“Cardiology Pearls for the Hospitalist”

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Disclosures

I have nothing to disclose
Outline

- Five cases you will encounter
  - Diagnostic or management challenges
  - Time & early decisions matter

- Take-home points
Case 1

- 62 y.o. man:
  - 1 year PTA: Elective bioprosthetic AVR and aortic root replacement for bicuspid AV/dilated root
  - 2 days PTA: Sees PCP for 7-10 days of fevers/chills
    - 2 sets of blood cultures drawn
  - DOA: Blood cultures positive for 4/4 bottles GPC
    - Admitted to hospital
    - Remainder of vital signs & physical exam normal
    - Otherwise well except for fevers
What Do You Do?

What antibiotics do you choose?

1) Vancomycin
2) Vancomycin and nafcillin
3) Vancomycin and piperacillin-tazobactam
4) Vancomycin and gentamicin
5) Vancomycin and rifampin
6) Vancomycin and gentamicin and rifampin
7) Vancomycin and gentamicin and cefipime
What antibiotics do you choose?

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4) Vancomycin and gentamicin
5) Vancomycin and rifampin
6) **Vancomycin and gentamicin and rifampin**
7) Vancomycin and gentamicin and cefipime
General Principles of Empiric Antibiotics

• Native valve or prosthetic valve?
• Stable or unstable?

• Native valve:
  • Stable: Hold off awaiting culture results
  • Unstable: Vancomycin

• Prosthetic valve:
  • Stable: Hold off awaiting culture results
  • Unstable: Vancomycin, gentamicin, cefepime/carbapenem

• Rationale:
  • Need for gentamicin for staph (harder to clear) and higher likelihood of GNR endocarditis
Causative Organisms

- **Prosthetic valve endocarditis**
  - **Early (first 2 months):**
    - S. aureus/Coag. Neg staph > GNR > Enterococcus/Fungi
  - **Middle (2-12 months):**
    - Coag negative staph > Strep/S. aureus > Fungi
  - **Late (>12 months):**
    - Strep > S. aureus > Coag Neg staph > Enterococcus

- **Native valve endocarditis**
  - S. aureus > S. viridans > Enterococcus > Coag neg staph
  - Less common: Other strep, HACEK, GNR, Fungal
General Principles of Antibiotics When More is Known

- Remember: Blood cultures are very sensitive (>90%) if no antibiotics have been given
  - True “culture-negative” endocarditis is rare
  - Multi-organism endocarditis is very rare
- Once early data is back, can pare down antibiotics accordingly (i.e. no GNR coverage if GPC)
- Once organism is identified, antibiotic course based on bug, susceptibilities, native vs. prosthetic valve
  - Rifampin added particularly for staphylococcal infection on prosthetic valve due to ability to kill staph on biofilm
Early Hospital Course

- TTE: Normal LV size/function. Large mobile mass (1.5 cm) on bioprosthetic valve, prolapsing into LVOT. No abscess seen, mild AR.
- Started on vancomycin/gentamicin/rifampin
  - Within hours of starting antibiotics → TIA, MRI with tiny acute infarct in left MCA territory
- Blood cultures: 4/4 coagulase negative staph
- EKG: Normal
What is the Absolute Indication for Surgery?

1) Prosthetic valve endocarditis
2) Staphylococcal endocarditis
3) Embolic TIA
4) Size of the vegetation
5) He has no absolute indication for surgery
What is the Absolute Indication for Surgery?

1) Prosthetic valve endocarditis
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Indications for Surgery

- Heart failure due to valve dysfunction
- Severe valvular regurgitation
- Fungal endocardritis
- Abscess or fistula formation
- Persistent infection or recurrent emboli despite appropriate antibiotics
- Vegetation size (?) > 1 cm
  - Higher risk of emboli but not clear that this should be indication in and of itself
  - Is an indication to screen for emboli with scanning
Now What?

