Autoimmune Diseases, Cold

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Risk

- Rare
- Autoimmune hemolytic anemias occur in 1 of 80,000 persons; of these, 17.3% are due to cold antibodies.

Perioperative Risks

- Acute hemolysis due to cold
- Hemoglobinemia
- Hemoglobinuria
- Rarely, vascular occlusion

Worry About

- Cooling to 28–31° C will cause hemolysis.
- These temperatures can be reached in extremities during cardiopulmonary bypass.

Overview

- In two circumstances antibodies will react in the cold to produce hemolysis:
  - IgG antibodies associated with mononucleosis, mycoplasmal pneumonia.
  - IgM antibodies found in the idiopathic form of the disease and in lymphoproliferative disease.
- Hemolysis usually occurs at temp below 31°C.

Etiology

- Idiopathic
- Lymphoid (B-cell) malignancy
- Infections: mycoplasmal pneumonia, mononucleosis, cytomegalovirus, varicella, EBV

Usual Treatment

- Keep warm; administer folic acid.
- For severe cases, chlorambucil or cyclophosphamide.
- Plasmapheresis.
  - Rituximab.
  - Prednisone.

Assessment Points

System	Effect	PE	Test
HEME	Mild to moderate anemia		Hgb, blood bank antiglobulin tests
GU	Hemoglobinuria		
CV	Dyspnea on exertion if anemia is severe		
DERM	Agglutination of RBCs in cold	Acrocyanosis	

Key References: Young S, Haldane G: Major colorectal surgery in a patient with cold agglutinin disease, *Anaesthesia* 61(6):593–596, 2006; Bratkovic K, Fahy C: Anesthesia for off-pump coronary artery surgery in a patient with cold agglutinin disease, *J Cardiothorac Vasc Anesth* 22(3):449–452, 2008.

Perioperative Implications

Preoperative Preparation

- Determine risks of operating vs. not operating.
- Plasmapheresis—may be used, but no more than 2 d before surgery.

Monitoring

- Temp
- Urine output

Maintenance

- Keep pt warm, including extremities.
  - Consider forced-air warming.
  - Warm all fluids.
- Normothermic cardiopulmonary bypass.
- No preferred agent or technique.
- Consider hemodilutional autologous transfusion or other techniques to avoid homologous transfusion and formation of new antibody.

Postoperative Period

- Warm fluids and extremities.
- Monitor for manifestations of cold agglutinin disease.

Anticipated Problems/Concerns

- Hemolysis if temperature falls.
- Renal dysfunction due to hemoglobinuria.
  - Molting or cyanosis of the skin can occur.

Autonomic Dysreflexia

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Risk

- AD occurs with greatest frequency in pts with spinal cord injury at T6 or above.
- Occurs with highest frequency following urologic or lumbar and thoracic spine procedures.
- Tetraplegic pts develop AD if cystoscopy and lithotripsy are performed without anesthesia.
- The higher the injury level, the greater clinical manifestations of CV dysfunction.
- Risk of AD greater with complete (91%) versus incomplete (27%) cord transections.
- AD more often a delayed finding in chronic SCI; minor clinical evidence seen in first d/wk.

Perioperative Risks

- AD most commonly triggered by irritation and/or manipulation of urinary bladder or colon, as well as in labor.
- Severe increased BP and increased or decreased HR is associated with stimulation below level of transection.
- Objectively, increased SBP >20-30 mm Hg is considered a dysreflexic episode. However, be aware that the usual resting ABP in these pts is 15-20 mm Hg less than in non-SCI subjects.
- Awake pts may complain of HA; anxiety; sweating, piloerection; and flushing above injury level; and dry, pale skin below. In anesthetized pts, SBP rising to up to 300 mm Hg heralds onset of severe, life-threatening AD.

Worry About

- Untreated, uncontrolled hypertensive episodes, which can lead to intracranial hemorrhage, retinal detachment, seizures, and death.

Overview

- Physiologically, AD is caused by a massive sympathetic discharge triggered by a noxious or nonnoxious stimulus originating below the level of the SCI.
- Specifically, destruction of the vasomotor pathways results in a loss of inhibitory and excitatory supraspinal input to the sympathetic preganglionic neurons, thus causing labile BP.
- Also, changes in spinal sympathetic neurons and primary afferents underlie abnormal CV Δs.
- Symptoms are usually short-lived because of treatment or self-limiting nature of the episode.

Etiology

- Most common cause is traumatic interruption of the spinal cord.
- Can also occur due to infectious or oncologic processes causing destructive spinal lesions.

Usual Treatment

- Stop initiating stimulus as first-line therapy when possible.
- Can decrease or prevent AD by use of neuraxial blockade (spinal >>epidural).
- When signs of AD are evident, administer ganglionic blockers (trimethaphan), direct vasodilators (nitroprusside) or α-antagonists (phentolamine), GA, or spinal anesthesia.
- Level 1 evidence that intrasphincteric anal block with lidocaine limits the AD response in pts undergoing anorectal procedures; level 1 evidence that topical lidocaine does not.
- Level 1 evidence that prazosin is superior to placebo in prophylactic management of AD.
- Level 2 evidence that nifedipine can prevent BPΔs during cystoscopy in SCI pts with AD.
- Level 4 evidence that epidural anesthesia may be effective in pts with AD during labor and delivery.
- Centrally acting hypotensive agents (e.g., clonidine) are NOT effective in treating AD.
- Treat tachyarrhythmias with β-blockers in combination with antihypertensives.
- Nicardipine may be preferable in a pt with an upper spinal cord injury undergoing operation in the paralyzed area.
- Magnesium sulfate has significant beneficial effects on AD in labor in a pt with a high spinal cord injury.
- Complete bladder deafferentation does not abolish AD during bladder urodynamic studies.