial. This could be an easy method to determine the most appropriate position prior to induction of anesthesia. The visualization of tracheal lumen during positioning for surgery and administration of muscle relaxants (whenever essential), with the help of fiberscope would help in determining the appropriate surgical position and tube length. We suggest that the above steps be routinely followed in all patients with anterior mediastinal mass presenting for surgery under general anesthesia, especially if radiological findings are older than 1 week and/or mass/symptoms are rapidly progressing.

The patients with SVCS may face post-extubation breathing

difficulty due to airway edema. Our patient was extubated over the Frova intubating stylet. Such patients need intensive postoperative monitoring and care especially after diagnostic procedures where cause of obstruction has not been relieved. The likely mechanisms of acute respiratory failure are unique to SVCS and include acute laryngospasm and bronchospasm, impaired respiratory muscle function, and increased airway obstruction by the tumor^[7] and airway instrumentation. Our patient was managed in the intensive care unit in the sitting position, given supplemental oxygen through a facemask, nebulized with adrenaline, and given supplemental steroids.

In summary, this case demonstrates that a significant airway obstruction can occur during general anesthesia in a diagnosed case of anterior mediastinal mass with insignificant CT findings. An integrated team approach with immediate preoperative clinical evaluation and fiberoptic airway evaluation after administration of muscle relaxants in the desired surgical position, along with vigilant monitoring may prove to be sagacious. Regional or local anesthetic techniques, maintenance of spontaneous ventilation, and semi-upright position during surgery should be favored, wherever possible.

References

- Narang S, Harte B, Body S. Anesthesia for patients with a mediastinal mass. Anesthesiol Clin North Am 2001;19:559-79.
- Levin H, Bursztein S, Heifetz M. Cardiac arrest in a child with an anterior mediastinal mass. Anesth Analg 1985;64:129-30.
- Keon TP Death on induction of anesthesia for cervical node biopsy. Anesthesiology 1981;55:471-2.
- Takeda S, Miyoshi S, Omori K, Okumura M, Matsuda H. Surgical rescue for life- threatening hypoxemia caused by a mediastinal tumor. Ann Thorac Surg 1999;68:2324-6.
- Wilson LD, Detterbeck FC, Yahalom J. Clinical practice. Superior vena cava syndrome with malignant causes. N Engl J Med 2007;356:1862-9.
- Seelig MH, Oldenburg WA, Klingler PJ, Odell JA. Superior vena cava syndrome caused by chronic hemodialysis catheters: Autologous reconstruction with a pericardial tube graft. J Vasc Surg 1998;28:556-60.
- Wilson WC, Benumof JL. Anesthesia for thoracic surgery. In: Miller RD, editor. Miller's Anesthesia. 6th ed. Philadelphia: Elsevier Churchill Livingstone; 2005. p. 1922-3.
- Barbeito A, Yosef SB, Lowe JE, Atkins BZ, Mark JB. Unusual case of superior venacava syndrome diagnosed with transesophageal echocardiography. Can J Anesth 2008;55:774-8.
- Morgan GE, Mikhail MS, Murray MJ. Anesthesia for thoracic surgery. In: Morgan GE, Mikhail MS, Murray MJ, editors. Clinical Anesthesiology. New York City, U.S.: McGraw Hill Medical Publishing Division; 2008. p. 612-3.
- Benumoff JL. Anaesthesia for special elective therapeutic procedures In: Benumoff JL, editor. Anaesthesia for thoracic surgery. 2nd ed. Philadelphia: W.B. Saunders; 1995. p. 573-5.
- Goh MH, Liu XY, Goh YS. Anterior mediastinal masses: An anaesthetic challenge. Anaesthesia 1999;54:670-82.
- Szokol JW, Alspach D, Mehta MK, Parilla BV, Liptay MJ. Intermittent airway obstruction and superior vena cava Syndrome in a patient

- with an undiagnosed mediastinal mass after cesarean delivery. Anesth Analg 2003;97:883-4.
- 13. Cone AM, Stott S. Intermittent airway obstruction during anaesthesia in a patient with an undiagnosed anterior mediastinal mass. Anaesth Intensive Care 1994;22:204-8.
- 14. Tonnesen AS, Davis FG. Superior vena caval and bronchial obstruction during anesthesia. Anesthesiology1976;45:91-2.
- 15. Northrip DR, Bohman BK, Tsueda K. Total airway occlusion and superior vena cava syndrome in a child with an anterior mediastinal tumor. Anesth Analg 1986;65:1079-82.
- Buvanendran A, Mohajer P, Pombar X, Tuman KJ. Perioperative management with epidural anesthesia for a parturient with superior vena caval obstruction. Anesth Analg 2004; 98:1160-3.

- 17. Pullarits J, Holzman R. Anaesthesia for patients with mediastinal masses. Can J Anaesth 1989;36:681-8.
- Kumari I, Gupta S, Singhal PP. Superior vena caval syndrome in children. Middle East J Anesthesiol 2006;18:933-8.
- Rowell NP, Gleeson FV. Steroids, radiotherapy, chemotherapy and stents for superior vena caval obstruction in carcinoma of the bronchus. Cochrane Database Syst Rev 2001;4:CD001316.
- Wan JF, Bezjak A. Superior vena cava syndrome. Emerg Med Clin North Am 2009;27:243-55.

How to cite this article: Chaudhary K, Gupta A, Wadhawan S, Jain D, Bhadoria P. Anesthetic management of superior vena cava syndrome due to anterior mediastinal mass. J Anaesthesiol Clin Pharmacol 2012;28:242-6.

Source of Support: Nil, Conflict of Interest: None declared.