

Anterior Mediastinal Masses

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A 16-year-old female presents for a diagnostic biopsy of a cervical lymph node and central venous port placement. She had been previously healthy until several weeks prior to admission, when she was noted to have cervical and supraclavicular lymphadenopathy, facial plethora, and occasional stabbing chest pains. On examination, HR = 110 beats/min, RR = 32 breaths/min. Orthopnea is present except when her head is elevated to $> 30^\circ$. Laboratory testing is significant for a WBC of 63,000 per microliter, of which 57% were blast cells. An electrocardiogram demonstrates electrical alternans. The AP chest radiograph shows an enlarged mediastinum and right pleural effusion.

What Are the Different Compartments of the Mediastinum?

The mediastinum can be divided into superior and inferior compartments, and the inferior compartment consists of the anterior, middle, and posterior sections. There is no actual anatomic separation between these compartments; however, on chest radiographs (Figure 19.1), the compartments can be delineated as:

The *superior mediastinum* is an area that is bound by the thoracic inlet superiorly, the thoracic cage inferiorly, the mediastinal pleura laterally, the manubrium anteriorly and the bodies of the upper four thoracic vertebrae posteriorly;

The *inferior mediastinum* is subdivided into anterior, middle, and posterior sections:

The *anterior mediastinum* lies between the sternum and pericardium.

The *middle mediastinum* contains the pericardium, heart, ascending aorta, lower half of superior vena cava, trachea, main bronchi, pulmonary artery, pulmonary veins, and phrenic nerve.

The *posterior mediastinum* is located between the pericardium and the vertebral column.

What Is the Differential Diagnosis of an Anterior Mediastinal Mass?

While pathologies can occur in any mediastinal region and cross over to adjacent areas, it is pathology in the anterior mediastinum that is most associated with perioperative risk. Leukemias (especially T-cell) and lymphomas (Hodgkin's and non-Hodgkin's) have a predilection for the anterior mediastinum. Although thymomas and germ cell tumors with anterior mediastinal involvement may also occur in children, most anterior mediastinal masses will occur in adolescents and will likely be a type of lymphoma.

What Are the Symptoms Associated with Anterior Mediastinal Masses?

Signs and symptoms associated with anterior mediastinal masses can be divided into those related to the airway, cardiac, and vascular systems, as well as constitutional symptoms. Respiratory and cardiovascular symptoms may be dependent on the patient's position, such that lying supine may increase pressure of the mass on the trachea and cardiac structures.

Airway/Respiratory Symptoms

- Inspiratory stridor
- Dyspnea
- Nonproductive cough
- Hoarseness (due to recurrent laryngeal nerve involvement)
- Orthopnea (symptoms worsen with supine position due to tracheal compression)
- Tracheomalacia (weakened tracheal walls) caused by prolonged tumor compression
- Decreased breath sounds
- Expiratory wheeze

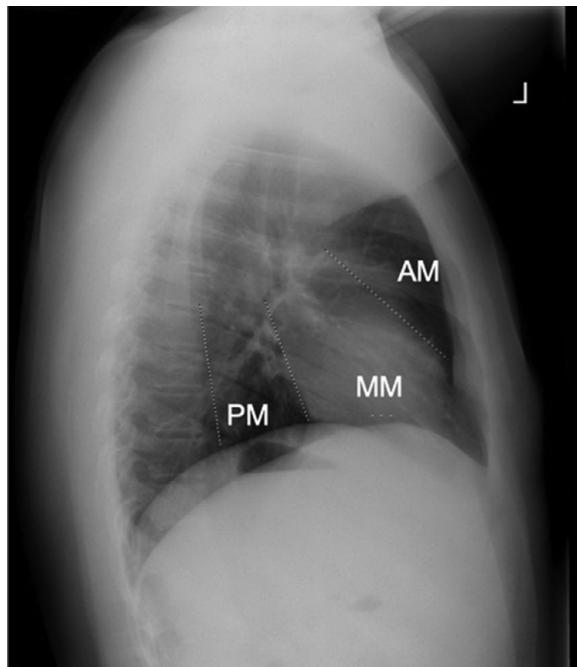


Figure 19.1 Lateral chest radiograph highlighting the anterior (AM), middle (MM), and posterior mediastinal (PM) compartments.

Cardiovascular Signs and Symptoms

- Syncope
- Tachycardia
- Plethoric facies (vascular compression): SVC syndrome
- Cyanosis
- Pleural effusion due to impaired lymphatic drainage
- Paradoxical decrease in blood pressure occurs when going from upright to supine. This is due to obstructed right ventricular filling or ejection
- Pericardial effusion
- Cardiac tamponade

Constitutional (“B” Symptoms)

- Fever, chills
- Night sweats
- Weight loss

The most significant risk factors predisposing patients to anesthetic complications include:

- Orthopnea
- Upper body edema
- Great vessel compression
- Tracheal or main stem bronchus compression

What Are the Anesthetic Risks of an Anterior Mediastinal Mass?

