Dilated Cardiomyopathy

Dilated cardiomyopathy is a syndrome with a broad list of etiologies, characterized by impaired systolic function of one or both ventricles

ANESTHETIC CONSIDERATIONS:

- Cardiac dysfunction
  - Isolated or biventricular systolic dysfunction
  - Associated MR / TR
  - Pulmonary HTN
  - Arrhythmias
  - End-organ dysfunction
- Underlying etiology
  - Ischemia
  - HTN
  - Alcohol / toxins
  - Viral
  - Idiopathic
- Medications (diuretics, nitrates – vasodilators, beta-blockers)
- Pacemaker / AICD
- Need for antibiotic prophylaxis if associated valvular abnormalities

ANESTHETIC GOALS:

- Maintain forward flow
- **Preload**: maintain but avoid increases in LVEDP
- **Afterload**: reduce
- **Contractility**: avoid depression
- **Rate**: normal (avoid tachycardia with adequate depth of anesthesia d/t risk of ischemia)
- **Rhythm**: NSR
- **MVO_2**: monitor for ischemia

HISTORY

- Goals:
  - Establish the etiology of DCM
  - Establish symptomatology and functional capacity of patient
  - Identify current treatment
- Congestive Heart Failure
  - Classify based on NYHA classification
  - Forward and backwards failure
  - End-organ dysfunction
    - Renal dysfunction
    - Liver dysfunction from hepatic congestion
- Look for evidence of associated illness that can be optimized:
  - CAD
  - HTN
  - Valvular disease
  - Alcohol
  - Infiltrative diseases
  - Other: SLE, ESRD

PHYSICAL

- **GENERAL**
  - Decreased CO
    - Patients compensate from decreased SV by increasing sympathetic outflow to divert flow to vital organs
    - Sinus tachycardia and peripheral vasoconstriction
    - Cool, pale and cyanotic extremities
    - Pulsus alternans
  - Volume overload
    - Pulmonary congestion
    - Peripheral edema
      - Leg swelling
      - Ascites
      - Hepato- / splenomegaly
    - Elevated JVP
- **CVS**
  - Lateral displacement of apical pulse
  - Parasternal heave
  - S3
- **RESP**
  - Pulmonary HTN
    - Increased P2, palpable pulmonic tap

INVESTIGATIONS

- **Laboratory**
  - CBC: risk of anemia in heart failure
  - E-lytes, BUN, Cr for associated end-organ damage and medications used in heart failure
  - LFTs: hepatic congestion
  - Thyroid function
  - Others as dictated by etiologies
  - BNP
- **Imaging**
  - CXR
    - Useful to differentiate CHF from primary lung disease
    - May also demonstrate cardiomegaly
  - EKG
    - Evidence of ischemic heart disease
    - LVH
    - Heart blocks frequent in DCM: 1° AVB, LBBB, LAHB, IVCD
    - Low limb lead voltages common in idiopathic DCM
    - Diagnosis of arrhythmias
  - Echocardiography
    - Typically patients with DCM have four chamber dilatation with decreased LV function
    - Sensitivity 80% and specificity 100% in heart failure
    - Also useful for:
      - Evaluation of ventricular thrombi
      - Evaluation of other etiologies of heart failure (valvular, ischemic etc.)
      - RV function and estimation of PAP
  - MIBI
    - Useful to determine if ischemic etiology of DCM

OPTIMIZATION

- Are there any potentially treatable causes of DCM?
  - Ischemic DCM may benefit from revascularization
  - HTN
  - Valvular CM
- Is the patient medically optimized on appropriate HF medications?
  - Pulmonary edema optimally managed medically preoperatively if possible (diuretics and nitrates to ↓ preload, afterload reduction) or intubation, high FiO_2, PEEP if critical
  - Pacemaker / AICDs programmed preoperatively if elective (magnet available if emergency)
  - Ensure that pump failure compensated through diuretics, vasodilators, beta-blockers
  - Arrhythmias should be promptly treated
  - If unstable may need inotropic or IABP support
- Optimize Hb
- Manage anticoagulation
- SBE prophylaxis if appropriate

