

Adjuvants

- H1/H2 blockers and steroids may attenuate the biphasic response.
- In case unresponsive to repeated doses of epinephrine, consider norepinephrine, vasopressin, and ECMO.
- Sugammadex has been used in the treatment of anaphylaxis to rocuronium.

- Epinephrine is drug of choice in true anaphylaxis. Delayed administration increases the incidence of the biphasic response and death.

Postoperative Period

- Tryptase levels should be drawn within 30 min to 2 h of a suspected reaction, then compared to baseline levels at 2 wk.

- Skin testing may be done several weeks after initial event to assess etiologic agent.
- Advise patients exactly which drugs they received.
- Obtain immunology consult.

Anticipated Problems/Concerns

- Early aggressive treatment may be critical.

Anemia, Aplastic

Joanne Shay

Risk

- Incidence in USA: 2000 new cases/y.
 - Per million up to age 9.
- Southeast Asia and South Africa have 10-20 times higher incidence.
- Within USA, related to agricultural areas or petrochemical industry and chemical exposures.

Perioperative Risks

- Infection
- Hemorrhage
- LV dysfunction due to high-output state and fluid overload

Worry About

- Sepsis
- Coexisting congenital anomalies, especially renal and cardiac
- Concomitant GI and intracranial hemorrhage
- Difficulty cross-matching blood products after previous multiple transfusions

Overview

- Self-perpetuating disorder resulting in pancytopenia due to a congenital or acquired loss of hemopoietic pluripotent stem cells.

- Fanconi anemia is congenital familial marrow hypoplasia associated with intellectual disability and kidney, spleen, and skeletal hypoplasia.
- Estren-Dameshek anemia is inherited marrow hypoplasia without physical abnormalities.
- Pathophysiology: Reduction or dysfunction of pluripotent stem cells or their microenvironment from toxic or immunologic causes.
- Prognosis for long-term survival has increased to 40% to 75% in those treated with antilymphocyte serum and 60% to 80% in those treated with BMT.
- Two forms of drug-induced aplastic anemia are possible:
 - Hypersensitivity: Not related to dose or duration.
 - "Reversible" reaction: Often resolves with discontinuation; severity proportional to dosage.

Etiology

- Of cases, 50% to 75% are idiopathic.
- Fanconi anemia demonstrates autosomal recessive inheritance with heterozygote frequency of 1 in 300,000-600,000 in USA.
- Drug-induced: Chloramphenicol, NSAIDs, anti-epileptics, and gold and sulfa group-containing compounds.

- Environmental toxins include aromatic hydrocarbons (benzene, naphthalene, toluene, and glue), pesticides (DDT and lindane), and radiation.
- Infectious causes include hepatitis C, CMV, EBV, HIV, TB, and toxoplasmosis.
- Sequelae of other processes such as pancreatitis, pregnancy, lupus erythematosus, paroxysmal nocturnal hemoglobinemia, thymoma, and thymic CA.

Usual Treatment

- Pts <55 y are managed with HLA-matched BMT or hematopoietic stem cell transplant.
- Pts >55 y or those unable to find HLA-matched donor receive immunosuppression and immunomodulation Rx, incl ATG, cyclosporine, steroids, androgens, and G-CSF.
- Hematopoietic growth factors such as G-CSF and GM-CSF may improve the short-term hematologic recovery at the risk of long-term clonal evolution to myelodysplastic syndrome and AML.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Epistaxis Oral/mucosal friability	Headache	Stomatitis	CBC, differential, plt PT, PTT, CT scan
RESP	Pulm embolism Pneumonia Interstitial pneumonitis Pulm edema	Dyspnea Hypoxemia	Tachypnea Lung field consolidation Wheezing Crackles	CXR, V/Q scan CT scan ABGs, bronchoscopy, BAL CXR, ABG
CV	LV failure ASD/VSD	Dyspnea Lethargy	Tachycardia, S ₃ Displaced posterior MI	ECG ECHO
GI	GI bleeding GI GVHD Hepatic veno-occlusive disease	N/V, diarrhea Melena	Acute abdomen Hypoactive bowel sounds Jaundice	Endoscopy, bleeding scan Selective angiography Albumin, transferrin LFT, liver biopsy
CNS	Microcephaly, meningitis, intracranial hemorrhage	Irritability, lethargy Headache, seizures	Meningismus Papilledema	Lumbar puncture after coagulopathy treated, head CT, MRI
HEME	Pancytopenia Leukemia Paroxysmal nocturnal hemoglobinuria	Bleeding gums, infections Easy bruisability Fatigue, dark urine	Petechiae Retinal hemorrhage Pallor	CBC, differential Reticulocyte count BM biopsy Ham test
METAB	Electrolyte abnormalities Glucose intolerance Hypoproteinemia Hypothyroidism	Long-term hyperalimentionation GI GVHD Hypotension, cold intolerance		Electrolytes Ca ²⁺ , Mg ²⁺ , phosphate, albumin, transferrin TSH, T3, T4

Key References: Miano M, Dufour C: The diagnosis and treatment of aplastic anemia: a review, *Int J Hematol* 101(6):527-535, 2015; Samarasinghe S, Webb DKH: How I manage aplastic anaemia in children, *Br J Haematol* 157:26-40, 2012.

Perioperative Implications**Preoperative Preparation**

- Reverse isolation precautions.
- Adequacy of blood products.

- Evaluate for severe neutropenia; coexisting congenital HD may warrant prophylactic antimicrobial therapy.
- Avoid IM and rectal sedation.
- Concomitant steroid therapy and necessity of stress doses.

