Cardiac Complications

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I. ACUTE PULMONARY EDEMA

Introduction
A change in alveolar-capillary membrane permeability leads to pulmonary interstitial edema. The initiating pathology can be either cardiac or non-cardiac in origin.

Pathogenesis of Pulmonary Edema

Alveolar-Capillary Membrane
a. Capillary Endothelial Layer: Microvascular Barrier
b. Alveolar Epithelial Layer: Alveolar Barrier

Fluid Dynamics of the Alveolar-Capillary Membrane
a. Hydrostatic pressure
b. Osmotic pressure
c. Membrane permeability

Etiology of Pulmonary Edema

Cardiogenic Pulmonary Edema
Cardiogenic pulmonary edema, which is the most common type of acute pulmonary edema, occurs when there is an increase in hydrostatic pressure within the pulmonary capillary bed as a result of heart failure. Causes include:
a. Heart Failure
b. Myocardial Infarction
c. Cardiac Ischemia
d. Acute Mitral Regurgitation
e. Cardiac Tamponade
f. Tachy Dysrhythmias
g. Hypertensive Crisis

Non-Cardiogenic Pulmonary Edema
Non-cardiogenic pulmonary edema results from one of four primary abnormalities (or a combination):
a. Impaired endothelial integrity
b. Decreased colloidal oncotic pressure
c. Elevated capillary hydrostatic pressure
d. Lymphatic obstruction

The impaired endothelial integrity (change in permeability) is typically caused by a direct or indirect injury to the lung tissue. Acute respiratory distress syndrome (ARDS) is a form of non-cardiogenic pulmonary edema.
Diagnosis

**History**
- Cardiogenic Pulmonary Edema
- Acute Cardiac Event
- Chest Pain
- Tachy-Palpitations
- New Dysrhythmia
- History of Ischemic Heart Disease
- Acute CP and/or SOB in the Absence of Any Other Pathologies
- Absence of Cardiac Hx Does Not Rule Out CPE

**Physical Exam**
- Dyspnea, Tachypnea & Apprehension
- Presence of S₃ Heart Sound
- Jugular Venous Distension
- Breath Sounds
- Increased Frothy Sputum Production
- Laterally Displaced Point of Maximal Impulse (PMI)
- New or Louder Cardiac Murmur
- Unilateral Lung Adventitious Sounds: more commonly assessed in non-cardiogenic
- Diffuse Decreased Breath Sounds: more commonly assessed in non-cardiogenic
- Peripheral Edema is non-specific

**Chest X-Ray**
There are chest x-ray changes that are unique to cardiogenic and non-cardiogenic pulmonary edema. If the pattern changes from day to day or significantly after treatment it is more indicative of cardiogenic.

**ECG Changes**
Tachycardia or acute ST-T segment changes are more commonly assessed in cardiogenic pulmonary edema

**Echocardiography**
A Transthoracic echo may be helpful to identify myocardial ischemia, wall motion abnormalities, ventricular dysfunction, valvular disease, and LV hypertrophy, all of which will suggest cardiogenic pulmonary edema.

**Laboratory Data**
Arterial Blood Gas:
- Low Oxygen Saturation
- Respiratory Alkalosis
- Refractory Hypoxemia
Treatment Options

Cardiogenic Pulmonary Edema
ACLS algorithm while treating the underlying cardiac condition:

a. Diuretics: Furosemide IV 0.5 to 1.0 mg/kg
b. Analgesics: Morphine IV 2 – 4mg
c. Preload Reduction: Nitroglycerin SL
d. Oxygen/Intubation
e. Afterload Reduction if SBP > 100mmHg
   • IV Nitroglycerin 10-20 µg/min or consider
   • IV Nitroprusside 0.1 – 5.0 µg/kg/min
f. Vasoconstriction if Hypotensive
   • Severe (SBP < 70mmHg) and S&S of shock
     o IV Norepinephrine 0.5 – 30 µg/min
   • Moderate (SBP 70– 100mmHg) with S&S of shock
     o IV Dopamine 5 – 15 µg/kg/min
   • Moderate without shock
     o IV dobutamine 2 – 20 µg/kg/min
g. Further Diagnostic Considerations
   • Pulmonary Artery Catheter
   • Intra-Aortic balloon pump
   • Cardiac Angiography

II. RUPTURED OR DISSECTING AORTIC ANEURYSMS

Definitions
These conditions can overlap at times with one leading to or increasing the risk of the other.

