Case #1

- 47 YO intoxicated WM
- Syncopal episode, complains of crushing chest pain
- + Tobacco, + FH
Initial EKG
Right Sided EKG
During Cath

http://www.ecglibrary.com/2_1avb.html
2:1 AV block

- Every other P wave is conducted to the ventricles
- 2 to 1 AV block starts after the 5th QRS in this 3 channel recording. The first non-conducted P wave is indicated with an arrow.
- the PR interval of conducted P waves is constant
- in this EKG there is a long PR interval
- 2 to 1 AV block cannot be classified into Mobitz type I or II as we do not know if the 2nd P wave would be conducted with the same or longer PR interval.

http://www.ecglibrary.com/2_1avb.html
Also During Cath

http://www.ecglibrary.com/vtavd1.html
Ventricular Tachycardia

A wide QRS tachycardia is VT until proven otherwise. Features suggesting VT include

- evidence of AV dissociation
- independent P waves
- capture or fusion beats
- beat to beat variability of the QRS morphology
- very wide complexes (> 140 ms)
- the same morphology in tachycardia as in ventricular ectopics
- history of ischemic heart disease
- absence of any rS, RS or Rs complexes in the chest leads
- concordance (chest leads all positive or negative)

http://www.ecglibrary.com/vtavd1.html
4 hours after admission
About 2 hours post cath
24 hours after admission
48 hours after admission
6 days after admission
RV Infarction

• **Clinical Findings**
  – Normal or depressed right ventricular function
  – Shock
  – Tricuspid regurgitation
  – Ruptured ventricular septum

• **Hemodynamic Measurements**
  – Abnormally elevated right atrial pressure
  – Normal right ventricular and pulmonary artery systolic pressures
  – Increased ratio of right ventricular to left ventricular filling pressure
  – Depressed right ventricular function curve
RV Infarction

• **Cardiac Catheterization**
  – Involvement of right (usually) or left (rarely) circumflex coronary arteries
  – Right ventricular akinesis

• **Differential Diagnosis**
  – Hypotension with acute myocardial infarction
  – Pericardial tamponade
  – Constrictive pericarditis
  – Pulmonary embolus

Braunwald, 1180.
RV Infarction

- **Scintigraphy**
  - Uptake in right ventricular free wall
  - Increased right ventricular dimensions and decreased wall motion

- **Echocardiography**
  - Increased right ventricular dimension
  - Absence of pericardial effusion

- **Cardiac Enzymes**
  - Increased magnitude of enzyme values relative to degree of left ventricular dysfunction

Braunwald, 1180.
Diagnostic features of RV infarct

- Elevated right ventricular filling pressure
- Steep right atrial y descent
- Early diastolic drop and plateau (square root sign) in the right ventricular pressure tracing
- Pulsus Paradoxicus (10 mm drop of Systolic with inspiration)
- Kussmaul’s sign (Increase in JVP with inspiration)
Right Sided EKG (V_{4R})

- Proximal RCA – ST elevation 1mm and positive T wave
- Distal RCA – No ST elevation, positive T wave
- Cx – ST depression 1mm and negative T wave

Braunwald, 1180.
Wellens
Echocardiography

- Dilated RV
- Dilated RA
- Regional wall motion abnormality (Left or Right Ventricle)
- No pericardial fluid
Case 2

• 50 yo AAF with hx HTN, +tob
• Vomiting, diaphoresis, +/- Chest pain
Initial EKG Case 2
Right Sided EKG Case 2
Right Atrial Waveform

- **a wave** - RA contraction
elevated in RV failure

- **c wave** - tricuspid closure

- **v wave** - passive filling of RA during ventricular systole = T wave on ECG
elevated in tricuspid regurgitation

- **x descent** - atrial diastole

- **x' descent** - RV systole

- **y descent** - atrial emptying
Hemodynamics of RV infarct

• Elevated right sided filling pressures
• Equalization of right and left sided diastolic pressures
• Low cardiac output
Features to watch

• Y descent (tricuspid opening/passive filling) is a measure of RV compliance
• A wave and X descent - RA contraction typically augmented with stiff RV, but if RCA occlusion is proximal this may be compromised

