Traumatic Cardiac Tamponade

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POS
Objectives

- Definition
- Pathophysiology
- Diagnosis
- Treatment
Cardiac Tamponade

• The decompensated phase of cardiac compression resulting from increased intrapericardial pressure
• Physiologically, tamponade is a continuum
• To tamponade the heart, the contents must:
  1. Fill the pericardial reserve volume
  2. Increase at a rate exceeding pericardial stretch
  3. Exceed the rate at which venous blood volume expands to support the small normal pressure gradient for filling the heart
Incidence

- In trauma, tamponade is most commonly associated with penetrating injuries.
- Can occur with blunt chest trauma causing the pericardium to fill with blood from the heart, great vessels, or pericardial vessels.
Acute Tamponade

- May be referred to as ‘surgical’ tamponade
- In the acute setting, tamponade can quickly overwhelm compensatory mechanisms
- As little as 150cc of blood and clots can be rapidly lethal
- Chronic: as much as 1L
Pathophysiology
Transmural Pressure

- Filling pressure of the heart is the transmural pressure (cavity pressure minus intrapericardial pressure)
- Negative transmural pressures can cause atrial and ventricular collapse
- Chamber collapse coincides with a drop of 15-25% of CO
Pressure-Volume Relations

- Pericardium has a J-shaped pressure volume curve
- Increasing fluid forces the heart to function on the steep portion of the curve
- At this point, small increases have large effects (**Tx***)
Tamponade – Early Stages

- Initially, fluid fills up the pericardial reserve volume (flat portion of curve)
- Eventually this exceeds the limit of pericardial stretch
- The increasing pressure ultimately can exceed the atrial and ventricular filling pressures (phasically negative transmural pressure)
Tamponade – Late Stages

- Right heart chambers are affected before the left
- Once the pressure from tamponade is great enough to overcome LV pressure, CO decreases dramatically
- ‘pure’ tamponade is when diastolic pressures in the LV & RV equilibrate with mean pressures in the LA & RA
Coronary Blood Flow

- With normal coronary arteries, coronary blood flow is adequate to support aerobic metabolism.
- This is due to a proportionate reduction in cardiac work: the ventricles are underloaded and operate at the lower end of the Frank-Starling Curve.
Frank-Starling Curve
Compensatory Mechanisms

• Elevated CVP: improves diastolic filling against the increased intrapericardial pressure
• Tachycardia: to maintain CO
• Vasoconstriction: peripheral vascular resistance to maintain BP
Other Organ Flow

- Renal and cerebral blood flow are partly supported by compensatory mechanisms.
- Significant hepatic and mesenteric ischemia occurs secondary to increased peripheral vascular resistance.
Diagnosis
Clinical Findings

• Beck’s Triad
  1. Hypotension
  2. Muffled Heart Sounds (with a relative accentuation of S2)
  3. Elevated Central Venous Pressure

• High index of suspicion must be used with pre-existing HTN, noisy ERs, and hypovolemia
Clinical Findings

- Kussmaul’s Sign (Inspiratory increase or lack of fall of JVP)
- Oliguria
- Tachycardia
- Tachypnea
- Pulsus Paradoxus
- Low-voltage EKG complexes
- EKG Electrical Alternans
Pulsus Paradoxus

- Systolic drop in arterial pressure of 10mmHg or more during normal breathing
- The difference between the pressures when systolic sounds are first heard and then are continuously heard gives the magnitude
- Usually signals large reductions in LV/RV volumes with equilibration of mean pericardial and all cardiac diastolic pressures
Pulsus Paradoxus

- Deep x descent and blunted y descent can be seen on RAP tracing
- Diastolic pressure equalization causes a relative RV filling at the expense of the LV
Electrical Alternans
Chest X-Ray

- Enlarged heart shadow on radiography with clear lungs
Echocardiography

- Short Axis/Cross-Section View of the Heart
- PE = pericardial effusion
Echocardiography

- 5% False -ve
- Echo-Doppler signs are less accurate
- Fluid in pericardium can be detected as part of a focused abdominal U/S exam
EKG

• Can be normal
• Often nonspecifically altered – ST-T wave abnormalities
• End-stage tamponade will show vagally mediated bradycardia and PEA (electromechanical dissociation)
Treatment
Medical Treatment

- Experimental evidence is conflicting
- Ultimately ineffective
- Aimed to support compensatory mechanisms, expand intravascular volumes, support inotropy and blood pressures
Surgical Intervention

- Echocardiographically monitored pericardiocentesis or surgical drainage are treatments of choice
- In cases with extreme urgency, percutaneous needle paracentesis is indicated, particularly when the patient has not responded to resuscitative efforts
Percutaneous Needle Paracentesis

- Subxiphoid approach safer than parasternal (decreased PTX, coronary artery laceration)
- 45° Head Tilt
- 18-23 gauge spinal needle, inserted just below and to the left of the xiphoid towards the Midclavicular point
- Continuous aspiration
Percutaneous Needle Paracentesis

- EKG guidance with an alligator clip on the needle can identify cardiac penetration (ST elevation or ectopic beats with myometrium contact; deep Q waves once in the ventricle; PR elevation with atrial contact)
- Removal of only 20cc of blood can be life saving due to the position on the pressure-volume curve
Surgical Drainage

- Subxiphoid pericardial windows and thoracoscopic drainage are effective treatments that have little morbidity and can be performed with local anesthesia.
- However, it is preferable to have the patient in the OR for these procedures.
- Blunt trauma patients with PEA are not candidates for resuscitative thoracotomy.
Final Notes

• The volume drained can not predict cardiac compression that still may exist
• Clots add a confounding issue with percutaneous pericardiocentesis
• All trauma patients with positive pericardiocentesis will require open thoracotomy or median sternotomy for further inspection of the heart