Patient with Posterior Mediastinal Mass Requiring Urgent Cardiopulmonary Bypass

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THE literature on anesthetic concerns and perioperative care of patients with mediastinal masses has focused almost exclusively on *anterior* mediastinal masses. Posterior mediastinal masses traditionally have been suggested to carry a low risk of anesthetic implications.[1] We present the case of a patient with a *posterior* mediastinal mass who experienced hemodynamic and respiratory decompensation upon induction of general anesthesia and required urgent transition to cardiopulmonary bypass (CPB).

Our case illustrates the importance of real-time imaging provided by transesophageal echocardiography (TEE) in explaining the etiology of intractable hemodynamic instability and a new finding of significant pericardial and left pleural effusion not seen on preoperative imaging. TEE made clear the urgent need to initiate CPB because it revealed a near total compression of the left atrium, which was obstructing delivery of volume to the left ventricle. In addition, this case demonstrates the inadequacy of "stand-by" CPB and the need for every institution to establish an interdisciplinary team to develop, before surgery, a formal plan for the perioperative care of patients with mediastinal masses.

CASE REPORT

A 45-yr-old, previously healthy woman was transferred to our facility for further management of her mediastinal mass. Six weeks before this admission, she had presented to her primary care provider with shortness of breath, cough, and intermittent low-grade fever. At that time, she received a clinical diagnosis of pneumonia and was prescribed a course of antibiotics and corticosteroids. Upon follow-up, her presenting symptoms had not improved, and she was experiencing dysphagia and right-side chest pain. A chest radiogram showed a large mediastinal mass.

Subsequent computed tomography scanning (fig. 1A and 1B) showed a 17-cm × 15-cm × 13-cm posterior mediastinal mass extending from the lateral right hemidiaphragm to the left upper chest wall. The scan showed that the mass displaced the trachea, mainstem bronchi, and right lung and widely splayed the tracheal carina. External compression of the airway was minimal, and at no point was the lumen caliber of the trachea or mainstem bronchi diminished significantly. A significant right pleural effusion was present, and a small amount of fluid was noted posterior to the left lung. The heart was shifted to the left, and encroachment of the left atrium by the mass could be seen. No pericardial effusion was present. Anatomic distortion and stretching of the pulmonary arteries and veins, the superior vena cava, and the proximal aorta were noted, although no significant compression of these vessels was present. There was almost complete compression of the esophagus in the thorax. No encasement or tissue invasion of thoracic structures by the mass was apparent.

Upon admission to our institution, the patient was hemodynamically stable and maintaining adequate oxygen saturation on 2 l/min O_2 by nasal cannula. In an upright, 90-degree–angle sitting position, where she reported she was most comfortable and experiencing the fewest symptoms, she was mildly short of breath but had no stridor. When supine, she experienced slightly increased shortness of breath and significant right-side chest pain, and the turbulent sounds associated with her breathing worsened. We did not directly examine the patient in the right or left lateral position before surgery because she reported experiencing increased chest wall pain when in the lateral position. She had no upper body edema and reported no history of syncope or other signs of hemodynamic instability. The electrocardiogram showed sinus tachycardia.