Aortic Aneurysm
A localized dilation of the arterial wall that can be saccular, fusiform or cylindrical. The dilation frequently renders the aorta weak in that region.

a. Complication of aneurysms
b. Management of aneurysms

Aortic Dissection
a longitudinal separation of the aortic wall between the intima and the adventitia. An acute dissection is one that is diagnosed within 14 days of the onset of symptoms. The risk of death is greatest during this acute period. A chronic dissection is one that is diagnosed after two weeks of the onset of symptoms.
Classification Systems

*Stanford Classification System*

**Type A Aortic Dissection**
The dissecting area involves the ascending aorta. It may be confined to only the ascending aorta or may also involve the descending as well. Typically occur in a younger patient population with a congenital weakening of the ascending aorta. Type A dissection account for 2/3 of all dissections.

**Type B Aortic Dissection**
The dissecting area involves only the descending aorta distal to the left Subclavian artery. Typically occurs in the older patient population with a history of hypertension and atherosclerosis.

*DeBakey Classification System*

**Type I Aortic Dissection**
The dissection involves the ascending aorta but also extends beyond the left subclavian artery.

**Type II Aortic Dissection**
The dissection involves only the ascending aorta.

**Type III Aortic Dissection**
The dissection involves only the descending aorta. IIIa limited to the thoracic aorta, IIIb involving various degrees of the thoracic and abdominal aorta.

Common Risk Factors

a. Although hypertension does not appear to be the sole contributor to the occurrence of AAD, it plays a major role in the development and/or propagation of a dissection. The etiology of AAD is believed to be a combination of something that has caused a weakening in the vessel that ‘allows’ the original tear to occur and that, in combination of HTN (70-90% of the victims have a history of HTN) triggers the filling of the false lumen and dissection of the arterial layers.

b. Marfan’s Syndrome (a chromosomal mutation with many genotypes and phenotypes)

c. Annuloaortic Ectasia
d. Aortic Dilatation and Wall Thinning
e. Bicuspid Aortic Valve (congenital malformation)
f. Spontaneous Rupture of Vasa Vasorum
g. Aortic Coarctation
h. Trauma (blunt, penetrating or iatrogenic)
i. Aging
j. Arterial Hypertension
k. Cocaine Use
Diagnosis

**Presenting Signs & Symptoms**
AAD is Known as the Great Imitator
a. Sudden Severe Pain Not Relieved with Analgesics
b. Initially Normal or High Blood Pressure
c. Hypotension
d. Acute Aortic Valvular Insufficiency: High-Pitched, Blowing Diastolic Murmur
e. Audible S₃ Heart Sound
f. Abrupt Onset of a Pulseless Extremity
g. Peripheral Vascular Insufficiency
h. End-Organ Ischemia (brain, kidney, intestines, spinal, lower extremities)
i. Pericardial Effusion
j. Cardiac Tamponade
k. Acute Myocardial Ischemia

**Tests**
a. Chest X-ray
   - Wide Mediastinum
   - Wide Aortic Silhouette
   - Pleural Effusion
   - CHF
   - Pericardial Effusion (cardiomegaly)
b. ECG: Nonspecific Changes
   - Left Ventricular Hypertrophy
   - Acute Myocardial Ischemia
d. Transthoracic or Transesophageal (TEE) Echocardiography
e. Magnetic Resonance Imaging (MRI) Scan
f. Aortagraphy

**Treatment**

**Adequate Blood Pressure Management**
a. Antihypertensive Agents: Nipride 0.5μg/kg/min titrate up to maintain SBP below 110mgHg or at a level to maintain perfusion
b. Negative Inotropic Agents: β blockers
c. Pharmacological management may be the primary treatment for a dissection involving the descending aorta