ANESTHETIC OPTIONS

- Either general anesthesia or regional anesthesia OK if hemodynamic goals can be met
- Neuraxial anesthesia has favorable effect on hemodynamic goals

ANESTHETIC SETUP

- **Drugs**
  - Inotrope (e.g. ephedrine) preferred due to β stimulation
  - Avoid pure α agonists (e.g. phenylephrine) due to unfavorable effect on afterload
  - Anti-arrhythmics
- **Equipment**
  - Arterial line
  - CVP to monitor filling pressures
  - 5 lead EKG
  - Consider PAC particularly if large volume shifts expected
  - +/- TEE available
  - Have immediate availability for cardioversion or pacing
    - Pacer pads
    - Consider pacing PAC in high risk patients

MANAGEMENT OF ANESTHESIA

- **Induction**
  - Major goals of anesthesia are:
    - Avoid drug induced myocardial depression & tachycardia induced ischemia
    - Maintain normovolemia
    - Avoid increases in ventricular afterload
  - IV anesthetics may have a delayed effect due to prolonged circulation time

- Be wary of myocardial depression of induction agents
- Opioids generally safe given limited myocardial depression but they are poor amnestics

- **Maintenance**
  - Volatiles
    - All reduce myocardial contractility
      - Degree of depression: halothane > isoflurane = sevoflurane = desflurane
      - These effects are likely exaggerated in abnormal myocardium
    - Decrease preload and afterload
      - These favorable effects likely offset negative inotropy
  - Ensure patients adequately anesthetized to avoid sympathetic stimulation associated with surgery
- **Emergence**
  - Ensure adequate perioperative pain control
  - Conflicting evidence of epidural anesthesia and decreased perioperative adverse cardiovascular events

DISPOSITION & MONITORING

- Dependent on complexity of surgery and stability of cardiomyopathy → HDU may be required

COMPLICATIONS

- **Hypotension / Low cardiac output state**
  - Consider myocardial dysfunction as potential cause
  - Optimize filling pressure
  - Use ephedrine (high doses of phenylephrine could adversely affect cardiac performance)
- **Ischemia**
  - Once again, ensure that hemodynamic goals are optimized
  - Consider afterload reduction: NTG and β-blockers if appropriate
  - ASA
- **Arrhythmia**
  - Poorly tolerated
  - Consider early cardioversion
- **Tachycardia**
  - Ensure adequate depth of anesthesia and deepen if necessary
  - May treat with esmolol infusion (titratable), recognizing the possibility of further myocardial depression

PATHOPHYSIOLOGY

- Multiple diverse etiologies
  - e.g. CAD, valvular HD, EtOH, viral, drug related (esp. chemo – adriamycin, doxorubicin), muscular dystrophies, and idiopathic
  - Cause myocardial damage producing a dilated, poorly contractile ventricle
- The low cardiac output causes activation of the SNS (peripheral vasoconstriction, ↑ NE levels: results in ↑ wall tension, HR and MVO_2) and RAAS (↑ vasopressin), which leads to further adverse hemodynamic changes:
  - Increased afterload and preload, tachycardia
  - Salt and water retention, and further myocardial remodeling and damage
- The abnormal myocardium is very arrhythmogenic, with AF, non-sustained VT, conduction delays, and sudden cardiac death all common
- Intracardiac thrombosis (due to stasis) and thus systemic or pulmonary embolization may occur
- 1-year mortality is 30%, and 5-year is 60%
- Most deaths (80%) occur from arrhythmia or pump failure
- **Management**
  - Treat the underlying disease
    - HTN, valvular disease, ischemic disease etc.
  - Medical management of heart failure (complex)
    - ACEi and ARB
    - Hydralazine plus nitrates
    - Beta-blockers
    - CCB
    - Digoxin
    - Diuretics
    - Aldosterone antagonists
  - Treatment of associated conditions
    - Arrhythmias
      - A-fib common (30%)
    - Anticoagulation
      - Risk of stasis in hypocontractile ventricle
  - Cardiac transplant

REFERENCES

- Miller
- Hensley
- Kelly's Internal Medicine
- Barash