University of CINCINNATI

Preceptor: Heart Failure Academic Half Day

Agenda
1:10-1:20 Theory Burst
1:20-2:10 Case 1; Part A & B
2:10-2:20 Break
2:20-2:30 Questions for the Expert
2:30-3:20 Case 2; Parts A & B
3:20-3:30 Questions

Case 1, Part A:

Ms. Khristorozhdestvensky is a 58yo woman who presents as an Acute Visit for SOB in your first Tuesday PM clinic. You review her chart and find a history of poorly controlled diabetes, HLD, hypertension, obesity, chronic back pain, osteoarthritis of the knees, depression, and seasonal allergies. Her only medications are metformin and amlodipine. She has had swelling of her ankles in the last two weeks, but her PCP told her over the phone that it was probably the amlodipine.

Vitals:
T 98, HR 75, BP 133/75

Physical Exam:
General: no distress, appears well, short bungee cord is rigged to hold the front of her pants together, hand is warm when you shake it
HEENT: EOMI, PERRLA, MMM
CV: S1 and S2 normal, S3 present, regular rate and rhythm
Lungs: bilateral basilar crackles.
Extremities: pitting edema to the knees bilaterally
Neuro: answering questions appropriately, moving extremities spontaneously and equally
Abdomen: no tenderness, no distention, +BS

1. What other history do you need? What are key components of her exam? Which historical or physical exam finding has the highest likelihood ratio?

- SOB 2x weeks, worsening DOE, worsened yesterday evening **(DOE +LLR 1.3)**
- Could not fall asleep in bed, slept in recliner **(PND +LLR 2.6, orthopnea 2.2)**
- Edema **(+LLR 2.1, CI 0.9 – 5.0)**
- No chest pain, no lightheadedness, no cough
- Has not changed diet significantly recently
- Exam: S3 **(+LLR 11)**, Hepatojugular Reflux **(+LLR 6.4, CI 0.81 – 51)***, JVD **(+LLR 5.1)**, Rales **(+LLR 2.8)**, LE Edema **(+LLR 2.3)**, Wheezing **(-LLR 1.3)**

  * Check out the confidence interval on that!

1b. What is a likelihood ratio? Calculate the Positive LLR of PND if it has a sensitivity of 0.41 and a specificity of 0.84

LR+ = sensitivity / 1- specificity = 0.41/(1-0.84) = 2.56

LR expresses the odds that a given finding on the medical history or physical examination would occur in a patient with, as opposed to a patient without, the target disorder. When a finding's LR is above 1.0, the probability of disease increases (because the finding is more likely among patients with than without the disorder); when the LR is below 1.0, the probability of disease decreases (because the finding is less likely among patients with than without the disorder); finally, when the LR is close to 1.0, the probability of disease is unchanged (because the finding is equally likely in patients with and without the disorder)

2. What are the possible etiologies for these symptoms? Make a heart failure differential you can be proud of.

- Middle age female with cardiac risk factors (HTN, DM, HLD, obesity), unknown family Hx.
- Longstanding uncontrolled hypertension -> NICM -> HFpEF vs HFrEF
- Valvular dysfunction (AS or MR)
- CAD with ischemic cardiomyopathy
- Drugs (cocaine, EtOH, anthracyclines: doxorubicin – if history of leukemia/lymphoma)
- Restrictive CM / Systemic signs/symptoms (sarcoidosis, hemochromatosis, or amyloid)
- High output heart failure (chronic anemia or hyperthyroidism)

3. What do you do next?

Get a pulse ox = 92%, not too shabby but also not really normal.

Get an EKG: Ask them to eval the attached EKG. Subendocardial ischemia: The most striking abnormality is the widespread ST depression, seen in leads I, II and V5-6. This is consistent with widespread subendocardial ischemia. There is also some subtle ST elevation in V1-2 and aVR with small Q waves in V1-2, suggesting that the cause of the widespread ischemia is a proximal LAD occlusion.

4. Form a management plan based on the EKG.

First of all: safety check. They need to talk with attending in clinic.
Would they call rapid in clinic? Would they send to the ED? Try a direct admission? Can they send straight to cath lab?