**Pain Relief**
Typically done with Morphine

**Reduction of Environmental and Emotional Stresses**
May need anti-anxiety agents.
Surgical Repair
a. All patients with ascending aortic dissection require immediate repair
b. Descending dissections repairs have high mortality and morbidity

III. HYPERTENSIVE CRISIS

Introduction

Pathophysiology of Hypertension
Definition and Current Guidelines

Definitions

Hypertensive Crisis
A diastolic blood pressure greater than 120mmHg. Global term does not denote physiologic response or need for immediate treatment.

Hypertensive Emergency
A diastolic blood pressure of greater than 120mmHg with acute or ongoing end organ (neurological, cardiac or renal) damage. Immediate blood pressure reduction is required within a few hours to prevent or limit target organ damage. The reduction does not necessarily need to be back to normal pressure just out of the dangerous range.

Hypertensive Urgency
A diastolic blood pressure of greater than 120mmHg without end organ damage. Reduction of blood pressure is important to limit the risk of potential end organ damage but not emergent. The goal is to bring down the blood pressure within 24 – 48 hours.

Malignant Hypertension
Described by Volhard and Fahr in 1914, MHT is characterized by severe accelerating hypertension with evidence of renal, neurological, vascular and retinal damage/dysfunction that can be rapidly fatal ending in heart attack, stroke or heart and renal failure. The modern criteria for MHT are severe hypertension (DBP > 120mmHg) associated with retinal hemorrhages, exudates and papilledema (group 4 Keith-Wagener-Barker retinopathy) (Laragh, 2001). Some authors define it simply as elevated BP accompanied by encephalopathy or nephropathy (Varon, 2000).

Accelerated Hypertension
A more ‘mild’ form of MHT without the presence of papilledema and a group 3 Keith-Wagener-Barker retinopathy.

Post Operative Hypertension
Defined as systolic blood pressure of greater than 190mmHg and/or diastolic blood pressure of greater than or equal to 100mmHg on two consecutive readings following surgery. Because of
the unique and transient physiological factors following surgery and anesthesia this clinical syndrome is separated from the other hypertensive crises.

**Gestational Hypertension**
There are multiple names for this syndrome. A blood pressure is considered an emergency in a pregnant woman and requires immediate pharmacologic management when the systolic pressure is greater than 169mmHg or diastolic greater than 109mmHg.

**Hypertensive Crisis – Pathophysiology & Management**

**Etiologies**
There is not one cause of HTN Crisis. A history of preexisting hypertension is the common denominator regardless of the secondary causative factor(s).

**Pathophysiology**
Although the exact physiological mechanism(s) of hypertensive crisis are unknown, there appears to be a vicious cycle of increased vasoconstriction which leads to increasing pressure.

**Assessment**
In addition to the blood pressure, the presence and relative degree of end organ damage/dysfunction is important to assess for and essential to identify before selecting the appropriate treatment option.

a. Previous Diagnosis of HTN
   - How long?
   - Prescribed Medications?
   - Adherence to Prescription Medication?
   - General Level of Control or Typical Blood Pressure?

b. All Other Medications: Prescription, Over the Counter, Dietary Supplements and/or Illicit Drugs

c. Cardiac Assessment

d. Renal Assessment

e. Neurological Assessment – Hypertensive Encephalopathy

f. Laboratory Data

g. Evaluate Secondary Causes

**Treatment for Hypertensive Emergency**
The goal is to reduce the mean arterial pressure by 25% within the first two hours (preferably within the first few minutes) in a controlled, predictable and safe fashion and then toward 160/100mmHg within two to six hours.

a. Nitroprusside Sodium (Nipride)

b. Fenoldopam Mesylate (Corlopam)

c. IV Vasodilators

d. IV Adrenergic Inhibitors

e. Diuretics
Treatment for Hypertensive Urgency
Blood pressure should be lowered within 24-48 hours and frequently oral agents are adequate in this patient population.
   a. ACE inhibitors
   b. Calcium Channel Blockers
   c. Alpha₂ Adrenergic Stimulators (Clonidine)

