18-Year-Old With an Anterior Mediastinal Mass and Pericardial Tamponade for Mediastinoscopy

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Stem Case and Key Questions Content
A previously healthy 18-year old male (86kg, 188cm) presents to the ER with severe dyspnea and is diagnosed with a large anterior mediastinal mass. Chest CT demonstrates an anterior mediastinal mass extending from the great vessels to the xiphoid. The mass encases the great vessels; there is mild compression of the SVC, main pulmonary artery, and right and left pulmonary veins. The mass compresses both his right and left mainstem bronchus, but the trachea appears patent. On CT, there is a collection of pericardial fluid. On physical exam, the patient is sitting upright, and appears to be in mild respiratory distress (RR 30-40 SpO2 96-98% FiO2 60%). The thoracic surgeon wants to perform an anterior mediastinoscopy for biopsy in the OR today (Sunday).

Questions:
1. What other preoperative information do you want?
2. What specific concerns do you have given the patient's history/presentation?
3. Are you concerned about additional co-morbidities that can exist with an anterior mediastinal mass?

On history, he describes having a progressive cough for the past month with worsening dyspnea, now orthopnea. He had been misdiagnosed with a URI several times at an Urgent Care and received antibiotics. His CBC, chemistries and coagulation studies are normal and EKG shows sinus tachycardia (HR 105-120). A right radial arterial line is placed and there is no variation in blood pressure if the patient is supine versus sitting. On physical exam he has mild JVD; however, no upper extremity or facial swelling. Airway exam is unremarkable.
Questions:

1. What additional preoperative tests are indicated? Pulmonary function tests? Echocardiogram?

2. What is pulsus paradoxus? Can you test for it?

3. Are there any clinical symptoms that are predictive of potential intraoperative complications?

4. Are there any alternatives to performing a mediastinoscopy for biopsy?

TTE was performed and demonstrated early findings of tamponade with right atrial collapse and right ventricular diastolic collapse; left ventricle is hyperdynamic. Interventional cardiology states that due to the location of the mass, percutaneous pericardial drain placement is not possible. The oncology team feels that given the patient’s rapid presentation, an open biopsy is best, as it will allow for better histologic tissue diagnosis and immediate treatment.

Questions:

1. What are your intraoperative concerns? Regarding securing his airway and ventilation? Regarding hemodynamics?

2. What preoperative monitors are required? What IV access? Is central IV access indicated? Is location important? CVP monitoring?

3. How will you induce general anesthesia in this patient? Inhalational or IV induction?

4. How will you secure the airway? What will you do if the patient were to develop airway collapse?

5. Is neuromuscular blockade contraindicated?

6. What if hemodynamic collapse occurs?

7. Is there a role for extracorporeal life support (ECLS)? How will this be initiated? Who will initiate it?

Cardiac surgeon and perfusionist (with ECLS circuit) are available in OR. Airway is secured thru an awake fiberoptic intubation using an 8.0 endotracheal tube with the patient in seated position. Fiberoptic intubation demonstrates severely compressed trachea at the level of carina and mild compression of left and right mainstem bronchus. The patient can be ventilated and the decision is made to induce anesthesia with ketamine and maintenance with sevoflurane.
Post-induction vitals: HR 120’s, BP 90-100s/50-60s SpO2 100% (FiO2 1.0). Cardiac surgeon and perfusionist leave since patient is stable. Thoracic surgeon requests that the patient’s bed be flattened. The patient is not able to be ventilated and BP drops to 65/45mmHg.

Questions
1. What is happening?
2. Does patient position matter?
3. How does tamponade affect the patient?
4. Does endotracheal tube selection matter for this patient?

Patient is placed in sitting position and is now able to be ventilated and SpO2 is >90%. Peak airway pressures are 30-40mmHg. BP still 60s/40s. Epinephrine infusion started at 0.02mcg/kg/min and patient receives 1L LR. BP mildly improves 70-80s/40s. Epinephrine infusion increased incrementally to 0.08mcg/kg/min and fluid resuscitation continues. Patient starts to cough and move despite the Et-Sevoflurane 2.2%. The surgeon requests that the patient be paralyzed.