ACS Management Interleaving: ASA, statin, ticagrelor (prasugrel now!), lovenox, sublingual nitro.

What would they do about the beta blocker? Give it due to ACS or withhold due to suspected acute systolic decompensation. ACC/AHA says BB is contraindicated if you think acute heart failure. Can they explain why?

Give Oxygen? No. Use only in patients with oxygen saturation < 90%, respiratory distress, or high-risk hypoxemia

5. What labs and what studies do you want and WHY?

Renal: normal except Na 131
CBC: normal
Troponin: 12
BNP or NTproBNP: elevated
TSH: normal
Lipids: LDL 155
A1C: 9.2
Bedside TTE: left systolic function looks grossly reduced. Cards fellow thinks there is possible anterior wall motion abnormality.
Formal TTE: LVEF 20-25%, G1DD, anterior wall motion abnormal, LVH, normal valves
CXR: some bilateral pulmonary edema.
LHC: 90% occlusion of proximal LAD. DES placed. TIMI flow after intervention is 3.

6. You are now the primary team for this patient after her intervention. She feels better, but still is short of breath when lying down. Still has crackles. Still has pitting edema to the knee. What medications for heart failure do you want to start?

- Ask them to discuss how they conceptualize heart failure therapy as a whole – acute vs chronic, afterload reduction/volume control/rhythm control, treating the compensatory mechanisms, alleviating symptoms vs improving mortality

Medicine	Targets	Outcome	Titrate
ACE-i	Afterload Reduction, Block RAAS (compensatory mechanism)	Decrease mortality, decrease symptoms of heart failure, improve functional capacity, and improve left ventricular ejection fraction.	Increase as tolerated based on blood pressure and symptoms. Higher doses of ACE inhibitors compared with lower doses have been shown to decrease heart failure hospital admissions but not mortality.
Diuretics (Furosemide, Bumex, Torsemide)	Volume control	Improve symptoms, reduce readmissions	Titrate to lowest tolerated dose that produces/sustains euvolemia
Beta Blocker (bisoprolol, carvedilol, Metop XL)	Block chronic adrenergic compensatory mechanism and decrease remodeling	Decrease mortality, reduce heart failure symptoms, and improve left ventricular ejection fraction	**AFTER acute decompensation** treated and HDS. β-blockers should be started at a very low dose. titration of a β-blocker should be performed slowly at 1- to 2-week intervals, on an outpatient basis. Titrate to max tolerated dose. HR <60.
Aldosterone Antagonist (spironolactone, eplerenone)	Block RAAS component, some volume control, decreasing remodeling post-MI		STEMI patients with systolic dysfunction within 3-14 days. Ephestus Trial.

7. She's ready for discharge now. How will you keep her from coming back with readmission?

-Discharge instructions! Be CRYSTAL CLEAR with med rec. Stop amlodipine. Heart's Connect
-Education of low sodium diet and fluid intake (2g/2L). Don't just TELL them to eat a low sodium diet. Give them examples. Tell them the hidden places sodium can be found (canned foods, bread, frozen veggies).
-Home weights and log
-Outpatient follow up within 7 days.

8. When do you repeat the TTE? Why?

Repeat in 3-6 months. Need to give the meds time to titrate to optimal doses. Need to give systolic function a chance to recover. Heart failure from CAD with successful intervention has a better chance to recover EF!

Case 1, Part B:

Clinic six months later…

She has had improvement in her symptoms from when you first met her at the acute visit. No crackles or peripheral edema. She is able to cook, clean and shop without symptoms, but still has significant shortness of breath with exercise. A quick medication reconciliation reveals she is on optimal doses of her BB, ACE-i, and diuretic. The results of the follow up TTE you ordered reveals an EF of 30%. Vitals normal with HR 60. EKG is normal (thank goodness. You were scarred from last time).