An anterior mediastinal mass may grow so large that it causes tracheal and/or bronchial compression. Compression of the superior vena cava or right atrium may lead to obstruction of blood flow into (superior vena cava syndrome) or out of the heart. When this obstruction is severe, the negative intrathoracic pressure generated by spontaneous ventilation precariously maintains the patency of the lower airway and great vessels and is often made worse when the patient is in the supine position (i.e., orthopnea). Administration of sedatives or anesthetic agents may result in potentially life threatening airway obstruction and great vessel compression. This obstruction cannot always be overcome by administration of positive-pressure ventilation.

In one notable case from 1981, a 9-yr-old boy with a known anterior mediastinal mass developed cardiac arrest and died during inhalational induction with halothane in the sitting position despite initially breathing spontaneously. Although the child was easily intubated and ventilated, he developed asystole that was unresponsive to all resuscitative efforts. The history revealed that four days prior to the procedure, the child became cyanotic and lost consciousness while straining during a bowel movement. An autopsy revealed that a large lymphoma had enveloped the heart and pulmonary artery, but in 1981, pre-operative echocardiograms were not yet standard of care for these diagnoses. If this large mass surrounding the heart had been revealed prior to the procedure, perhaps the team might have chosen a less sedating technique. This is only one of many similarly reported cases of children with anterior mediastinal masses who have succumbed during administration of general anesthesia.

What Preoperative Diagnostic Studies Should Be Performed in a Patient with an Anterior Mediastinal Mass?

Preoperative evaluation should focus on identification of risk factors associated with anesthetic complications, specifically airway collapse and cardiovascular compromise. A CT scan of the chest will delineate airway and cardiopulmonary involvement (Figure 19.2), and an ECG and echocardiogram will reveal the extent of

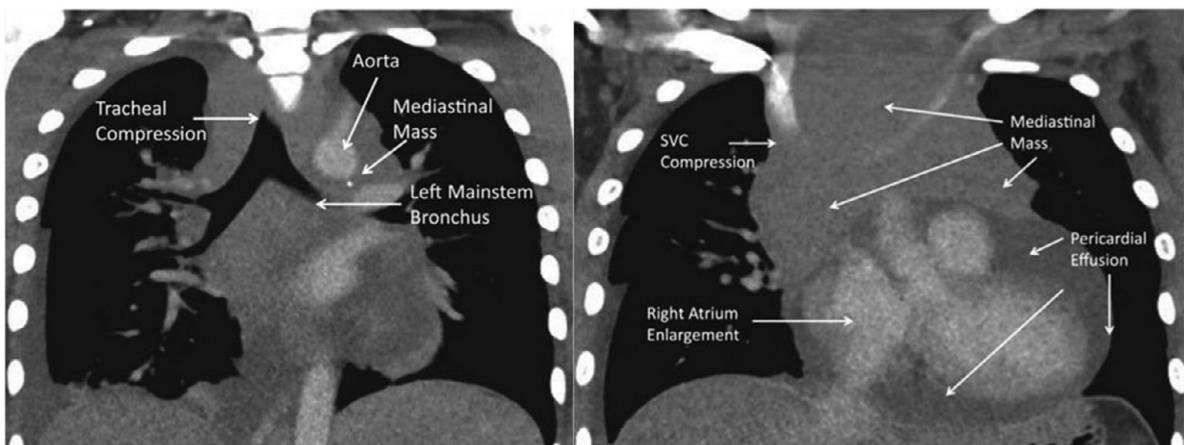


Figure 19.2 CT scan demonstrating significant tracheal compression, pericardial effusion, SVC compression, and aortic encasement. This can be compared with Figure 19.3, an AP chest X-ray from the same patient. Reproduced from: Adler AC, et al., *Anesthesiology* 2015;123(4):928 with permission. Copyright © 2015, Wolters Kluwer Health

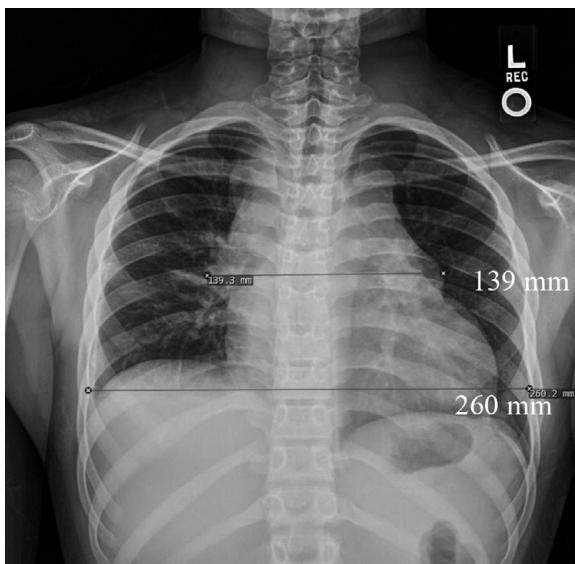


Figure 19.3 AP chest radiograph demonstrating the calculation of the mediastinal mass ratio. In this patient it is 139 mm/260 mm or 53%.

cardiovascular compression from the mass. Pulmonary function tests have not proven predictive of poor outcomes and will delay intervention, and thus, are not indicated.