- TEE: Vegetation slightly larger (1.6 cm), no abscess seen.
  - False negative
  - No other explanation for PR prolongation
- In light of worsening AV block ➔ scheduled for urgent surgery
- OR findings: Large vegetation with abscess
- Postoperative: Originally pacemaker dependent, ultimately recovered AV conduction
Endocarditis: Common Mistakes

- Waiting too long for surgery if indication is present
- Not getting daily EKGs early in management
- Not using a “cidal” drug once the organism has been identified
- Antibiotics given prior to blood cultures
- Writing off coag-negative staph bacteremia as a contaminant in a patient with a prosthetic valve
Case 2

- 54 y.o. man with no significant PMH other than EtOH abuse (6 beers/day)
  - Sought new primary care physician for new-onset DOE
  - Exam revealed BP 110/75, HR 62, possible ascites → ultrasound ordered
  - Abd U/S: Moderate ascites, portal venous flow pulsatility, hepatic vein engorgement, all c/w hepatic congestion.
  - 3 days later: Worsened dyspnea → ER
  - Exam in ER:
    - BP: 110/90, HR 145
    - Appears in moderate distress
    - Elevated JVP, tachycardic/regular, mild edema
  - Labs: BUN/Cr 25/1.6 (up from 15/1.2), trop negative
HR: 146  

SINUS TACHYCARDIA  

NONSPECIFIC T ABNORMALITIES, DIFFUSE LEADS  

PROLONGED QT INTERVAL  

QRS: 90  

QT: 336  

QTc: 524  

--- AXIS ---  

P: 0  

QRS: 4  

T: 188  


Reason: tachycardia  

Requestor: KLOFAS, EDWARD  

Order #: 406915417  

Enc ID: 131018185588  

Service Code: 56100050-S  

Device: ED10  

Speed: 25 mm/sec  

Lead: 10 mm/mV  

Chest: 10 mm/mV  

P 60- 0.5-100 Hz W 

P0905A  

P?
What is the Most Likely Rhythm?

1) Sinus tachycardia
2) Atrial fibrillation
3) Atrial flutter
4) Ventricular tachycardia
5) Junctional tachycardia
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HR: 146  SINUS TACHYCARDIA

NONSPECIFIC T ABNORMALITIES, DIFFUSE LEADS

PROLONGED QT INTERVAL

QRS: 90
QT: 336
QTc: 524

-- AXIS --
P: 0
QRS: 4
T: 168


Order #: 404915417
Enc ID: 131018185588
Reason: tachycardia
Standard 12
Requested By: KLOFAS, EDWARD

Stanford Hospital/Clinics - Stanford Hospital (1-01-59)
Edited Electronically Signed: Kenneth Sakamoto MD 13-Aug-2012 18
HR: 140

- SINUS TACHYCARDIA
- NONSPECIFIC T-ABNORMALITIES, DIFFUSE LEADS
- PROLONGED QT INTERVAL

Order #: 4949165417
Enc ID: 131014180588
Reason: tachycardia
Requested By: KLOPAS, EDWARD

Stanford Hospital/ Clinics - Stanford Hospital (1-01-59)

Device: ED10
Speed: 25 mm/sec
Link: 10 mm/mV
Chest: 10 mm/mV
What is Your Next Move?

1) Amiodarone 150 mg IV bolus
2) Amiodarone gtt 1 mg/min
3) Amiodarone 150 mg IV bolus, then 1 mg/min gtt
4) Diltiazem gtt 10 mg/hr
5) Diltiazem 20 mg IV bolus, then 10 mg/hr gtt
6) Metoprolol 5 mg iv, repeat q15-30 min prn
7) DC Cardioversion
8) Emergent TEE & DC Cardioversion
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7) DC Cardioversion
8) Emergent TEE & DC Cardioversion
What Happened…

- Diltiazem 20 mg iv bolus given
- Quick decompensation & frank shock, lactic acidosis
- TTE: LVEF <25%, normal LV size
- Diagnosis: Tachycardia-induced cardiomyopathy with cardiogenic shock
- Outcome:
  - DC-cardioversion, amiodarone, inotropes
  - Lactic acid peaked >10, nearly received percutaneous LVAD
  - Gradually recovered over ensuing days
- F/U 2 months later:
  - Remained in NSR
  - Echo: Normal LV function
About Anticoagulation...

- General rule:
  - Okay to cardiovert without TEE if duration of AF is <48 hours
- The problem: You almost never really know if they have been in AF for longer!
- Do not trust patient-reported symptoms of palpitations, etc.
  - Trial evidence clearly demonstrates that patients under-recognize when they are in AF
- General rule: Sustained anticoagulation or TEE needed, other than in emergent setting
- Remember: Even with negative TEE, they still need anticoagulation!
4 Week Study of 22 Patients with PAF

110 Patients with Pacer, Prior AF: % With AF Episodes vs. Detected by ECG at MD Follow-up

Figure 2. Cumulative incidence of detection of any atrial fibrillation recurrence by electrocardiographic recording during follow-up (FU) (solid line) versus information from the implanted device (dashed line). n = number of patients at risk.

