University of CINCINNATI

Spacing AHD 2019

Agenda

1:00 – 1:10: Theory Burst

1:10 – 2:00: Small group with questions

2:00 – 2:10: Break

2:10 – 3:30: Small group with questions; End AHD

Case 1. The MegaCase

Ms Spacey is a 61-year-old female with h/o hypertension, HLD, schizophrenia with frequent medication noncompliance, and type 2 diabetes who presents with altered mentation and hyperglycemia. EMS was called to her home when her daughter found her at home combative and agitated. EMS found her POC glucose reading to be >600 and she was brought to the ED. When able to comply with examination, she complains only of nausea and lower chest / epigastric abdominal pain. She reports being out of her insulin for multiple days. Meds include: metformin 1000 mg BID, glipizide 5 BID, lispro 4 TID, lantus 20 qHS, lisinopril 20 mg, ASA 81 mg, risperdone 1 mg BID

Vitals: T 98.1, HR 104, BP 153/79, RR 18, SPO2 97% RA

Gen: awake, alert, slow to respond to questioning, intermittently does not respond at all, intermittently drowsy
Head: normocephalic, atraumatic
Eyes: EOM intact, Conjunctiva normal, pupils equal and reactive to light
Neck: normal range of motion w/o tenderness; no lymphadenopathy
Cardiovascular: Tachycardic rate, without murmur
Pulm: CTAB, no w/r/r, slightly tachypneic
Abd: soft, non-tender, nondistended, +BS
Neuro: alert, oriented to self, city, situation. No CN deficit. Moves all 4 extremities 5/5 strength.
Skin: No rashes, lesions, wounds, edema

Labs:

80%Seg	\ 12.7 /	129 | 87 | 62 /
	16.1 ----- 250	---------- 962
	/ 35 \	4.2 | 11 | 2.66 \
		Prior Cr 1.2

1.) You are the ED resident in B pod. What is your initial working diagnosis and work up?

Diabetic patient presenting with AMS in setting of hyperglycemia, AMS, nausea, abdominal pain with hyperglycemia should raise concern for HHS v DKA.

Next steps: VBG, BHB, infectious work up with UA, CXR

Acid/Base Discussion

Provide learns with VBG: 7.19/23/9. Work through this acid base case.

80 – 19 = 61 == 61 = 24 x 23/9 (valid)

AG? 129 - (87 + 11) = 31 (AGMA)

Winters? 1.5 x 11 + 8 +/- 2 = 24.5 +/- 2 =~= PCO2 23 (approp respiratory compensation)

Delta? 31-12 / 24 – 11 = 19 / 13 = 1.4 (pure AGMA; recall general delta delta principle <1 = + NAGMA; >2 = +metabolic alkalosis; 1-2 = pure AGMA)

BHB 7 (elevated); UA +ketones, 1-3 WBC, 0 RBC, no bacteria; CXR wnl

Lactic acid 3.1, ECG NSR, troponin pending, BCx pending.

AGMA with AMS + AKI, reasonable to discuss toxic alcohol ingestions. Is this a patient you should check a serum osmolality? Calc Osm = 334; Measured Osm = 332.

DKA/HHS Discussion

DKA	HHS
• Short (< 1-2d) clinical course of fatigue, polyuria, polydipsia, and weight loss	• Progressive (several days) fatigue, polyuria, polydipsia, blurry vision
• GI complaint	• Gradual decline in mental status
• Altered mental status	• Dehydration
• Dehydration	• Typically older patient with other acute illness who has delayed seeking care
• Kussmaul respirations	
• Fruity breath odor	