Kern, 274
“W” and “M” patterns

• W wave
  – RCA occlusion distal to RA branches
  – Augmented a waves
  – Steep x descent

• M wave
  – RCA occlusion proximal to RA
  – Depressed a waves
  – Blunted x descent
Progression of waveforms

- RVI – augmented a wave and x descent
- RAI – blunted a wave and x descent

Kern, 275
Hemodynamics of RV infarct occlusion distal to RA

- “W” pattern formed by prominent a waves and sharp x descent
- X descent is a SYSTOLIC event, and correlates with RVSP
- Note elevated RVEDP corresponding with tall a wave

Kern, 276
Hemodynamics of RV infarct occlusion proximal to RA

- “M” pattern formed by blunted a wave and x descent
- X’ descent occurs during t wave
- X’ descent occurs during PASP peak (systolic event)

Kern, 276
Another “M” pattern

• X descent occurs during t wave
• RV tracing reveals that sharp descent is x
• Diastolic y blunted

Kern, 277
Treatment of RV infarct

• **Maintain Right Ventricular Preload**
  – Volume loading (IV normal saline)
  – Avoid use of nitrates and diuretics
  – Maintain AV synchrony
  – AV sequential pacing for symptomatic high-degree heart block unresponsive to atropine
  – Prompt cardioversion for hemodynamically significant SVT

• **Inotropic Support**
  – Dobutamine (if cardiac output fails to increase after volume loading)
Treatment of RV Infarct

• **Reduce Right Ventricular Afterload with Left Ventricular Dysfunction**
  – Intraaortic balloon pump
  – Arterial vasodilators (sodium nitroprusside, hydralazine)
  – ACE inhibitors

• **Reperfusion**
  – Thrombolytic agents
  – Primary PTCA
  – CABG (in selected patients with multivessel disease)
Pearls for RV infarction

• If hypoxia is present, consider PFO with Right → Left shunt due to increased right sided pressures

• Tricuspid Valve replacement/repair can be performed for severe TR in setting of RV infarct
Case #3

- 74 yo F with hx of CABG
- Symptoms of syncope
- Ultimately referred for cath as outpatient
- Hx of DM, CLL, HTN
Cath procedure

- Difficult stick
- R femoral access obtained, above anatomic crease
- No problems during cath
- Perclose procedure performed successfully
15 minutes later…

- Pt discharged, had syncopal event on ride home
- Pt admitted to CCU
- Hgb 10
- CT scan + for retroperitoneal bleed
- Hypotensive
- Attempted L subclavian TLC
- Successful L femoral TLC
Off to surgery

• Surgical consult on board stat
• To OR post cath day 1
• L subclavian 8.5F sheath + swan
• L femoral 6F sheath for art line
• Repair of R external iliac
  – Perclose sutures noted ‘nearby’
• Stabilized post op
Medications

• Ativan 1mg
• Demerol 50 mg
• Kcl 20 meq
• Lasix 40mg
• Phenergan 25 mg
• Hydroxyzine 25mg
• Albumin 5%
• Nicardipine IV
• Clonidine 0.3 patch
• Pepcid 20mg bid

• Hydralazine 10mg q6h
• Solumedrol 40mg
• Zaroxolyn 5mg po bid
• Lopressor 50mg bid
• Versed prn
• Morphine prn
• NaCl 75 cc/hr
• Diprivan
• Terazosin
Interesting labs...

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Uh oh

- On POD #4, on routine assessment pt found to have cold, mottled L leg. Distal pulses previously dopplerable, not able to doppler now.
- L fem 6F arterial line still in place
To the cath lab

- Thrombus proximal and distal to sheath
- SFA 100%
- Deep femoral with decreased flow
- 100% popliteal
- 100% posterior/anterior tibial, peroneal

- To the OR for thrombectomy
What happened

• Underlying CLL
• Cath with difficult stick
• Retroperitoneal bleed
• Acute renal failure
  – ATN due to shock, low Hgb, decreased perfusion
• Thrombocytopenia
• Thrombus formation
Heparin induced thrombocytopenia?
(Well, that’s what I’m going to talk about, anyway)