Questions
1. Would use a neuromuscular blocking agent since the airway is secured? Are there alternatives to stop the patient from coughing?
2. Are the peak airway pressures significant?
3. Given the nature of the surgery, is paralysis necessary?
4. What will you do if BP is still marginal?

Patient is now prepped and surgeon starts procedure. Sternal retractor aids in ventilation and peak airway pressure decreases to 20-25mmHg. At the end of the procedure, the patient is still on epinephrine infusion, and has received 6L of crystalloid. Pressure support ventilation (Pressure support 10cmH2O, CPAP 5cmH2O) VT 200-300mL; RR 20-30; SpO2 100%; EtCO2 51.
Questions

1. Will you extubate the patient?

2. Where will the patient go post-op?

3. Is an ABG necessary? What if the ABG shows a respiratory acidosis?

Model Discussion Content

Common causes of an anterior mediastinal mass include: lymphoma (Hodgkin's or non-Hodgkin's), thymoma, germ cell tumor, granuloma, bronchogenic cyst and cystic hygroma. Patients with anterior mediastinal masses are at increased risk of perioperative complications ranging from hemodynamic collapse to airway compression. Therefore, a thorough preoperative evaluation for patients presenting with an anterior mediastinal mass is paramount. Increased perioperative risk for dynamic airway compression can be signaled by dyspnea/orthopnea and coughing; this can be particularly concerning if the symptoms are present when the patient is supine. Orthostatic hypotension, syncopal symptoms or the presence of a pericardial effusion can indicate increased risk of cardiovascular complications as the mass may be compressing the great vessels or cardiac chambers. Therefore, it is prudent to determine prior to surgery, the position in which the patient experiences the fewest symptoms so that repositioning could be used during surgery should intraoperative cardiopulmonary compromise occur.

The preoperative evaluation should include a thorough history and physical exam. If patient can lay supine, a CT scan should be obtained to definitively evaluate the tracheobronchial tree (TBT) for the site, severity and extent of airway compromise. CT scan can also demonstrate if the great vessels (superior vena cava (SVC), pulmonary arteries and veins) and cardiac structures are involved (right and left atria). CT scan is the most important diagnostic modality, as it defines the size and degree of compression of adjacent structures. Anesthetic considerations will vary according to each patient’s anatomy and proposed surgical procedure. Obstruction of the TBT commonly occurs at or below the level of the carina. Pulmonary obstruction can be evident on pulmonary function tests, as flow-volume loops may demonstrate a pattern of variable intrathoracic obstruction with forced expiration increasing intrapleural pressure, thereby narrowing the airway lumen. During inspiration, intrapleural pressure is negative so airways distend, creating a normal inspiratory flow-volume loop. Given the variability, flow-volume loops are not reliable and have shown a poor correlation with airway obstruction.
Anterior mediastinal masses (AMM), given their location, may also cause SVC compression leading to SVC syndrome, with resultant venous distention and edema of the head/neck and upper extremities. When SVC syndrome is present, airway management may be difficult, especially if there is venous engorgement of the airway and mucosal edema. Strategies to minimize edema include supportive care and medical management. It may be beneficial to consider keeping the patient’s head raised to decrease hydrostatic pressure in the head and neck. Steroids may be effective in reversing symptomatic SVC syndrome caused by steroid-responsive malignancies; however, the responsiveness in other malignancies has not been well-studied and remains controversial. Diuretics have been used to treat SVC syndrome, although the benefit is unclear. In a retrospective study of 107 patients with SVC syndrome, the rate of clinical improvement was similar among patients receiving steroids, diuretics, or both. Lastly, the role of emergent radiation therapy (RT) is no longer considered necessary as symptomatic obstruction generally develops and the duration of symptoms has not been shown to influence treatment outcomes; additionally, RT before biopsy may obscure the histologic diagnosis. Also, if SVC syndrome is present, it may be prudent to consider lower extremity IV access as upper extremity access may be compromised. Since the pulmonary arteries and atria are more posterior structures, compression is less common with AMM. However, careful attention should be paid to these structures as compression can have deleterious consequences on ventricular filling and output. Patients with cardiovascular symptoms should have a transthoracic echocardiogram performed to assess for compression of the cardiac chambers or great vessels. Therefore, maintenance of preload and cardiac output may necessitate the use of positive inotropes and intravascular volume expansion.