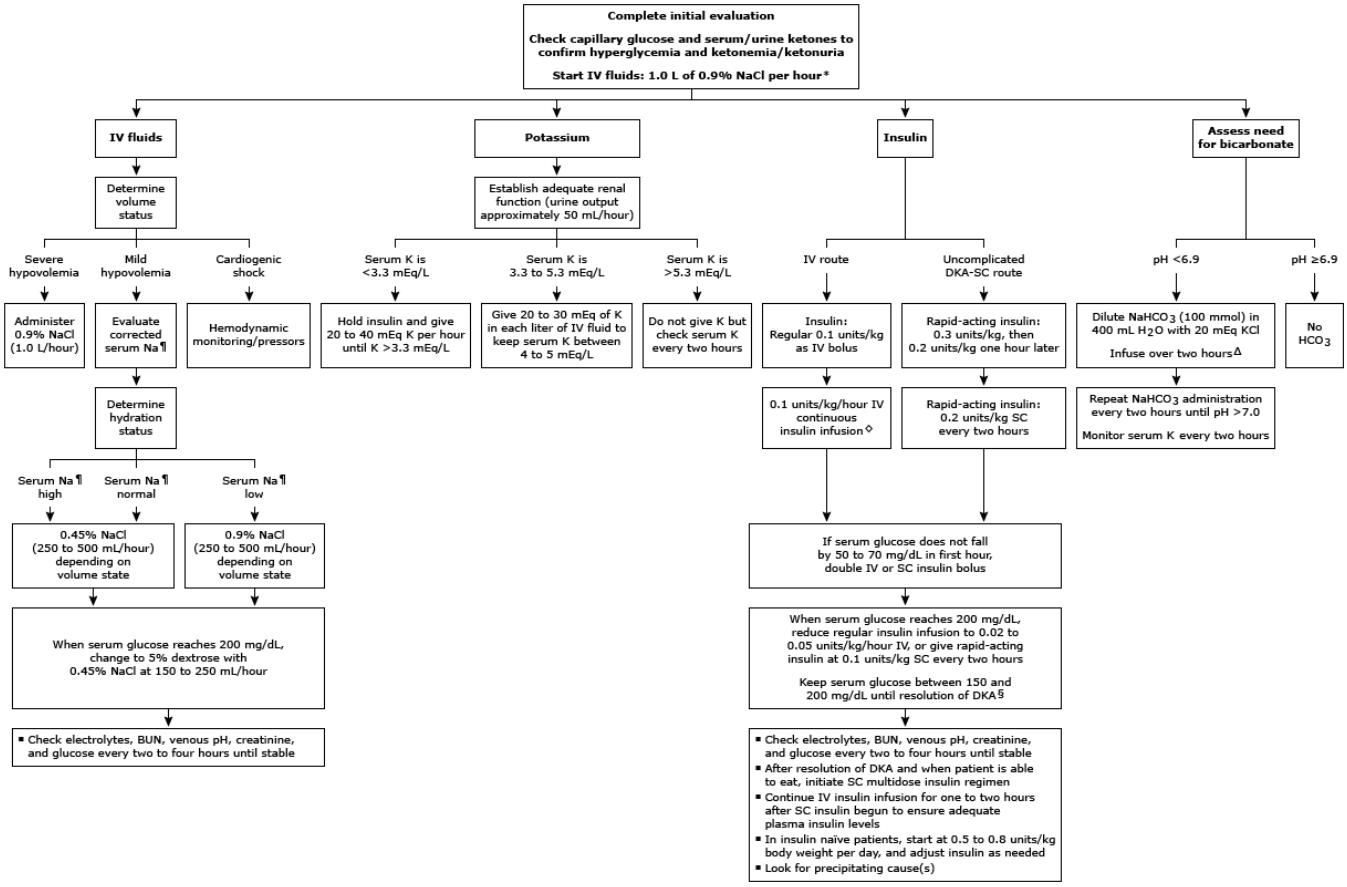
Table 1—*Diagnostic criteria for DKA and HHS*

	DKA			HHS
	Mild (plasma glucose >250 mg/dl)	Moderate (plasma glucose >250 mg/dl)	Severe (plasma glucose >250 mg/dl)	Plasma glucose >600 mg/dl
Arterial pH	7.25–7.30	7.00 to <7.24	<7.00	>7.30
Serum bicarbonate (mEq/l)	15–18	10 to <15	<10	>18
Urine ketone*	Positive	Positive	Positive	Small
Serum ketone*	Positive	Positive	Positive	Small
Effective serum osmolality†	Variable	Variable	Variable	>320 mOsm/kg
Anion gap‡	>10	>12	>12	Variable
Mental status	Alert	Alert/drowsy	Stupor/coma	Stupor/coma

*Nitroprusside reaction method. †Effective serum osmolality: 2[measured Na^+ (mEq/l)] + glucose (mg/dl)/18. ‡Anion gap: $(Na^+) - [(Cl^- + HCO_3^-$ (mEq/l)]. (Data adapted from ref. 13.)

2.) What is your assessment and plan? Discuss level of care (stepdown versus ICU). Frequency of labs?

- **Fluids** – Critical first step. Patient with DKA or HHS are dehydrated with an estimated water deficit of ~100 ml/kg of body weight among patients with DKA and ~100–200 ml/kg among patients with HHS. Fluid therapy restores intravascular volume and renal perfusion and reduces the level of counter-regulatory hormones which helps correct the hyperglycemia
- **Insulin** – DKA arises due to an absolute lack of insulin. HHS arises due to a relative lack of insulin. Insulin administration restores cellular metabolism, reduces hepatic gluconeogenesis, and suppresses further lipolysis and ketogenesis
- **Potassium** – Patients with DKA and HHS have a total-body potassium deficit. Despite this deficit, the serum K+ level measured is frequently within the normal range or even elevated owing to the shift of intracellular K+ to the extracellular compartment in the setting of hypertonicity, insulin deficiency and acidosis. Must frequently check during therapy because insulin stimulates movement of K+ intracellularly.
- **Bicarbonate –** This is rarely required in DKA and NEVER used in HHS



3) Why did the patient go into DKA/HHS?