• Two types
  – Nonimmune
    • Benign, self-limited
    • Dose dependent
    • Rarely causes large drop in platelet count or clinical complication
    • 15% of patients treated with heparin
  – Immune mediated
    • 2-3% of all exposed patients
    • Heparin Induced Thrombocytopenia +/- Thrombosis
    • 25-30% mortality rate, 25% amputation rate

Braunwald
Walenga, 262
Risk factors for HIT

• Initial low platelet count
• Renal impairment
  – Increased risk of death
• Recent cardiovascular surgery
  – Increased risk of amputation

Kelton, et al.
Diagnosing HIT

- Thrombocytopenia
  - Decrease of 50% from baseline
  - Start thinking when there is 30% decline
- Absence of other cause of thrombocytopenia
- Confirmation by heparin associated antibody assay
- Return of normal platelet levels when heparin is discontinued
Clinical Features of HIT

- Usual onset day 5-6 (3-14)
- Nadir of platelets usually 30-60k
- Occurs with all types/methods of heparin
  - Continuous infusion > flushes > heparin coated catheters
  - Bovine heparin > porcine heparin > LMW heparin
- Can start within hours of exposure
- Surgical patients have increased venous risk
- Cardiovascular patients have increased arterial risk
- No relation to gender, age, or inherited clotting disorders
Thrombosis

- 35% of patients with HIT will develop clinically significant thrombosis
- Thrombosis can form anywhere
- Arterial > Venous or Venous > Arterial depending on the study
- DVT, PE, Mesenteric ischemia, CVA, Myocardial infarction
Pathogenesis in a nutshell

- Heparin attaches to Platelet factor 4 (PF4) on platelet surfaces
- IgG Fab antibody binds heparin/PF4 aggregate
  - Fac portion recognizes FcγIIa receptors on platelets
- This causes the platelet to release PF4
- Heparin/PF4 complexes can form on endothelial surfaces; IgG attaches to this and damages the cell

Walenga, 263
Lab tests

• Platelet aggregation assay
  – Normal plt + pt serum + heparin evaluated for aggregation
• Serotonin release assay (SRA)
  – Donor plt incubated with radiolabeled serotonin
  – 40-80% sensitive
• ELISA to heparin-PF4 complex
  – False negatives common
• Clinical HIT may have negative lab tests!

Walenga, 264-5
Management of HIT

• DC all heparin
  – Flushes, catheters
  – This is not sufficient to prevent thrombosis, however (35% develop thrombosis)

• Direct thrombin inhibitor
  – Lepirudin
  – Argatroban
    • 6 day therapy (up to 14)
    • PTT 1.5-3x baseline
    • New thrombosis/amputation/death (25.6% vs. 38.8% control)

Walenga, 265-6
Other meds (not recommended) for HIT

- ASA, dextran, warfarin are not indicated for sole treatments of HIT
- Danaparoid
  - Low molecular weight heparinoid
  - Favorable in vitro activity vs heparin
  - Anecdotal/small series support
  - Unable to monitor, no dosing regimen
- Ancrod – a defibrinating agent that is not recommended
Future meds for HIT

- Hirulog (Angiomax)
  - Currently in development for HIT

- Factor Xa inhibitors
  - Arixtra

- Antiplatelet agents
  - 2b/3a, plavix have limited, but successful, clinical experience
Other management of HIT

- Coumadin may be started if otherwise indicated when platelet counts >100k
- Plasmapheresis can be used to filter out IgG antibodies if heparin exposure is unavoidable
- Immunoglobulin can be used in patients refractory to plasmapheresis
- Thrombolysis with urokinase can be used for organ/limb threatening ischemia. Selective thrombolysis causes less trauma than thrombectomy
Risk of heparin use

• Heparin risk is primarily bleeding complications
  – Risk factors:
    • advanced age
    • serious concurrent illness
    • heavy consumption of alcohol
    • concomitant use of aspirin
    • renal failure
Other risks of heparin

• Anaphylaxis
  – 5-10 minute onset
  – Wheezing, chills, fever, tachycardia, diaphoresis
  – Limited patients, have been successfully treated with LMWH without reaction

• Osteoporosis
• Skin reactions (urticaria, necrosis)
• LFT abnormalities
• Eosinophilia
• Hyperkalemia
• Hyperaldosteronism
• Priapism
• Alopecia

Walenga, 260
References