IV. VALVULAR HEART DISEASE

Pathophysiology

   a. Congenital Malformations
   b. Connective Tissue Disorders
   c. Degenerative Disease
   d. Rheumatic Heart Disease
   e. Infective Endocarditis
   f. Dysfunctional Ruptures

Specific Valvular Dysfunction

   a. Mitral Stenosis
   b. Mitral Insufficiency/Regurgitation
   c. Aortic Stenosis
   d. Aortic Insufficiency/Regurgitation

Management of Valve Disorders (pre-op and post-op)

   a. Oxygenation
   b. Hemodynamic Stability
   c. Dysrhythmias
   d. Activity
   e. Anticoagulation
   f. Antibiotic Prophylaxis
   g. Patient/Family Education

Surgical Management of Valve Defects

   a. Indications
   b. Valve Repairs
   c. Prosthetic Valve Replacement
      • Mechanical
      • Biological
Septal Defects

a. Locations: Atrial and Ventricular
b. Types: Congenital and Acquired

V. VASCULAR DISEASE

Peripheral Vascular Disease
Note: DVT is addressed in the Pulmonary section combination with the Pulmonary Emboli

Pathophysiology
a. Smoking
b. HTN
c. DM
d. Lipid Disorders
e. Hyperhomocysteinemia

Assessment
a. Intermittent Claudication
b. Resting Pain
c. Cool Temperature
d. Diminished Pulses
e. Leg/Skin Changes

Diagnosis
Acute occlusion

Acute Arterial Occlusion
5Ps
a. Pain
b. Pulselessness
c. Pallor
d. Paresthesian
e. Paralysis

Management/Treatment
a. Risk Factor Modification
b. Vasodilators
c. Antiplatelets
d. Exercise
e. Angioplasty
f. Vascular Bypass Surgery
g. Minimally-Invasive Interventions (stents, endografts)
Compartmental Syndrome
a. Compartments are closed spaces containing muscles, nerves, and vascular structures
b. Internal and external causes can increase pressure within a compartment
c. Increased pressure can lead to ischemia, injury and necrosis to the contents within the compartment
d. Signs and Symptoms of CS
   • Throbbing Pain (localized)
   • Firmness of area
   • Altered Sensation: Numbness, tingling, sticking feelings
   • Pulselessness
   • Decreased Voluntary Limb Movement
e. Treatment for CS
   • Eliminate the Cause
   • Elevation
   • Pain Management
   • Fasciotomy

Carotid Artery Disease

Pathophysiology

Assessment

Management/Treatment
a. Risk Factor Modification
b. Vasodilators
c. Antiplatelets
d. Neuro Monitoring
e. Carotid Endarterectomy
f. Carotid Stents

VI. CARDIAC TAMponade

Introduction
A cardiac effusion is the accumulation of fluid within the pericardial space. A cardiac tamponade occurs when an effusion causes compression on the heart and the external pressure effects cardiac function.
Etiology

Any pathology that can lead to a pericardial effusion can cause a tamponade. Pericarditis is the most common cause:

a. Any Type of Pericarditis (inflammatory, infectious, immunologic or physical)
   - Neoplastic
   - Renal Insufficiency → ESRD
   - Post Acute Myocardial Infarction
   - Collagen Vascular Disease
   - Autoimmune Diseases
   - Chemotherapy
   - Radiation Therapy
   - Nephrotic Syndrome
   - Tuberculosis
   - Hepatic Cirrhosis

b. Invasive Cardiac Procedures (cardiac surgery or biopsy)

c. Indwelling Cardiac Instrumentation

d. Anticoagulant or Thrombolytic Therapy

e. HIV/AIDS

f. Valvular Heart Disease

g. Trauma

h. Pregnancy

i. Aortic Dissection

j. Chronic Heart Failure

Pathophysiology

**Pericardium**

The pericardium is a membrane that surrounds the heart. The pericardial space is between the visceral (next to the myocardium) and parietal layers and there is typically 15-35ml of serous fluid in the space. The function of the fluid is to provide a cushion for the heart. Fifty mls or more of fluid (serous fluid, blood, pus, clots or gas) is considered an effusion. Tamponade can be classified as acute vs chronic, surgical vs medical.

