Mesothelioma

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Risk

- Incidence in USA: Approximately 2000–3000 new cases annually and decreasing. Increasing incidence in developing countries due to poor regulation of asbestos in mining and industrial use
- Attributable mortality: 14 deaths per million in USA
- Male to female ratio: 3–6:1
- 0.16% of all malignancies

Perioperative Risks

- Usually discovered in geriatric male undergoing lung biopsy
- Pleural effusion
- General debilitation from malignancy

Worry About

- Previous needle biopsy of lung and thoracentesis make pneumothorax a concern.

Overview

- Diffuse malignant mesothelioma arises from the mesothelial surface of the pleura, peritoneum, and pericardium and the tunica vaginalis of the testis.
- 80–90% percent originate from the pleura.
- Peak incidence 20–40 y after asbestos exposure.
- Usual onset of symptoms at age 55–70 y.
- Median survival after onset of symptoms is approximately 18 mo.

Etiology

- Diffuse mesothelioma related to asbestos exposure in 12–93% of cases
- Also associated with radiation therapy, erionite exposure, chronic inflammation and fibrosis, and other agents

Usual Treatment

- Treatment has been controversial and largely ineffective.
- Therapy has consisted of combinations of radiation to hemithorax, chemotherapy, and sometimes surgery (parietal pleurectomy and decortication or extrapleural pneumonectomy).

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Tracheal displacement Superior vena cava syndrome			Lateral and AP CXR
CV				ECG, ECHO
RESP	Pneumothorax	Cough, chest pain, increased SOB		ABG, PFTs (for lung resections) CXR (post biopsy; in expiration)
	Restrictive lung disease	Dyspnea with exercise	Percussion and auscultation of chest	
GI	Weight loss, debilitation, peritoneal tumors	Past body weights		CT scan of abdomen (not for periop care) Albumin (for degree of malnutrition) CBC (for malnutrition)
ENDO	Not associated with paraneoplastic syndromes			

Key References: Rusch VW: Diagnosis and treatment of pleural mesothelioma, *Semin Surg Oncol* 6(5):279–284, 1990; Ng J, Hartigan PM: Anesthetic management of patients undergoing extrapleural pneumonectomy for mesothelioma, *Curr Opin Anaesthesiol* 21(1):21–27, 2008.

Perioperative Implications

Preoperative Preparation

- Usually come to surgery for lung biopsy via thoracoscopy or open-lung biopsy; some pts are scheduled for pleuropneumonectomy.
- Assess pulmonary status; size of effusion, no pneumothorax.
- Pt often had one or more recent needle biopsies of lung or thoracenteses.
- Review radiographic studies for size and location of tumor.

Monitoring

- Routine monitors
- Resp system via stethoscope, SpO_2, and $PETCO_2$
- Intra-arterial catheter for complex surgical procedures

Airway

- Look for tracheal and mediastinal displacement on radiographic studies.

Induction

- Propensity for hypoxia, particularly from restrictive lung disease.

Maintenance

- High FIO_2 may be necessary.
- One-lung ventilation.
- Lateral positioning.

Extubation

- Ensure pt meets extubation criteria.

Adjuvants

- Pain control after thoracoscopy or thoracotomy
- No special considerations for muscle relaxants, reversal agents, local anesthetics, or special drug interactions

Postoperative Period

- Monitor ventilation and oxygenation.
- Pain relief; consider epidural or spinal analgesia after thoracotomies.
- May have air leak postop.

Anticipated Problems/Concerns

- Anesthesia with one-lung ventilation for a geriatric pt with incurable malignancy.
- Recent lung biopsy and thoracentesis prior to surgery and potential for complications from those procedures, including pneumothorax and dehydration.
- With extrapleural pneumonectomy, a possibility of massive blood loss, dysrhythmias, and hemodynamic instability during pericardial window and patch.
- Effective pain relief and monitoring of resp function postop.
- Consider ICU stay for those undergoing complex procedures.

Methemoglobinemia

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Risk

- Incidence within USA: Rare
- Gender prevalence: None
- Socioeconomic or ethnic prevalence: None

Perioperative Risks

- Inadequate O_2 carriage and delivery to tissues.
- Hemolysis may be induced by methylene blue, especially in pts with G6PD deficiency.

Worry About

- Percent of MetHb. Symptoms vary and depend on the level of MetHb present: Cyanosis appears when MetHb reaches 10–20%. Tachycardia and tachypnea can appear when MetHb reaches 20–50%. CV collapse, coma, and seizures can occur when MetHb reaches 50–70%. Death may occur at MetHb levels >70%

Overview

- MetHb is produced when Hb is oxidized and Fe^{2+} is converted to Fe^{3+} so that Hb cannot bind O_2, and the O_2-Hb dissociation curve is shifted to the left.
- Hereditary forms due to cytochrome b5 reductase deficiency or abnormal hemoglobin M.
- Acquired methemoglobinemia is largely due to oxidizing medications, including local anesthetics (benzocaine, prilocaine), antibiotics (dapsone), and nitrites. Toxic dosages can vary between individuals. Other medications and drugs (e.g., cocaine) have also been known to cause methemoglobinemia.

Etiology

- Endogenous mechanisms (NADH-MetHb reductase and NADPH-MetHb reductase) normally maintain MetHb levels to <1%. Oxidizing agents convert Hb to MetHb and can overwhelm protective mechanisms, resulting in toxic methemoglobinemia.