⊕ Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are available in both the HTML and PDF versions of this article. Links to the digital files are provided in the HTML text of this article on the Journal's Web site (www.anesthesiology.org).
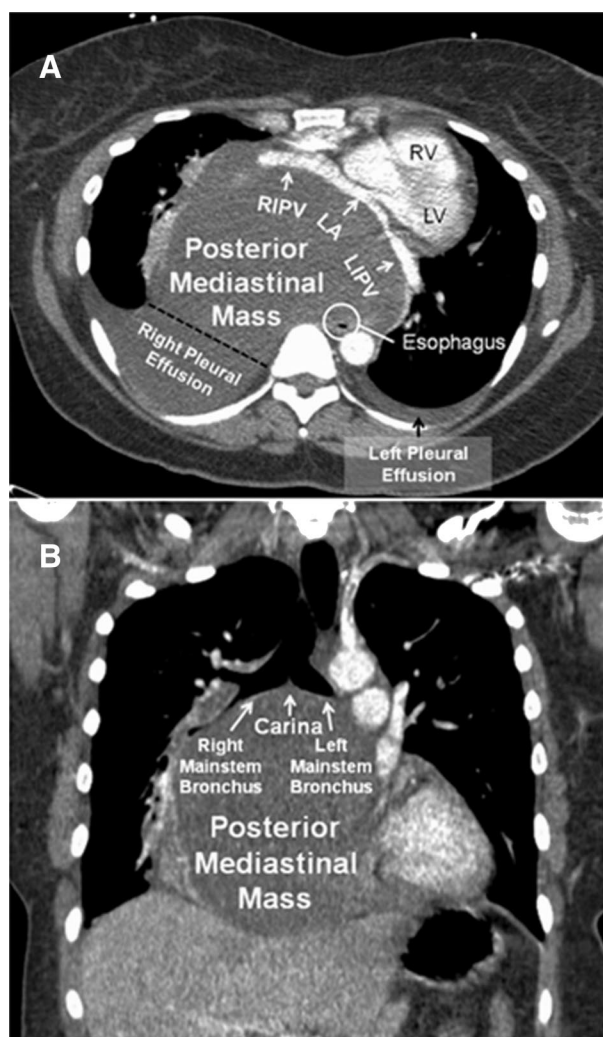
Fig. 1. (*A*) Transverse section of a computed tomography scan with intravenous contrast taken 1 week before surgery showing a large posterior mediastinal mass displacing the heart and encroaching on the left atrium (LA) and ventricle (LV). Patent right and left inferior pulmonary veins (RIPV and LIPV) can be seen at this level, as can the compressed esophagus. Adjacent levels showed minimal compression of the trachea or mainstem bronchi and patency of thoracic vascular structures. (*B*) Coronal section of a computed tomography scan with intravenous contrast showing the posterior mediastinal mass splaying open the carina. Adjacent levels show minimal compression of the tracheobronchial tree. RV = right ventricle.

The interventional radiologists could not perform a computed tomography-guided biopsy because of concern that the patient could not tolerate supine positioning and that the biopsy could result in acute decompression of the mass, as well as uncertainty about the vascularity of the mass. The patient's care was transferred to the thoracic surgery service. She underwent a brief flexible bronchoscopy and esophagogastroduodenoscopy for tissue biopsy and evaluation of her airway and esophagus. Throughout the procedure, a 90-degree sitting position and spontaneous respirations were maintained. Topical anesthetics were applied to the upper airway, and the anesthesiologist performed mask induction

with sevoflurane and bolus doses of intravenous etomidate (2–3 mg) as needed, with a total of 7 mg given during the 15-min procedure. A number 4 *Laryngeal Mask Airway*™ (Laryngeal Mask Company Limited, San Diego, CA) was placed. She remained hemodynamically stable without respiratory compromise throughout the procedure. While performing the bronchoscopy, the surgeon noted a patent airway with normal mucosa showing signs of posterior bulging at the distal trachea and mainstem bronchi during spontaneous breathing. External compression of the esophagus and normal mucosa was noted during esophagogastroduodenoscopy. Unfortunately, the tissue diagnosis was inconclusive.

Video-assisted thoracoscopic surgery for biopsy was scheduled for the following day. Before the surgery, the new attending anesthesiologist assigned for this second case discussed with the attending surgeon the need for CPB initiation before induction of anesthesia. Additional discussion involving the division chief of thoracic surgery resulted in a decision to insert guide wires into the femoral artery and vein under local anesthesia before general anesthesia induction to facilitate rapid intraoperative cannulation and transition to percutaneous femorofemoral CPB, should the patient decompensate during induction. In addition, two perfusionists would be in the operating room with the CPB machine primed and ready.