The mediastinal mass ratio (MMR) and tracheal cross-sectional area (T_{CSA}) have been cited as predictors of intra-operative complications:

Mediastinal Mass Ratio (MMR)

The MMR can be calculated from an AP chest radiograph as the largest diameter of the mass divided by the largest chest diameter as measured at the level of the diaphragm (Figure 19.3). In one investigation, the risk of airway obstruction under anesthesia was 2%, 10.5%, and 33% for MMRs of <31%, 31–45%, and >45%, respectively.

Tracheal Cross-Sectional Area (T_{CSA})

The T_{CSA} is the difference between the smallest AP tracheal diameter and the largest AP tracheal diameter. A $T_{CSA} < 50\%$ raises concern that airway collapse may occur during or after administration of general anesthesia, especially in children, owing to the highly compressible and cartilaginous pediatric tracheal structure.

What ECG Findings Are Suggestive of Cardiac Involvement?

- Sinus tachycardia
- Right or left axis deviation
- Electrical alternans: seen with pericardial effusion (Figure 19.4), a beat-to-beat variation in QRS axis and amplitude due to the swinging motion of the heart in the pericardial cavity



Figure 19.4 ECG strip demonstrating electrical alternans in a patient with a large pericardial effusion.

Which Echocardiographic Signs Are Concerning in Patients with Mediastinal Masses?

- Depression of right heart function may signify elevated pressures from pulmonary-tracheal compression
- Depression of left heart function may occur from direct compression or lack of preload
- Pericardial effusion
- Right atrial collapse
- Left atrial compression
- Pulmonary venous obstruction

Overall, there is no perfect algorithm for predicting the effects of general anesthesia on pulmonary or cardiovascular compromise. In general, it appears that positional dyspnea, stridor or orthopnea, presence of superior vena cava syndrome, tracheal airway compression of >50% by CT, and compression of cardiac vasculature or large pericardial effusions identified by echocardiogram all may be important indicators of further compromise during administration of general anesthesia.

In Patients with SVC Syndrome, Where Should IV Access Be Obtained?

Superior vena cava compression leads to engorgement of the veins of the head and neck and may cause thrombosis and reduce cardiac preload. IV access should be obtained in a lower extremity to ensure adequate and rapid circulation of medications. IV access should be obtained prior to induction of anesthesia in these cases.

What Are the Risks of Administering a Neuromuscular Blocking Agent to a Patient with a Mediastinal Mass?

Administration of a neuromuscular blocker will result in the loss of negative intrathoracic pressure during

ventilation and may lead to life-threatening airway obstruction and great vessel compression. Unless strongly indicated by the clinical situation, paralysis should be avoided in these patients.

What Is the Preferred Anesthetic Technique in Patients with Anterior Mediastinal Masses?

The ideal anesthetic is one that maintains adequate airway tone and cardiac preload during spontaneous ventilation. Nearly all sedative or anesthetic agents can be slowly titrated to maintain spontaneous respiration. For biopsies and pericardial/pleural effusion drainage, generous use of local anesthetics will decrease the use of IV or inhaled agents. Some anesthesiologists prefer to use agents that can be reversed (i.e., opioids and benzodiazepines) and others prefer use of medications that have a history of preserving spontaneous ventilation (e.g., ketamine, dexmedetomidine) when slowly titrated. Since the mass will cause further compression in the supine position, the patient should be positioned with the head of the bed elevated during all phases of the procedure, when possible.

How Does the Ventilation Technique Affect Intrathoracic Pressure?

Patency of the airways and major vessels is best maintained during spontaneous ventilation, which preserves negative intrathoracic pressure (Table 19.1).

Describe the Possibilities for Intubation in a Patient with Airway Compromise

Patients with external airway compression pose a risk for airway collapse with induction of anesthesia. Smooth muscle supporting and maintaining airway patency relaxes with anesthesia and may result in life-threatening airway compression.

Table 19.1 Effect of ventilation on airway patency for patients with anterior mediastinal masses

	Intrathoracic pressure	Tracheal/bronchi patency
Spontaneous ventilation (inspiration)	Decreased	Improves
Positive-pressure ventilation	Increased	Potential for complete collapse

Patients should be positioned with the back of the table upright. Awake intubation can be performed although this is generally not well tolerated in the pediatric population. Small doses of Midazolam can be helpful for amnestic purposes. Ketamine and dexmedetomidine via bolus or continuous infusion have been used for sedation while maintaining airway patency. Adequate topicalization with local anesthetic should be employed to the upper airway as well as the vocal cords.