- Diagnosis:
  - An ABG with co-oximetry (spectrophotometry), which uses multiple wavelengths of light to determine the amount of normal versus abnormal Hb (e.g., MetHb, COHb), is necessary.
  - Keys to diagnosis are a discrepancy between pulse oximeter saturation and measured Pao_2 (Spo_2 <90% while Pao_2 >70 mm Hg) and hypoxia that does not improve with increasing FIO_2.
  - "Chocolate brown" blood may be seen.

Usual Treatment

- Supportive (ventilation, CV support); discontinuation of the offending agent
- Pts without G6PD deficiency: Methylene blue (a transient false decrease in SpO_2 may be seen after administration)
- Pts with G6PD deficiency: Ascorbic acid (methylene blue causes hemolysis)

Assessment Points

System	Effect	Assessment by Hx	PE	Test
RESP	Tachypnea, dyspnea, hypoxia	Exposure to oxidizing drugs	RR	Co-oximetry
CV	Tachycardia, arrhythmias, death if MetHb >70%	Exposure to oxidizing drugs	HR	Co-oximetry

Key References: Cortazzo JA, Lichtman AD: Methemoglobinemia: a review and recommendations for management, *J Cardiothorac Vasc Anesth* 28(4):1043–1047, 2014; Guay J: Methemoglobinemia related to local anesthetics: a summary of 242 episodes, *Anesth Analg* 108(3):837–845, 2009.

Perioperative Implications

Preoperative Preparation

- Because medications are the most frequent cause of acquired methemoglobinemia, a complete medication Hx should be sought, including OTC medications.

Monitoring

- Traditional pulse oximeters that use two wavelengths of light will not detect MetHb. Spo_2 values are inaccurate and trend toward 85% because MetHb absorbs equally at 660 and 940 nm.
- Newer "pulse co-oximeters" can detect MetHb levels and have accurate SpO_2 values.

Airway

- Routine

Induction

- Routine

Maintenance

- Routine

Extubation

- Severe methemoglobinemia may preclude extubation until levels fall to normal.

Adjuvants

- Methylene blue or ascorbic acid to treat; avoid further administration of oxidizing agents.

Postoperative Period

- Rebound methemoglobinemia can occur for up to 24 h, so pts should have MetHb levels closely monitored and additional treatment given if necessary.

Anticipated Problems/Concerns

- Oxygen-carrying capacity is decreased proportional to the concentration of MetHb present. Pts with preexisting conditions (e.g., CAD, PVD, anemia) may have tissue hypoxia even with lower levels of MetHb, necessitating earlier treatment.

Mitochondrial Disorders

Sau Yee Chow

Risk

- Incidence 4–7:100,000; might be under-reported

Perioperative Risks

- CNS: Facial and bulbar weakness, mental retardation, seizures, learning disabilities, deafness, visual impairment, and stroke-like episodes
- CVS: Cardiomyopathy and cardiac conduction defects
- Respiratory: Weakness of muscles of respiration and respiratory failure
- Other: GI disturbances, diabetes mellitus, exocrine pancreatic insufficiency, lactic acidosis, liver failure, and renal tubular defects

Overview

- Characterized by pathologic mitochondrial dysfunction in oxidative phosphorylation.
- Variable clinical presentation due to numerous possible mutations in genes coding for the electron transport chain proteins or the ancillary machinery involved in oxidative phosphorylation.
- Cardinal features of progressive muscles weakness and exercise intolerance.
- Prognosis is variable, ranging from functional impairment to death.

Etiology

- Defects of:
  - Mitochondrial DNA (maternal inheritance)
  - Nuclear DNA (autosomal dominant or recessive mendelian pattern)

Usual Treatment

- Alleviate symptoms (e.g., anticonvulsant therapy for seizures).
- Dietary modifications: Avoid fasting, addition of midchain triglycerides.
- Avoidance of toxins (e.g., alcohol, cigarette smoke).
- Vitamins and supplements to improve efficacy of ATP generation and antioxidant to slow progression of disease: Coenzyme Q10, levocarnitine, riboflavin.
- Avoidance of physiologic stress: Cold, heat, starvation, lack of sleep.
- Current treatment and medications for mitochondrial disorder.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Aspiration risk	Abnormal speech, recurrent aspirations, aspiration pneumonias	Bulbar palsy speech pattern	Videofluoroscopic swallowing study
CV	Cardiomyopathy, cardiac conduction abnormalities	Effort tolerance, palpitations, syncope, thromboembolic stroke	Signs of heart failure, abnormal heart rate	ECG Consider transthoracic ECHO, stress testing
RESP	Poor lung function, possible aspiration pneumonia	Effort tolerance, ability to cough, symptoms to suggest pneumonia	Looking for consolidation	Baseline ABG CXR Lung function test (spirometry)
CNS	Acute neuromuscular decompensation	Triggers and manifestation, current neurologic status, preexisting deficits, prior strokes	Neuro exam	Review any previous MRI brain scans available
METAB	Acute metabolic decompensation	Triggers and manifestation, severity and frequency		Consider baseline ABG and serum lactate levels

Key References: Shipton EA, Prosser DO: Mitochondrial myopathies and anaesthesia, *Eur J Anaesthesiol* 21(3):173–178, 2004; Chow SY, Woon KL: General anesthesia for adults with mitochondrial myopathy, *A A Case Rep* 4(5):52–57, 2015.