The anesthesia team transported the patient to the operating room in the sitting position. Standard American Society of Anesthesiologists monitors were placed, and initial blood pressure (112/58 mmHg), heart rate (104 beats/min), respiratory rate (22 breaths/min), and oxygen saturation (99% on 2 l/min O_2 *via* nasal cannula) were recorded. No sedatives or opiates were administered. With local anesthesia, a right radial arterial line was placed, and the surgeon prepared and draped the patient's groin and inserted guide wires into the right femoral artery and vein. An arterial blood gas was recorded at this point (pH 7.49, $PaCO_2$ 36 mmHg, PaO_2 97 mmHg, O_2 saturation 98%). After preoxygenation with 100% oxygen by facemask, anesthesia was induced with sevoflurane in 100% oxygen followed by atraumatic tracheal intubation with a standard 8.0-mm cuffed Mallinckrodt® (Juarez, Mexico) endotracheal tube using the GlideScope® GVL (Verathon, Bothell, WA) laryngoscope. The patient was kept in an upright, 90-degree sitting position for induction and intubation because that was the position in which she experienced the fewest symptoms before surgery. Spontaneous ventilation was maintained, and no neuromuscular blockers were given.

Immediately after the patient underwent anesthetic induction and tracheal intubation, while she was still in the sitting position, her hemodynamic and respiratory status deteriorated rapidly. Her blood pressure decreased into the 50–60/30–40 mmHg range, and heart rate was 140 beats/min. The patient's oxygen saturation decreased into the low 80s, and she appeared cyanotic. Significant airway resistance was noted during assistance of spontaneous respirations with bag ventilation. Preload augmentation with fluid and pharmacologic support in the form of boluses of inotropic agents and vasoconstrictors were administered. However, there was no improvement in hemodynamics or oxygenation.

The patient continued to have no response to multiple boluses of epinephrine, norepinephrine, vasopressin, phenylephrine, and fluid administration. A TEE probe was placed
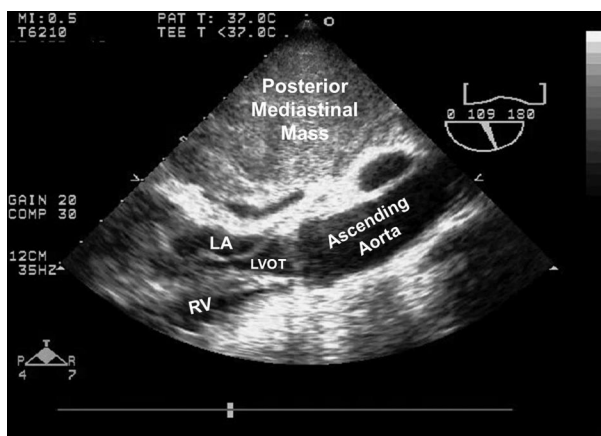
Fig. 2. Transesophageal echocardiogram, midesophageal long-axis view. The left atrium (LA) is severely compressed because of the direct mass effect of the posterior mediastinal mass. LVOT = left ventricular outflow tract; RV = right ventricle.

despite concern of esophageal compression by the mass and passed without resistance. Because of the effect of the mass, the standard TEE views were difficult to obtain and the images were rotated and distorted. Although interpretation of the images was challenging, TEE made clear that there was a direct and near complete compression of the left atrial chamber by the mass, obstructing left atrial filling and, therefore, the delivery of volume to the left ventricle (fig. 2) (see also Supplemental Digital Content 1, http://links.lww.com/ALN/A740, which is a midesophageal long-axis–view TEE video, and Supplemental Digital Content 2, http://links.lww.com/ALN/A741, which is a midesophageal four-chamber–view TEE video, both showing direct compression of the left atrium by the mass). The TEE also revealed new findings of a significant pericardial effusion (see Supplemental Digital Content 3, http://links.lww.com/ALN/A742, which is a deep-transgastric–view TEE video showing the significant pericardial effusion) and a significant left pleural effusion (see Supplemental Digital Content 4, http://links.lww.com/ALN/A743, which is a TEE video showing the significant left pleural effusion), which had not been present on preoperative imaging. TEE showed that the pericardial and pleural effusions superimposed on the direct compression of the left atrium by the mass increased the tamponade effect and worsened the hemodynamic compromise.