Monitoring

- Arterial line if indicated.
- Consider CVP or PA cath as indicated.
- Urine output for new-onset hemoglobinuria as first sign of transfusion reaction.

Airway

- Avoid nasal manipulation.
- Use extreme caution with friable oral and pharyngeal mucosal surfaces.

Preinduction/Induction

- May exhibit hypotension and excessive fluid requirements to maintain adequate CO.
- Central neuraxial blockade contraindicated in ongoing thrombocytopenia requiring transfusion.
- Peripheral neural blockade may be approached cautiously if coagulation status is judged adequate.

Maintenance

- PEEP assures adequate tissue oxygenation at lower FIO₂ as hyperoxia depresses normal erythropoietin synthesis and marrow function.

- Nitrous oxide depresses BM function even after brief exposure; best to use O₂-air mixture.
- Normothermia promotes coagulation.
- Chronically anemic pts may tolerate lower Hct; adequacy of tissue O₂ must be addressed if CV decompensation ensues.
- Avoid induced hypotension in anemic pts.

Extubation

- Period with greatest O₂ demands

Postoperative Period

- Continued monitoring of coagulation status
- Transfusion requirements >normal
- Increased susceptibility to infection
- Pain management requires balance between pulm toilet versus sedation

Anticipated Problems/Concerns

- Age of RBC in pts with aplastic anemia is older than usual, with lower 2,3-DPG levels inside cells resulting in increased O₂ binding by Hgb (shift to the right) and decreased delivery of oxygen to tissues for same SaO₂.

Anemia, Chronic Disease/Inflammation

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Risk

- Incidence in USA: 5%; incidence in surgical population: 5% to 75%.
- Historically thought to be due to chronic infectious, inflammatory, or malignant conditions. Now known to occur with severe trauma, DM, aging, and acute immune activation.
- More than 130 million Americans living with chronic diseases.

Perioperative Risks

- Risks related to underlying diseases
- Transfusion related risks (e.g., TRALI, TACO, hemolytic reactions, immunosuppression)
- Risks related to compensatory mechanisms for increasing O₂ delivery (e.g., angina, heart failure, dysrhythmias)

Worry About

- Underlying diseases and their periop complications.
- Impaired tissue O₂ delivery and compensatory mechanisms aimed at correcting it.
- Delayed wound healing and infection.

Overview

- WHO definition of anemia: children 6 mo to 6 y: Hgb <11 g/dL; 6 to 14 y: Hgb <12 g/dL; nonpregnant females: Hgb <12 g/dL; pregnant females: Hgb <11 g/dL; males: Hgb <13 g/dL.
- Usually mild with Hgb 8-11 g/dL.
- Usually normochromic, normocytic with low reticulocyte count.
- Low serum Fe, TIBC, and transferrin levels.
- ACD/I due to disturbances of Fe homeostasis – diversion of Fe from the circulation into storage sites

within the reticuloendothelial system and reduced GI absorption of Fe.

Etiology

- Relative Fe deficiency
- Reduction in RBC production and mild decrease in RBC survival time
- Certain treatments for chronic conditions

Usual Treatment

- Treatment of underlying disease
- Fe, folic acid, and cobalamin supplementation
- Erythropoiesis-stimulating agents
- Allogeneic blood transfusion

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Hyperdynamic circulation Myocardial ischemia CHF	Palpitation Pounding pulse Angina Sx, dyspnea Exercise intolerance	Tachycardia Wide pulse pressure	ECG Exercise ECG
RESP		Dyspnea		
GI	Chronic blood loss Hypoperfusion	Blood in stool Angina equivalent (pain, nausea, indigestion)		Occult blood in stool See CV
HEME	Hgb below WHO definition level (see Overview)	Decreased exercise tolerance		Hgb
RENAL	Chronic renal failure	Decreased urine output Dialysis	Shunt	Cr K ⁺
CNS	Decreased cerebral O ₂ delivery	Dizziness Headache Transient cerebral ischemia		
MS	Low exercise capacity	Fatigue		

Key References: Gangat N, Wolanskyj AP: Anemia of chronic disease, *Semin Hematol* 50:232, 2013; Shander A: Anemia in the critically ill, *Crit Care Clin* 20(2):159–178, 2004.

Perioperative Implications

Preoperative Preparation

- Standard monitoring.
- Warm the room.
- CVP, Hgb, electrolytes.
- ST-segment analysis in pts with signs of CAD.
- PA cath for large fluid shifts or pts with signs of LV dysfunction or advanced renal failure.
- ABG.

Airway

- None

Preinduction/Induction

- Prehydrate liberally if CV status will tolerate.

- Avoid CO reduction.
- Avoid hypoxemia.
- Choose drugs according to underlying conditions.

Maintenance

- Avoid hypoxemia.
- Maintain CO.
- Avoid hypovolemia.
- Keep pt warm.
- Maintain Hgb above critical level for pts taking comorbidities into account.

Extubation

- Keep pt warm.
- Maintain high PaO₂.

- In pts with CAD, this is the period of greatest risk for ischemia.

Postoperative Period

- Keep pt warm, prevent shivering.
- Maintain high PaO₂.

Adjuvants

- According to underlying disorder

Anticipated Problems/Concerns

- Myocardial ischemia/infarction or CHF in pts with concomitant CAD.
- Deterioration of renal function in pts with CRI.
- Prolonged effects of drugs in pts with impaired renal and/or hepatic function.