Take-Home Points for AF with RVR

- Take this diagnosis very seriously – *think* before loading with calcium-blockers or beta-blockers
- Digoxin: Usually not enough, but can be a useful adjunct!
- Consider quick bedside TTE for LV function
  - May be best use of this diagnostic aid
- If signs of heart failure are present → think about early TEE/cardioversion
- Tachy-induced cardiomyopathy: Can happen quickly
  - Most common underlying rhythm = 2:1 atrial flutter
- Low threshold to anticoagulate
Patient 3

- 76 y.o. woman with HTN is taken to the ER from her 4th of July BBQ because of sudden SOB
- PE: Wt 75 kg (baseline 74 kg) BP 185/110, HR 105, SaO2 85% RA, diffuse bibasilar rales.
- Baseline meds: ASA 325 mg qd, HCTZ 25 mg qd, amlodipine 10 mg qd, lisinopril 20 mg qd
- CXR: Normal cardiac silhouette, diffuse pulmonary edema
- ECG: Sinus tachycardia at 105 bpm, LVH criteria with repolarization abnormality
Patient 3

- Labs: Na 137, K 4.1, Cr 1.6 (baseline 1.6), BNP 450, troponin T <0.01. ABG: 7.49/28/54 on RA

- Baseline echo: Normal LV size/function, moderate LVH, 2+ MR
What Do You Do?

What should you do immediately?

1) Intubation, furosemide
2) BIPAP, sublingual nitroglycerin, furosemide
3) BIPAP, nitroglycerin drip, furosemide
4) BIPAP, dobutamine, furosemide
What Do You Do?

What should you do immediately?

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2) **BIPAP, sublingual nitroglycerin, furosemide**
3) BIPAP, nitroglycerin drip, furosemide
4) BIPAP, dobutamine, furosemide
What is the Problem?

- Characteristic findings in a patient who develops “flash” pulmonary edema:
  - Poorly compliant ventricle (often with LVH)
    - Can be worsened by ischemia
  - Small weight gain, relatively unimpressive BNP
  - Often have significant mitral regurgitation
  - Almost always hypertensive at presentation
What is the Solution?

• In this patient, the main problem is *increased pressure*
  • ↑ afterload in noncompliant ventricle → ↑ LVEDP → ↑ wedge pressure (especially with MR) → pulmonary edema

• Acute increase in preload (e.g. high Na intake) can also cause increased filling pressures/flash pulmonary edema with noncompliant ventricle
What is the Solution?

- Patient is in a vicious cycle
  - Pulmonary edema/hypoxia $\rightarrow$ distress/raised BP $\rightarrow$ worsened pulmonary edema/hypoxia
  - Pulmonary edema/hypoxia $\rightarrow$ ischemia $\rightarrow$ worsened pulmonary edema/hypoxia

- Time is of the essence – you are at a crossroads
  - Quick, decisive action $\rightarrow$ rapid improvement
  - Delayed (or unaggressive) action $\rightarrow$ worsening of vicious cycle
How to Treat this Patient

- Vasodilator at reasonable doses
  - Nitroglycerin (can start with SL)
  - Nitroprusside
  - Nesiritide
- Diuresis
  - Important, but not as important
- Respiratory support
  - Oxygen
  - BIPAP (also helps lower preload)
  - Intubation – beware sudden hypotension!
What to Tell this Patient Long Term

• This is the patient *most* sensitive to sodium intake
  • Literally one indiscretion $\rightarrow$ flash pulmonary edema
• Focus on BP control
• Role of ‘conventional’ heart failure medications not clear
• No indication for device therapy (e.g. ICD, resynchronization)
Patient 4

- 60 y.o. man with sudden-onset “tearing” chest pain radiating to the back
- BP 180/100, equal pulses, cardiac exam normal
- CTA: Type B aortic dissection
Which Sx/Sign Is Most Common in Aortic Dissection?

1) Back pain
2) “Tearing” or “Ripping” description of pain
3) Sudden onset of pain
4) SBP > 150 mmHg at presentation
5) Pulse deficit on exam
Which Sx/Sign Is Most Common in Aortic Dissection?