The surgeon was instructed to urgently convert the guide wires placed in the femoral vessels to percutaneous femorofemoral bypass, and a second attending anesthesiologist was called to assist with the case. During the surgeon's attempt to dilate the femoral vessels for placement of cannulae, the guide wire from the femoral artery was lost outside the vessel. This required the surgeon to begin a cut-down for access to the femoral artery and vein. Approximately 20 min after the decision to proceed to CPB, percutaneous femorofemoral bypass was initiated. At this point, the patient was reclined to a supine position from the upright sitting position that had been maintained since entering the operating theater, and neuromuscular blockade was administered.

With CPB initiated, the surgeon was able to obtain adequate tissue for diagnosis. A total of 600 ml "straw colored"

fluid was drained from the right side of the chest with placement of the video camera and chest tube. Next, a subxiphoid pericardiocentesis with pericardial drain placement returned 150 ml pericardial effusion. Finally, a tube was placed in the left side of the chest and returned 800 ml fluid. TEE confirmed resolution of the pericardial effusion and pleural effusion on the left (see Supplemental Digital Content 5, http://links.lww.com/ALN/A744, which is a deep transgastric view TEE video after drainage of the pericardial effusion).

In light of the patient's cardiopulmonary deterioration after induction, a transition from CPB to longer term support with an extracorporeal membrane oxygenator was considered the safest course. However, the patient would have been unable to receive urgent radiation treatment had she remained on mechanical support. We anticipated that there would be an improvement in hemodynamic and respiratory function when the pleural and pericardial effusions were drained. A left internal jugular central venous catheter was placed. The patient was weaned from CPB, returned to the upright sitting position, and observed in the operating room for 30 min. She remained hemodynamically stable without inotropic or vasoconstrictor support during this period while receiving positive pressure ventilation. Heparinization was reversed with protamine. The femoral cannulae were removed, and the patient was transferred to the surgical intensive care unit in the sitting position. Radiation therapy was started the next morning after high-grade myxofibrosarcoma was diagnosed via a tissue sample. The patient was neurologically intact after surgery. She was extubated on postoperative day 2 and subsequently discharged home. Three months later, after radiation and chemotherapy, her mass was less than 10% of its size on initial presentation.

Discussion

Our case report illustrates four important points: (1) posterior mediastinal masses can cause hemodynamic and respiratory near collapse requiring urgent CPB; (2) intraoperative TEE provides real-time feedback that is important for explaining intractable hemodynamic instability and guiding intraoperative management, and it can provide new diagnoses (pericardial and left pleural effusions); (3) stand-by CPB is not an adequate rescue measure. Femoral artery and vein cannulae need to be placed before induction to ensure smooth, rapid transition to CPB in cases deemed "unsafe" or of "uncertain" risk of cardiovascular collapse after induction of general anesthesia; and (4) every institution needs to formalize a plan for the perioperative care of patients with mediastinal masses, including a prearranged interdisciplinary team to meet and discuss the case before surgery.

Posterior Mediastinal Masses

Patients with mediastinal masses present unique and formidable challenges for the anesthesiologist. These masses have a well-documented association with severe cardiorespiratory compromise and death at all perioperative stages.[1–13]

The difficulty is compounded by how infrequently the typical anesthesiologist encounters physiologically significant mediastinal masses[14] and by the tremendously variable clinical presentations and physiologic effects, which can differ widely depending upon the size, location, and pathology of a given mass and the thoracic structures affected by the mass.[15]

The literature discussing the anesthetic concerns and perioperative care of patients with mediastinal masses has focused almost exclusively on *anterior* mediastinal masses. Posterior masses traditionally have been suggested to carry a low risk of anesthetic implications. However, our patient with a *posterior* mediastinal mass experienced the hemodynamic and respiratory collapse typically associated with anterior mediastinal masses. To our knowledge, this is the first published report of a posterior mediastinal mass requiring urgent CPB upon induction of anesthesia. Together with the report of cardiopulmonary collapse in a patient with a middle mediastinal mass,[11] this case demonstrates that mediastinal masses outside the anterior compartment demand the same cautious approach recommended for patients with anterior mediastinal masses.