A flexible fiberoptic scope is often used for intubation of the trachea in these situations to allow for internal assessment of airway narrowing and positioning of the endotracheal tube distal to the point of narrowing.

What Precautionary Plans Should Be Considered Prior to Sedation of These Patients?

In patients where the anesthesia provider has significant concern for airway collapse during intubation the following should be considered. A stretcher should remain in the room allowing for the patient to be flipped prone should sedation result in tracheal occlusion.

A plan of action should the patient decompensate must be discussed with the providers involved prior to sedation to avoid confusion if facing cardiopulmonary collapse.

What Is the Role of Rigid Bronchoscopy in These Patients?

Should airway collapse occur during administration of sedative or anesthetic agents, insertion of a rigid bronchoscope past the level of obstruction may be used to establish airway patency. In cases where a

strong likelihood for airway obstruction exists, the bronchoscope should be opened in the OR, white-balanced, and connected to the monitor. A skilled operator, such as an ENT surgeon, should be immediately available. A separate stretcher or gurney should be kept in the OR to place the patient in the lateral or prone position in the event of ventilatory or cardiopulmonary compromise, as this may help alleviate airway compression.

Is There a Role for Extracorporeal Membrane Oxygenation (ECMO) in a Patient with an Anterior Mediastinal Mass?

If cardiopulmonary arrest results from compression of the trachea or heart, the ability to perform high quality CPR and maintain adequate cerebral perfusion is minimal. If the mass is so severe as to cause life-threatening obstruction, while the patient is conscious and prior to the administration of sedating agents, groin cannulation should occur as the time from cannulation to cardiopulmonary bypass (CPB) (5–10 minutes) generally exceeds the time in which significant morbidity and mortality will occur should cardiac arrest occur. ECMO is a theoretical rescue tool that, in reality, is unlikely to save a patient following arrest without prior preparation.

Is a Lymph Node Biopsy for Tissue Diagnosis Always Indicated in a Patient with an Anterior Mediastinal Mass?

In some cases, the risk of administration of sedating agents outweighs the need for an immediate tissue diagnosis. In patients with critical respiratory or vascular compromise, an attempt should be made to obtain tissue for diagnosis in a minimally invasive manner without heavy sedation or general anesthesia. Examples of diagnostic interventions include peripheral blood flow cytometry for leukemia; or lymph node, mediastinal mass, or bone marrow biopsy performed under local anesthetic. Chemotherapy and/or radiation can be used to decrease the size of the mediastinal mass prior to diagnostic procedures. Elective placement of central vascular lines should be postponed until the patient's symptoms have largely resolved and the anesthetic risk is minimal.

What Is Tumor Lysis Syndrome?

Tumor lysis syndrome (TLS) describes a constellation of metabolic abnormalities resulting from spontaneous or treatment-induced tumor cell death. Rapid cell death results in severe hyperphosphatemia and hyperuricemia, leading to acute kidney failure. Hyperkalemia may also occur leading to heart failure or cardiac dysrhythmias. Other symptoms may include gastrointestinal upset, lethargy, hematuria, seizures, muscle cramps, tetany, or syncope. Tumors with rapid growth rates such as T-cell leukemia and Burkitt's lymphoma are at highest risk of TLS. High WBC counts ($>100,000/\mu\text{L}$) and evidence of significant disease burden (hepatosplenomegaly or lymphadenopathy) also increase the likelihood of developing TLS. The risk for tumor lysis is greatest during the first 3–7 days after the initiation of chemotherapy. Prevention of TLS includes aggressive IV hydration with non-potassium-containing fluids and sometimes diuretic therapy with furosemide. Uric acid reduction with allopurinol can be initiated

immediately. However, if the patient already has a very elevated uric acid, requires ICU level care, or is at very high risk of TLS, rasburicase is recommended for its more efficient onset. Hyperphosphatemia should be treated with oral agents such as phosphate binders. Fluid restriction of patients with renal insufficiency will worsen the crystal induced nephropathy and should be avoided. Temporary dialysis is sometimes required to manage fluid overload, metabolic abnormalities and/or renal insufficiency. Replacing potassium or calcium should be done carefully.

It is important for anesthesia personnel to recognize that administration of steroids, especially dexamethasone, may cause or exacerbate TLS. Numerous case reports exist demonstrating TLS after a single dose of an IV steroid. Dexamethasone is routinely given during the chemotherapy induction phase and is preferably given to these patients under the care of the oncology team. Steroids should be used with caution in patients with vague symptoms and lymphadenopathy and without a defined diagnosis.

Suggested Reading

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