9. What other medications and interventions could you consider?

- What is her NYHA class now? (This in the appendix)

Medication	When to use? Why?
Isordil + hydralazine	Afterload reducers. This combination may be added for black patients who remain symptomatic despite therapy with ACE inhibitors and beta blockers, and as tolerated without reducing the doses of the ACE inhibitor or beta blocker to subtarget dosages. Also could be used for patients who did not tolerate ACE/ARB due to renal disease.
Aldosterone antagonist	All symptomatic patients with heart failure due to reduced ejection fraction, and three to 14 days post–myocardial infarction in patients with reduced ejection fraction and symptomatic heart failure or concomitant diabetes mellitus.
Valsartan-sarcubitril	Paradigm-HF trial. Superior to ACEi or ARB if prior success with ACEi or ARB and not hypotensive. Do not cross with ACEi within 36 hours
Ivabradine	Patients with sinus rhythm and HR > 70 with optimal BB onboard. EF<=35%. Sinus modulator, I_f channel inhibitor, Decreases CV death.
Digoxin	Digoxin should be considered for those who remain symptomatic despite therapy with all other disease-modifying agents. Positive inotrope. Digoxin may provide symptomatic benefit. If there is no benefit, the drug may be withdrawn; however, withdrawal may lead to clinical deterioration and should be done with caution.
ICD	EF <=35%, life expectancy >1 yr, max medical therapy, NYHA II-III

CRT	EF <=35%, NYHA III-IV (II?), max medical therpay, QRS >150, LBBB. In approximately 30% of patients, heart failure progression is accompanied by dyssynchrony (dysfunctional ventricular electromechanical coordination) manifested by prolongation of the QRS duration or a left bundle branch block.

For bonus: Explain how they get the leads into place for a BiV device.

In addition to the usual placement of a pacer lead in the apex of the right ventricle, an additional lead is placed through the coronary sinus down a coronary vein on the lateral wall of the left ventricle

Case 2, Part A:

Mr. Fartlek is a 58yo man with a past medical history of chronic non-ischemic systolic heart failure (last EF 20% on TTE 1 year ago), HTN, mild COPD who presents overnight after 1 week of nausea, anorexia, weight gain, and belt notch increase. He is short of breath after walking from his hospital bed to the bathroom. Typically, he can walk about 1 block without shortness of breath. His medications include: lisinopril, carvedilol, and spironolactone. He denies chest pain, lightheadedness, and cough. Reports occasional palpitations.

VS – T 98.6, P 100, R 16, 110/70, 91% on 2LNC
Gen – minimal distress, sitting up in bed, warm handshake
Neck - + JVD to the level of the jaw while sitting up in bed
CV – normal S and S2, S3 gallop, regular
RESP – Crackles at bases bilaterally
GI – Soft, nontender, nondistended. +hepatojugular reflux
EXT – 3+ pitting edema bilaterally to knees

Renal Panel: 140/4.0/101/24/21/1.3
CBC normal
Troponin 0.06
NTproBNP 21000

The EKG shows a QRS duration of 110 msec and normal sinus rhythm.

TTE shows ejection fraction of 20%, left ventricular enlargement, and moderate mitral regurgitation but otherwise no anatomic abnormalities of the mitral valve (unchanged from previous echocardiogram 1 year prior).

CXR with pulmonary edema and cardiomegaly

1. **Describe common precipitants of acute decompensations of heart failure.**

Nonadherence with medication, Na or fluid restriction
Acute myocardial ischemia
Uncorrected Hypertension
AF and other dysrhythmias
Recent addition of negative inotropic Rx: verapamil, diltiazem, BB

Initiation of Rx that increase salt retention: steroids, TZDs, NSAIDs
EtOH or illicit substance use
Endocrine abnormalities: uncontrolled DMII and thyroid dysfunction
Concurrent infections
Additional Acute Cardiovascular Dx: endocarditis, pericarditis

2. Where would you admit this patient, what is your initial plan?
He's wet and warm. Could admit to the floor with telemetry. Respiratory status and trajectory might make a good argument for step down in a more cautious resident.

3. What should we do with his Lisinopril? What should we do about his carvedilol?
The patient's usual outpatient medications (for example, ACE inhibitor, β-blocker) should be continued unless the patient is hypotensive or demonstrates signs of poor perfusion, in which case dose reduction or discontinuation of both the ACE inhibitor and β-blocker should be considered. In patients with signs of low-output heart failure (hypotension, worsening kidney or liver function, cool extremities), the β-blocker should be discontinued.