Cardiovascular collapse associated with anterior mediastinal masses typically is caused by compression of the superior vena cava, pulmonary arteries, and/or the right-side heart structures, in addition to airway compression. In contrast, posterior mediastinal masses typically compress posterior structures such as the left atrium and ventricle. The left atrium is particularly susceptible to compression by posterior masses because of its inferoposterior location and increased compressibility relative to the left ventricle.[16]

Compression of the airway or cardiovascular structures in patients with mediastinal masses may be position dependent, and there are reports of dramatic improvement in cardiorespiratory function after repositioning in some patients with anterior[6,17–19] or middle mediastinal masses.[11] It is prudent to determine before surgery the position in which the patient experiences the fewest symptoms and to consider repositioning in the event of intraoperative cardiorespiratory compromise.[14,15,20]

In this case, the patient was certain that she was most comfortable in an upright sitting position and that her symptoms, particularly her chest wall pain, increased in other positions. We intended to induce anesthesia in this "most comfortable" position. When hemodynamic compromise caused by direct compression of the left atrium by the mass was noted intraoperatively, it may have been beneficial to move the patient to a lateral or prone position in an attempt to relieve the compression. However, because of the patient's reported discomfort in the lateral position, the uncertainty that repositioning would improve hemodynamics given the size and posterior location of her mass, and the fact that lateral positioning would have severely limited the surgeon's access to the femoral vessels for cannulation, we opted to proceed to CPB without delay.

Intraoperative TEE

Intraoperative echocardiography proved valuable in the management of this case, both as a diagnostic tool and as a guide for initiating and weaning from CPB. During the critical period of hemodynamic instability, TEE explained the etiology of the cardiovascular compromise and the lack of response to pharmacologic support. A near-total loss of delivery of volume to the left ventricle caused by direct compression of the left atrial chamber by the mass was seen on echo (Supplemental Digital Content 1, http://links.lww.com/ALN/A740, and Supplemental Digital Content 2, http://links.lww.com/ALN/A741). TEE also provided new diagnoses of significant pericardial (Supplemental Digital Content 3, http://links.lww.com/ALN/A742) and significant left pleural (Supplemental Digital Content 4, http://links.lww.com/ALN/A743) effusions not indicated by preoperative imaging and revealed that the pericardial effusion was aggravating the effect caused by the mass's direct compression of the left atrium.

In a patient with known esophageal compression, we were concerned about the safety and difficulty of TEE probe placement. It was reassuring that an esophagogastroduodenoscopy the day before surgery was uncomplicated and showed normal mucosa. However, TEE probe placement must be accomplished without the direct visualization of the esophagus present during esophagogastroduodenoscopy probe placement. American College of Cardiology–American Heart Association practice guidelines rate persistent unexplained hemodynamic compromise unresponsive to treatment as a class 1 indication for intraoperative echocardiography.[21] In light of these considerations, we judged that the benefits of real-time imaging in managing this case outweighed the risks associated with placement in a compressed esophagus. With the information provided, we were able to advise the surgeon to initiate CPB urgently, and we subsequently confirmed drainage of the pericardial and pleural effusions during bypass.

The computed tomography scan available to us before surgery was only 1 week old, yet it contained no indication of the significant left-sided pleural or pericardial effusions that we diagnosed intraoperatively with TEE. More recent imaging or preoperative transthoracic echocardiography may have indicated the presence of these ominous signs. However, additional studies were deemed unnecessary because our patient presented without signs of hemodynamic instability and had experienced no change in her symptoms during the period since the computed tomography scan was performed.

Other case reports have described a rapid preoperative progression of disease in similar cases,[12,22] which could explain the surprising new findings of the TEE in this case. A recent retrospective study indicated that pericardial effusion is an independent risk factor associated with hemodynamic collapse after induction in patients with mediastinal masses.[1] Therefore, it seems prudent to recommend imaging or echocardiography in the period immediately before surgery to

rule out acute progression of disease and/or the presence of unexpected significant pericardial and pleural effusions. In addition, preoperative imaging suggesting encroachment of the heart or thoracic vascular structures should prompt the anesthesiologist to consider preoperative and intraoperative echocardiography, even in patients without clinical signs of hemodynamic compromise.