Presenting Signs & Symptoms

**Early Physical Signs & Symptoms**

May occur prior to full tamponade

a. Chest Pain

b. Anxiety

c. Tachycardic

d. Tachpneic

e. Diaphoretic

f. Bibasilar Rales

g. Fever

h. Unstable Blood Pressure
Cardiac Complications

i. S&S of Shock
j. Specific to Pericarditis
   - Diffuse ST elevation
   - Chest pain worse when supine
   - Pericardial friction rub

Beck’s Triad (1935)
   a. Muffled Heart Sounds: from the accumulation of fluid.
   b. Narrowing of the Pulse Pressure and Hypotension: The pulse pressure is the difference between the SBP & the DBP. Due to the increasing pressure outside the heart there is less dilation during diastole, which causes the DBP to either stay the same or rise. There is a lower EF because of the decreased filling and contractility so the SBP drops.
   c. Jugular Vein Distention (JVD): There is ‘back-up’ of blood into the venous system from a combination of the impaired filling and impaired emptying of the heart due to external pressure. Kussmaul’s sign is a pathological increase in jugular venous pressure (JVP) seen during inspiration.

Pulsus Paradoxus
   a. The negative pressure created in the thorax during normal inspiration limits cardiac filling, decreases CO and causes a weaker pulse. There is typically a difference of as much as 10mmHg in SBP between inspiration and expiration.
   b. During tamponade the ‘normal’ pressure is increased causing more of a compression, which is increased even more during the inspiratory phase of ventilation. A drop of >10mmHg systolic blood pressure heard during inspiration is considered a Pulsus Paradoxus. Commonly (present in 90% of cases) assessed with cardiac tamponade.
   c. Also found in other pathologies that cause increased pressure on the heart like COPD. PP was described by Kussmaul in 1873.

ECG Changes
   a. Electrical Alternans: beat to beat change in QRS amplitude (seen with late or rapid tamponade)
   b. Low Amplitude of the QRS Complex
   c. Absence of Ischemic Changes Despite Chest Pain
   d. Non-specific ST changes may be Present in Pericarditis
   e. In late tamponade there may be decreased coronary flow (typically in the elderly and patients with CAD)

Diagnosis

Chest X-Ray
Enlarged cardiac silhouette with clear lungs

Hemodynamic Parameters:
   a. Drop in Cardiac Output
   b. Pulsus Paradoxus Visible on Arterial Line. Note that if the patient is on positive pressure ventilation this response will be reversed.
Echocardiography
A non-invasive, relatively available test to identify the presence of a pericardial effusion. Occasionally if the effusion is primarily posterior it may be difficult to visualize on a transthoracic echo and a transesophageal echo is necessary. Of all the diagnostic tools for tamponade this is the most sensitive and specific.

Therapeutic Management Options

Pericardiocentesis
Needle aspiration of effusion. Subxiphoidal approach. Procedure can be performed blind, or with ECG clamp, echo or fluoroscopy guidance. If recurrent effusion is a concern a drainage system should be left in place for a few days.

a. 6 inch, 16-18 gauge, over-the-needle catheter.

Surgical Drainage

a. Subxiphoid surgical incision and thoracoscopic drainage
b. Video assisted thoracoscopy and drainage
c. Pericardial window and drainage
d. Pericardectomy: complete stripping of the pericardial sac is performed on some medical patients with chronic effusion/tamponade. This is typically a last resort.

Medical Management

ACLS
Oxygen, IV, CPR- if needed, Epinephrine, diagnosis and treat cause ASAP

General Management

a. Support compensatory and physiologic response
b. Expand Intravascular volume
c. Increase or decrease SVR
d. Support contractility and stroke volume