They should feel CONFIRM that patient is taking meds as prescribed. Talk to patient, look at pill bottles and count how many pills remain vs dispense date. Call pharmacy to see last fill date. Etc. Starting BB in patient who has not been taking their BB could trigger cardiovascular collapse through inotropic depression.

Case 2, Part B
At 6:30 AM, you get a page from Mr. Fartlek's nurse. He's not answering her questions normally and he seems much slower in his responses than he was earlier in the night. She's concerned that he has hospital delirium and was hoping for a Seroquel order to be placed for tomorrow night to avoid this happening again. Also, his breathing seems to be worse.

5. You tell your senior and then go to evaluate the patient. What thoughts are running through your mind as you walk over? What contingency plans are you making?
Just let them play the scenario out.

Evaluate his mental status while RN gets his vitals. Repeat the EKG. Review telemetry. Labs. Might need to call a rapid to escalate his level of care depending on situation. Might need to get nursing supervisor involved. Cards fellow? Cards attending?

6. He is cold to the touch and does not know where he is. His crackles are worse. Breathing is labored. Nurse says telemetry was showing intermittent tachycardia through the night, but it never sustained longer than 1 minute. Vitals: Afebrile, HR 110, BP 76/44, RR 26, O2 89% on 2L O2. You call a rapid response and help arrives quickly. The AOD asks you for information about what is going on. What do you say?

Some version of: "Mr. Fartlek is a 58yo man with a past medical history of chronic systolic heart failure and an EF of 20%, mild COPD, and HTN. He was admitted tonight with acute decompensation of his heart failure. Initial assessment was that he was volume overloaded but not in cardiogenic shock. We gave him 80 mg of IV Lasix at 10pm, Lisinopril, and Coreg. He is now cold to the touch and his vital signs and physical exam are worse. I am concerned he is now in cardiogenic shock. He is full code."

The AOD agrees and the patient is moved to the CVICU.

7. Stat labs return and show the following:

VBG: 7.39/35
BMP: 132/5.1/91/19/42/2.1

What's the acid base status? Does it make sense?

Anion gap metabolic acidosis from cardiogenic shock with concomitant underlying metabolic alkalosis from aggressive diuresis, with appropriate respiratory compensation. Yes, it makes sense.

1. Determine the anion gap (and osmolar and urinary gaps if indicated)
    a. AG is 22
2. Apply the rules of compensation
    a. Winter's Formula: 19 x 1.5 + 8 (+/-2) = 35
    Yes, it is appropriately compensated
3. Calculate the Delta/Delta
    a. 22-12/24-19 = 10/5 = 2
    b. Concomitant metabolic alkalosis

8. What do you think the CVICU should do?

*Cardiogenic shock requires intensive therapy with **intravenous inotropes**. Patients who remain in shock despite intravenous therapy and with worsening organ function, shock state should be re-evaluated, and then should be considered for mechanical support.*

*It is important to quickly rule out reversible causes in patients with cardiogenic shock. Reversible causes include acute myocardial infarction; ventricular septal or free wall rupture; and acute valvular regurgitation, possibly related to papillary muscle rupture, infection, or ascending aortic arch aneurysm with dissection of the aortic valve. **Bedside echocardiography** can be helpful in identifying structural causes of cardiogenic shock.*

Patients with cardiogenic shock secondary to progressive heart failure are generally given an inotropic agent, such as dobutamine or milrinone (cleared by the kidneys). Patients with peripheral vasoconstriction (increased systemic vascular resistance) often benefit from the addition of a pure vasodilator such as sodium nitroprusside. Placement of a right heart catheter can be helpful to assess filling pressures, cardiac output, and systemic vascular resistance to

help choose the appropriate medical regimen. Although the routine placement of a right heart catheter for patients admitted with heart failure has not been shown to improve outcomes, it should be considered to assist in therapeutic decision-making in patients with cardiogenic shock.