Inadequacy of Stand-by CPB as a Rescue Measure

Recommendations in the anesthetic literature regarding the use of CPB in patients with mediastinal masses are inconsistent and nonspecific. Publications routinely recommend having CPB on "stand-by" as a rescue measure, with the machine primed and perfusionists on hand.[13,14,23] However, multiple authors have questioned the utility of this "rescue measure," pointing out that the 5–20 min typically required to cannulate and initiate CPB is far too long to prevent significant morbidity or mortality in the event of cardiac arrest.[20,24,25] Furthermore, patient positioning deemed necessary to maintain respiratory or hemodynamic function in these patients may limit the surgeon's access to the femoral vessels and increase the technical challenge of cannulation at the groin for CPB.

The 20-min delay to initiate bypass in our case certainly supports the idea that stand-by CPB cannot be counted on as a rescue measure. Of note, we took the additional precaution of having the surgeon place guide wires in the femoral vessels to facilitate transfer to CPB. Unfortunately, one of these wires was lost during the attempt to use them for cannulation. Even without this complication, having guide wires seems unlikely to have hastened the initiation of CPB sufficiently to have prevented irreversible neurologic complications had the patient experienced a complete cardiorespiratory collapse or we had difficulty performing cardiopulmonary resuscitation while opening the chest to initiate CPB.

Several authors suggest initiation of bypass in patients with severe positional symptoms,[15,20,24,26] but the literature lacks clear guidelines for preinduction vessel cannulation and CPB for patients such as ours with milder symptoms or posterior mediastinal masses. The literature contained no reports of patients with posterior masses who required urgent initiation of CPB and only one case report of successful use of planned CPB for resection of a posterior mediastinal mass.[27] We therefore thought it reasonable to proceed with guide wires in place and CPB on stand-by. In retrospect, the risks and discomfort associated with preinduction cannulation of the femoral vessels seem to be outweighed by the risk of serious intraoperative complications or mortality.

There are multiple examples in the literature of safe initiation of bypass before induction,[26–29] and cannulation of femoral vessels before induction in patients classified as "unsafe" or "uncertain" based on definitions included in the system proposed by Erdös and Tzanova is, in our opinion, the safest way to manage large mediastinal mass cases. In this system, patients with neither symptoms nor signs of en-croachment of cardiovascular or pulmonary structures on preoperative imaging are considered "safe" for general anesthesia, whereas patients with severe positional symptoms are considered "unsafe." Patients such as ours, with mild positional symptoms, or patients who can not be examined adequately are deemed of "uncertain" risk.[15]

Team Approach and Formal Plan for Managing Mediastinal Masses

Formal discussions with the surgeon and ultimately the division chief of thoracic surgery regarding management and the risk associated with general anesthesia (e.g., the need for CPB before induction vs. placement of guide wires with primed CPB machine) did not take place until immediately before the surgery. Erdös and Tzanova recommend the development of standardized protocols and an interdisciplinary team approach for the perioperative care of patients with mediastinal masses.[15] We agree with this recommendation and are implementing it at our institution after this case. Had a formal preoperative discussion with a prearranged team (anesthesiology, surgery, radiology, pathology, and otolaryngology) taken place and generated agreement on the risks of the case and the proper management strategy, many aspects of this "near miss" case could have been avoided.

In conclusion, we present the case of a patient with a large posterior mediastinal mass who experienced a sudden hemodynamic near collapse upon inhalational induction of general anesthesia while in the sitting position and required the urgent initiation of CPB. Our case contradicts the traditional consensus that posterior masses lack the risk of anesthetic complications that are well known to be associated with anterior masses. The critical role of TEE in intraoperative real-time diagnosis and guidance of treatment of hemodynamic collapse not responsive to pharmacologic interventions is well illustrated in this case. The intraoperative diagnosis of significant pericardial and pleural effusions is a cautionary example of rapid progression of disease in the days preceding surgery. In addition, our experience demonstrates the inadequacy of stand-by CPB and supports the idea that institutions should implement a formal, standardized, team approach to the perioperative management of mediastinal masses.

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