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2) “Tearing” or “Ripping” description of pain
3) Sudden onset of pain
4) SBP > 150 mmHg at presentation
5) Pulse deficit on exam
Time-Course of Pain

Intensity of pain

Aortic dissection

Myocardial ischemia

Time (minutes)
Frequency of Sign/Symptom (N=464)

Risk-Factors for Aortic Dissection

- **Common**
  - Hypertension
  - Atherosclerosis

- **Less Common**
  - Collagen disorder (Marfan syndrome, Ehlers-Danlos)
  - Bicuspid aortic valve
  - Vasculitis
  - Cardiac catheterization
  - Cocaine
Thinking About Aortic Dissections

• Type A vs. Type B (Stanford Classification)
  • Type A: Involves ascending aorta
  • Type B: Involves descending aorta only

• Why does it matter?
  • Type A: Much higher mortality
    • Always needs emergent surgery if operative candidate
  • Type B: Can defer surgery in most cases
Management Principles

- BP & wall stress control
  - Beta-blockers
  - Pain control
  - Nitroprusside
- Type A → Emergent surgery
- Type B: Monitor for need for intervention (surgery or endovascular repair)
  - End-organ ischemia (e.g. kidneys, limbs)
  - Refractory pain
  - Expanding dissection/rupture
Mortality By Dissection Type & Management

Patient 5

- 45 y.o. man with idiopathic dilated cardiomyopathy → ER for nausea/vomiting, abdominal pain

- Exam:
  - Vitals: AF  BP 80/40  HR 120  RR 22  SaO2 95% RA
  - + scleral icterus/mild jaundice
  - JVP elevated to 20 cm H₂O
  - Loud S3 gallop
  - Abd: Distended, diffusely tender but worst over RUQ, equivocal Murphy’s sign
  - Ext: Clammy extremities, 2+ bilateral LE edema
Patient 5

- CXR: Cardiomegaly, mild interstitial thickening, no obvious pulmonary edema

- Baseline echo: Severe LV dilatation, LVEF 20%, 3+ MR, 2-3+ TR, RVSP = 55 mmHg
Patient 5

- Outpatient meds: Carvedilol 3.125 mg bid, lisinopril 2.5 mg bid, furosemide 80 mg bid, digoxin 0.125 mg qd, spironolactone 25 mg qd

- Labs: Na 128, K 5.6, Cr 2.0 (baseline 1.4), Bilirubin 5.4 (baseline 1.0), Alk phos 180, INR 1.5, AST 240, ALT 300, WBC 10k, BNP 2500, Lipase 60
Patient 5

- ECG: Sinus tach at 120, nonspecific ST-T changes (unchanged from baseline except HR)

- STAT RUQ U/S: + gallbladder wall thickening possibly c/w cholecystitis, + ascites, normal CBD
What Do You Do?

1) Consult surgery for cholecystectomy
2) Start on Abx/fluids for cholecystitis
3) Diurese
4) Diurese/afterload reduce
5) Diurese/pressors
6) Diurese/inotropes
What Do You Do?

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2) Start on Abx/fluids for cholecystitis
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4) Diurese/afterload reduce
5) Diurese/pressors
6) Diurese/inotropes
What is the Diagnosis?

- Low output heart failure (e.g. cardiogenic shock)
- Keys to the diagnosis: Hypotension, elevated JVP, S3
- Frequently present differently than you might think
  - GI complaints
  - Elevated LFTs (can be bili or transaminase pattern)
  - Worsened renal function
  - Much less common: Pulmonary edema/hypoxia
How to Functionally Manage This Patient

• Augment forward flow
  • Afterload reduce if possible (cannot now due to hypotension)
  • Inotrope (*different* from pressor!)

• Diurese

• Mechanical support
  • IABP
  • LVAD

• Transplant?

• *Remember to look for an inciting cause!*
Inotropes vs. Pressors

- These agents do three basic things:
  - Vasodilate
  - Vasoconstrict ("pressor")
  - Inotropy
- What agent to choose = what are you trying to achieve?
- Septic patient: Problem is inappropriate vasodilatation → use vasoconstrictor
- Hypertensive pulmonary edema (patient 2): Problem is inappropriate vasoconstriction → use vasodilator
- Cardiogenic shock patient: Problem is weak muscle/low cardiac output → use inotropic agent + vasodilator (as tolerated)
What Do the Drugs Do?

- \(\alpha-1\): Vasocostrict
- \(\beta-1\): Inotropy (& chronotropy)
- \(\beta-2\): Vasodilate
- NO: Vasodilate
- Natriuretic peptide: Vasodilate
- Vasopressin: Vasoconstrict (‘vaso’ ‘pressin’)
- Phosphodiesterase Inhibitor: Inotrope/vasodilator
What Do the Drugs Do?

