CASE CONFERENCE

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Challenging Perioperative Management of a Massive Anterior Mediastinal Mass in a Symptomatic Adult

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THE ANESTHETIC MANAGEMENT of an adult with an anterior mediastinal mass is considered to be extremely high risk due to the possibilities of airway obstruction and/or cardiopulmonary collapse during the perioperative period.[1–3] In this case conference, the perioperative course of a severely symptomatic young man with a giant anterior mediastinal mass is presented followed by 2 expert commentaries. The clinical value of a cohesive multidisciplinary approach to perioperative management and complications is emphasized in this challenging scenario.

CASE SUMMARY

A 24-year-old man with no past medical history presented for biopsy of a newly diagnosed anterior mediastinal mass. The patient initially presented to the referring hospital with 2 weeks of fatigue, fevers, hoarseness, severe coughing episodes, and dyspnea while lying flat. His physical examination was unremarkable. There was no evidence of great vein compression. The patient was unable to lie flat. Computed tomographic chest imaging revealed a giant anterior mediastinal mass with compression of the superior vena cava and right pulmonary artery and greater than 50% compression of the distal trachea. Clinical concern about the risk of a rapid cardiorespiratory decline prompted thoracic surgery consultation for urgent tissue diagnosis to guide further management. The patient was scheduled for urgent left anterior mediastinotomy (Chamberlain procedure).

Given the high operative risk, a thorough multidisciplinary discussion of the operative plan was undertaken among the anesthesia, cardiac surgery, and the thoracic surgery teams. The patient was anxious and expressed a strong reluctance to undergo neuraxial blockade but consented to local anesthesia with sedation, keeping general anesthesia as a rescue option. The anesthetic plan was to maintain sedation with a dexmedetomidine infusion.

On arrival to the operating room, the patient was placed in a comfortable Fowler's position. After standard noninvasive monitoring was established, oxygen was administered via a nonrebreathing mask. After titrated sedation with intravenous midazolam, large-bore peripheral venous access and left radial arterial monitoring were achieved. Further sedation was achieved by gradual titration of a dexmedetomidine infusion.[5] The cardiac surgical team then secured femoral venous and arterial access under local anesthesia. A perfusionist primed a cardiopulmonary bypass (CPB) machine and was immediately available in the operating room. The equipment for rigid bronchoscopy by the thoracic surgeon was also on hand in the operating room.

The thoracic procedure was commenced. Despite serial increases in the dexmedetomidine infusion and a surgical field block with local anesthesia, acceptable surgical conditions proved impossible. The team decided to proceed with induction of general anesthesia.

Following adequate denitrogenation, general anesthesia was induced with titrated intravenous propofol (at a dose of 3 mg/kg) and remifentanil (at a dose of 2 μg/kg) with the goal to achieve acceptable tracheal intubating conditions without neuromuscular blockade.[4] Mask ventilation was not attempted. Prompt direct laryngoscopy yielded a full laryngeal view, and an 8.0-mm endotracheal tube was passed easily through the vocal cords. Just after endotracheal intubation, vigorous coughing ensued that precipitated severe hypotension with a mean arterial pressure of 40 mmHg.

The coughing was aborted immediately by further increasing anesthetic depth with additional titrated propofol and volatile anesthetic. Rapid hemodynamic improvement was achieved with titrated bolus epinephrine and phenylephrine followed by infusion of both agents at doses sufficient to maintain his baseline hemodynamics. General anesthesia was maintained

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with titrated desflurane in oxygen. Since positive-pressure ventilation was not associated with hemodynamic instability, the patient was placed on standard mechanical ventilation.

Surgical dissection subsequently was completed via a left parasternal incision at the level of the second rib to access the aortopulmonary window. Sufficient tissue biopsies were obtained for histologic examination and analysis. The adequacy of the biopsy specimens for diagnosis was confirmed with the pathologist by the attending thoracic surgeon. Surgical closure was uneventful. The total operative time was 60 minutes. Given the hemodynamic instability associated with coughing during anesthetic induction and the tumor extent, a collaborative decision was reached to continue postoperative care in the intensive care unit with the patient sedated on mechanical ventilation.

The final histology of the mediastinal tumor was read as T-cell lymphoblastic lymphoma, an aggressive type of non-Hodgkin's lymphoma that is extremely responsive to combination chemotherapy.[6,7] The patient was started urgently on an institutional chemotherapy protocol for this tumor that included daunorubicin, vincristine and methylprednisolone. Furthermore, to prevent future relapse in the central nervous system, the patient also was subjected to prophylaxis with intrathecal chemotherapy.[6,7] There was a dramatic response with marked reduction in tumor size evident on serial chest imaging from the second postoperative day onwards.