Since airway and vascular collapse are well-recognized perioperative complications of patients with AMM, generally accepted tenets include avoidance of general anesthesia, if possible, in patients who are symptomatic, have orthopnea and tracheal compression with <50% cross-sectional area, in the presence of superior vena cava (SVC) syndrome, and in patients who have concomitant pericardial effusions. Since general anesthesia should be viewed as a “last resort,” the anesthesiologist needs to take a proactive approach as intubation and induction of anesthesia may represent multiple physiologic challenges depending on the location of the mass and its impact on surrounding structures. First, it must be determined if the procedure is diagnostic or therapeutic. For diagnosis, CT-guided needle biopsy carries a diagnostic accuracy over 90%. Awake anterior mediastinoscopy with local anesthesia or cytometric/immunocytochemical studies of pleural fluid is another viable option for diagnosis. If general anesthesia is necessary, it is prudent to have the surgeon present for induction. All mediastinal masses are not the same, given the nature of the mass and...
symptomatology of the patient. It may be important to have a cardiac surgeon available for extracorporeal life support (ECLS); however, there is no such thing as ECLS or cardiopulmonary bypass “standby” as compression can result in sudden and profound hemodynamic and ventilatory compromise in which anoxic brain injury can occur6. Indications for ECLS can include patient with severe positional symptoms in which there is obstruction of the tracheobronchial tree and/or compression of the pulmonary arteries and/or other cardiac structures; however, the literature lacks clear guidelines for the timing of vessel cannulation2. If ECLS is indicated, wires should be placed in the femoral artery and vein (or even cannulation) prior to induction of anesthesia, and a cardiopulmonary bypass or ECLS circuit should be primed and a perfusionist should be on-hand. It may be prudent to consider securing the airway through an inhalational induction or awake fiberoptic intubation. Awake intubation of the trachea allows for inspection of airway as well as placement of the endotracheal tube if it must be advanced past an area of compression. Once the airway is secured, spontaneous ventilation is thought to aid in airway patency due to increased chest wall tone and distending forces present in active inspiration. Muscle paralysis is traditionally avoided as it can lead to changes in extrinsic support of the airway and lead to obstruction of the TBT. If muscle relaxants are required, assisted-ventilation should be first gradually taken over manually to assure that positive-pressure ventilation is possible. If positive-pressure ventilation is possible, short-acting muscle relaxants should be used.

If intraoperative life-threatening airway compression occurs, it usually responds to (1) repositioning of the patient (2) rigid bronchoscopy and ventilation distal to the obstruction. The rigid bronchoscope has the potential to act like a stent and hold the airway open by relieving the extrinsic compression and airway collapse. Therefore, it may be prudent to have an experienced rigid bronchoscopist and scope available prior to the induction of anesthesia. Additionally, good intravenous access should be available prior to induction of anesthesia, as volume loading is indicated when there is compression of the pulmonary arteries and other cardiac structures. The patient should have cross-matched blood available, as there is the potential for injury to the great vessels during a mediastinoscopy.

Since the patient described has symptoms of pericardial tamponade, thereby complicating his presentation of anterior mediastinal mass, the anesthesiologist must have a sound understanding of tamponade physiology. Pericardial effusions can be classified as idiopathic, iatrogenic/traumatic, or related to an infectious, malignant, or autoimmune process. The disease states with an increased progression from pericardial effusion to tamponade include malignancies, as well as, bacterial and fungal infections7.
Anatomically, the pericardium serves to limit distention of the cardiac chambers and facilitates ventriculoatrial coupling. Since the pericardium is relatively noncompliant tissue, volume in the pericardial sac leads to increased pericardial pressure, which, in turn, leads to increased cardiac filling pressures (central venous pressure (CVP), pulmonary artery pressure (PAP), and pulmonary artery occlusion pressure (PAOP)), causing a decrease in diastolic volumes (preload) and subsequent decrease in cardiac output. These pressure-related changes are more dramatic for the thin-walled right atrium and right ventricle and may cause chamber collapse. In severe cases, venous return only occurs during systole; intrapericardial pressures are maximal and equal to right and left atrial pressures and left ventricular end-diastolic pressure (LVEDP) (15-20mmHg). Bulging of the interventricular septum into the left ventricle may be evident as preload decreases. Decreased LVEDP can impair systolic function and coronary perfusion. In cases of chronic pericardial effusion, the effusion has progressed slowly so it is generally better tolerated and does not show evidence of chamber collapse due to stretching of the pericardium over time.