- **Pressors:**
  - Pure: Phenylephrine, Vasopressin
  - Mixed: Norepinephrine, Epinephrine, Ephedrine

- **Vasodilators:**
  - Nitroglycerin, Nitroprusside, Nesiritide (BNP)

- **Inotropes/vasodilators:**
  - Dobutamine, Milrinone

- **Inotropes/vasodilator/vasoconstrictor:**
  - Dopamine
IV Drips – From Vasodilators to Pressors

NTG/Nitroprusside/Nesiritide

Dobutamine/Milrinone
Dopamine
Epinephrine
Norepinephrine

Phenylephrine/Vasopressin

Vasodilatation

Inotropy

Vasoconstriction
A Word on Dopamine…

- Used frequently in CCU/ICU setting
  - Familiarity with it
  - Some inotropy, some BP ‘support’/no hypotension
- Hits dopamine, β-1, β–2, α–1 receptors
  - Lowest doses: Predominantly dopamine receptor
  - Smaller doses: Dopamine/beta receptors
  - Middle-higher doses: All receptors
  - Remember: None of this is pure!
- Dopamine vs. Dobutamine
  - Do you want some vasoconstrictive action or not?
Finally – A Word on BNP Monitoring

- BNP’s use: Distinguishing HF vs. non-HF cause of acute dyspnea
- Should we be measuring regular BNPs & guiding therapy by it?
- General answer: NO!
- Biggest trial: TIME-CHF trial
  - 499 patients age >60 with NYHA II-IV HF
  - All with HF hospitalization within past year
  - Intervention: Symptom-guided management or NT-BNP-guided therapy
  - Primary endpoints: 18-month survival free of hospitalization & QOL at 18 months
  - Not blinded to physician – only patient (possible bias)
No Difference in Hospital-Free Survival

Adapted from Pfisterer et al. JAMA 2009;301:383-92.
No Difference in QOL
(If Anything – Better Without BNP!)

<table>
<thead>
<tr>
<th>Outcomes by Group</th>
<th>Baseline</th>
<th>Month 12</th>
<th>Month 18</th>
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<tbody>
<tr>
<td>Minnesota Living With Heart Failure questionnaire, mean (SD)&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
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<tr>
<td>Symptom-guided</td>
<td>42.0 (20.3)</td>
<td>27.0 (18.6)</td>
<td>27.3 (21.5)</td>
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<tr>
<td>N-terminal BNP-guided</td>
<td>38.3 (20.2)</td>
<td>27.7 (17.9)</td>
<td>28.2 (17.6)</td>
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<tr>
<td>Duke Activity Status Index, median (IQR)&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Symptom-guided</td>
<td>7.3 (2.7-15.4)</td>
<td>15.2 (7.2-27.5)</td>
<td>12.7 (4.9-27.0)</td>
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<tr>
<td>N-terminal BNP-guided</td>
<td>7.2 (2.7-18.6)</td>
<td>12.8 (7.2-27.0)</td>
<td>12.8 (4.5-25.7)</td>
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<td>Short Form 12, mean (SD)&lt;sup&gt;c&lt;/sup&gt;</td>
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<td>Physical component</td>
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<td>46.1 (11.0)</td>
<td>50.8 (10.4)</td>
<td>50.1 (10.3)</td>
</tr>
</tbody>
</table>

Abbreviations: BNP, brain natriuretic peptide; IQR, interquartile range.

<sup>a</sup> Range of possible values is 0 to 105; lower values indicate better quality of life.

<sup>b</sup> Range of possible values is 0 to 58; higher values indicate better quality of life.

<sup>c</sup> Range of possible values is 0 to 100; higher values indicate better quality of life (a value of 50 is the average in the population).

*Adapted from Pfisterer et al. JAMA 2009;301:383-92.*
Survival without Hospitalization or Need for Increased Diuretics in BNP-Guided Management

Summary

- Five tricky patients you will encounter
  - In each case, time matters…
- Endocarditis
  - Need to know when to go to surgery!
- Tachycardia-induced cardiomyopathy
  - Early cardioversion if signs of heart failure
- “Pressure-overload” heart failure
  - Sublingual nitroglycerin, quick action
- Aortic dissection
  - *Sudden* onset of pain. Type A or Type B?
- Low-output heart failure
  - Abdominal symptoms, early inotropes
Thank you!