The remainder of his hospital course was unremarkable. After significant tumor shrinkage due to chemotherapy, the patient underwent successful tracheal extubation during the second postoperative day. Following successful completion of his inpatient chemotherapy, he was discharged home in a stable condition. His further oncologic management has been continued on an outpatient basis.

CASE DISCUSSION

Although patients with large anterior mediastinal masses are recognized as high risk for cardiac and/or pulmonary collapse, recent evidence suggests that the contemporary risk may be lower, especially when comparing adults to pediatric patients.[8–10] Identified important risk factors for perioperative cardiopulmonary instability include a large mass, severe symptomatology, and evidence of cardiac, great vessel, and/or pulmonary compression.[9–11]

All of these major risk factors were present in this patient. Given this high risk, prompt management is indicated: The options include surgical intervention, chemotherapy, and/or mediastinal radiation, depending on the diagnosis. The differential diagnosis of anterior mediastinal masses includes retrosternal goiter, thymomas, lymphomas, germ cell tumors, and various cysts.[10–13] Since these tumors often are highly responsive to specific therapeutic intervention, an accurate diagnosis remains paramount in importance to guide therapy. Although advances in radiographic imaging may provide suggestive findings, tissue biopsy typically is required for histologic examination to secure the definitive diagnosis.[12–15] In cases of suspected lymphoma or germ cell tumors, the sensitivity and specificity of the diagnostic biopsy specimen significantly improve with sample size.[12–16] Therefore, surgical biopsy is preferred to secure adequate tumor sampling for definitive histologic diagnosis. This management approach explains the presentation of these high-risk patients to the operating room. As in this case, it remains essential to confirm that the biopsy specimens are secure and adequate for tissue diagnosis prior to completing the surgical procedure given the associated high risks undertaken.[16]

The cardiopulmonary risks in this setting depend on tumor extent and adjacent structure compression, as outlined earlier. Dynamic compression of the trachea, major bronchi, heart chambers, and/or great vessels can precipitate eventual cardiopulmonary compromise.[17,18] This dynamic compression of vital structures in a tight mediastinum may be precipitated by multiple factors including changes in patient position, coughing, positive-pressure ventilation, and neuromuscular blockade.[1,2,8–10,18,19] It is likely that hemodynamic instability in this setting may have more than 1 mechanism.

A recurring theme in the literature is the preservation of spontaneous ventilation during induction of general anesthesia in this high-risk setting.[2,8] Despite the recommendation of this anesthetic approach, clinical reports demonstrate both cardiopulmonary collapse with spontaneous ventilation techniques and the safe application of positive-pressure ventilation in this tight mediastinum scenario.[1,18,20] Anesthetic drugs that have been selected for preservation of spontaneous ventilation in patients with anterior mediastinal masses include the volatile anesthetics, propofol, dexmedetomidine, and ketamine.[5,8–10,19–21] Titrated sedation with dexemedetomidine and local anesthesia has been used successfully in adult patients undergoing anterior mediastinotomy.[21] Despite a similar initial anesthetic plan, factors such as the patient's anxiety level and inability to tolerate changes in table position mandated conversion to general anesthesia in the present case.

Given the possibility and risks of general anesthesia in this setting, thorough preoperative multidisciplinary discussion and contingency planning remain essential in these high-risk cases. A cohesive, comprehensive, and flexible team approach to patient management allows for seamless preparation and management of these challenging cases. Although rescue with percutaneous CPB rarely is required, it should be strongly considered in symptomatic patients with mediastinal masses.[2,3,8,22,23] This rescue application of CPB must be distinguished from its application to provide cardiopulmonary support to facilitate therapeutic intervention for a mediastinal mass such as resection or endovascular intervention.[24,25] In the present case, the preoperative team discussion favored immediate availability of rescue CPB with secured percutaneous access, given the highly symptomatic presentation of this giant anterior mediastinal mass with significant airway and great vessel compression. This conservative approach was selected, given the risk of rapid cardiopulmonary collapse from multiple mechanisms as outlined earlier.

Despite a brief period of profound hemodynamic instability during induction of general anesthesia, this patient never required initiation of rescue CPB. This hemodynamic instability was precipitated by acute increases in intrathoracic pressure due to coughing after endotracheal intubation. The severe hypotension disappeared with obliteration of his