The preoperative assessment of patients with cardiac tamponade should include a thorough history and physical exam. Clinical findings of tamponade include tachycardia, hypotension, dyspnea, chest pain, JVD, diaphoresis, pulsus paradoxus, muffled heart tones. Beck’s triad of distended neck veins, muffled heart sounds and hypotension is present in only 30% of patients with tamponade. The electrocardiogram may be low-voltage with electrical alternans and/or diffuse upsloping ST-segment elevation. In chronic pericardial effusions, the sympathetic response may produce systemic hypertension. If a pulmonary artery catheter is present, it may demonstrate equilibration of CVP, PAP and PAOP. Cardiac imaging should be performed whenever a pericardial effusion is suspected as chest x-ray (classic “flask-shaped” cardiac silhouette) and physical exam cannot lead to a definitive diagnosis. Echocardiography is the diagnostic test of choice, having an accuracy of nearly 100% with effusions graded as 1) small (<9mm) 2) moderate (10-19mm), and 3) large (>20mm). Characteristic signs on echocardiogram, though not highly specific, are collapse of the right atrium (RA) and right ventricle (RV). On the other hand, highly specific findings for tamponade on echo include RV free wall diastolic collapse, left atrial collapse and right-to-left bulging of interventricular septum. RA collapse and invagination is 100% sensitive for tamponade.

For patients who are hemodynamically unstable, pericardial drainage under local anesthesia, with judicious sedation with analgesic agents (ketamine, midazolam or fentanyl) should be considered. Pericardial drainage can be performed surgically through the creation of a subxiphoid window or by
percutaneous pericardiocentesis. Percutaneous pericardiocentesis had been the most useful procedure in many cases of large pericardial effusions, cardiac tamponade or pericardial effusions of unknown etiology. Regardless of the type of anesthetic employed, large-bore peripheral IV access and central access should be available for volume replacement. The anesthesiologist may want to consider volume loading to maintain preload during the time before the effusion is drained. In order to maintain filling pressures high enough to overcome diastolic filling restrictions, the CVP may need to be 20-30mmHg. Since tamponade can progress to cardiogenic shock, it is important to correct a metabolic acidosis as well as be prepared to treat arrhythmias, if present.

If general anesthesia is used, it is important to remember that positive-pressure ventilation can increase pulmonary vascular resistance and decrease RV outflow, further exacerbating the leftward septal shift and impair LV filling thereby worsening systemic hypotension. Therefore, some clinicians suggest maintaining spontaneous ventilation. However, if positive pressure ventilation is necessary, it is wise to avoid large tidal volumes and high peak airway pressures as this can minimize the impact of positive pressure ventilation on the patient’s hemodynamics. When inducing and maintaining general anesthesia, it is important to avoid vasodilation as it can worsen diastolic dysfunction as well as avoid bradycardia, as tachycardia is a compensatory mechanism to increase cardiac output in cardiogenic shock. Once the effusion is drained, a rebound in hemodynamics may occur, especially in the setting of acute tamponade; therefore, it is important to be prepared to treat such hemodynamic changes. In summary, the anesthetic management of a patient with an anterior mediastinal mass undergoing general anesthesia in the setting of pericardial tamponade physiology is challenging. The anesthesiologist needs to have a thorough understanding of both disease processes and the potential hemodynamic interdependence, as well as the effect of general anesthesia on each disease process.

References