Assess for underlying infection, silent MI.

This patient will have a troponin of 0.75 when checked. In addition, while you are beginning to assess the patient for possible ACS, the patient has new vital sign changes:

T 101.4, HR 123, BP 94/58, RR 32, SPO2 100% RA

She continues to be drowsy on exam and intermittently responding to questions. Family states she is not at her baseline.

Sepsis Discussion:

5) Is this sepsis?

1. Sepsis = life-threatening organ dysfunction caused by a dysregulated host response to infection
    a. qSOFA: AMS, RR>/= 22, SBP </= 100
    b. qSOFA is just a screening tool, 2/3 = high risk
2. Septic Shock = subset of sepsis with profound circulatory, cellular, and metabolic abnormalities resulting in increased mortality
    a. Sepsis + pressors for MAP > 65 in a fluid resuscitated patient + lactate >2

What is your assessment/plan for these new vital sign changes?

Patient now febrile, with +qSOFA. Has had leukocytosis. Possible infectious etiology driving DKA presentation. As discussed above, UA and CXR unrevealing. Blood cultures pending. Patient has been altered --> is it time to discuss lumbar puncture?

Do you empirically treat or wait for source? What is the associated mortality increase with each hour of delay in antibiotics? **7-12% PER EACH HOUR DELAY**

---> MENINGITIS REVIEW

Recall: Our patient had no meningismus on exam. The **Kernig** (inability to allow full knee extension when hip is flexed to 90 degrees) and **Brudzinski** (spontaneous flexion of the hips during attempted passive neck flexion) signs are not useful in ruling in or ruling out bacterial meningitis. The only test that might have diagnostic utility is the **jolt accentuation test** (patient with headache quickly turns head from side to side and testis positive if headache worsens), which has a high negative LR.

Does this patient need CT prior to LP?

- H/o CNS diseases - Includes those associated with CSF shunts, hydrocephalus, or trauma, those occurring after neurosurgery, or various space-occupying lesions.
- New-onset seizures, <1 week
- Immunocompromised state - Includes HIV/AIDS, immunosuppressive therapy, or hx transplantation
- Suspicious signs of increased intracranial pressure or space-occupying lesions – Papilledema, Focal neurologic signs (i.e. abnormal LOC, gaze palsy, abnormal visual fields, facial palsy, arm drift, leg drift, abnormal language)
- Moderate-to-severe impairment of consciousness (GCS <10) - an inability to answer 2 consecutive questions correctly or to follow 2 consecutive commands

Empiric antibiotics indicated if LP is delayed or purulent meningitis.

CTH delays LP by 1 hour. If CTH, then start ABX before LP.

Empiric Treatment:

- IV vancomycin 15-20 mg/kg q8-12h with goal serum trough of 15-20 mg
    - Indication: for ceftriaxone-resistant S. pneumonia
- IV ceftriaxone **2g** q12h or 4g q24h
    - Indication: for S. pneumoniae, H. influenzae, Neisseria meningitides
- IV dexamethasone 0.15 mg/kg q6h for 2-4 days
    - First dose 10-20 minutes before or with first antibiotic dose
    - Decreases CNS inflammation that leads to lower mortality, fewer short-term neurologic sequelae, and decreased hearing loss in pneumococcal meningitis when used as adjunctive therapy in developed countries.
    - Recommended in all adults with suspected/proven pneumococcal meningitis (IDSA Grade A-I). Insufficient data to recommend in adults with meningitis by other pathogens (IDSA Grade B-III).
- IV acyclovir 10 mg/kg IV every 8 hours
    - Indication: to all patients with meningoencephalitis pending outcome of diagnostic studies
- IV ampicillin 2 g q4hrs
    - Indication: for Listeria monocytogenes which is Indicated as empiric therapy for patients > 50 years old or immunocompromised (includes pregnancy!)

Begin IVF resuscitation: **30 cc/kg over 3 hours, Crystalloids > Colloid, with goal of lactate normalization and MAP >65**

If fails, initiate vasopressors:

1. Norepinephrine first (instead of dobutamine, SOAPII)
2. Add vasopressin (VASST) to help decrease NE dose
    a. This has a different mechanism than the catecholamine pressors, V1 mediated vasoconstriction
3. Add Hydrocortisone 200mg/day in divided doses if MAP goal not attained (APROCCHSS)
    a. physiologically we think septic patients are relatively adrenally insufficient
    b. No real benefit seen in ADRENAL and HYPRESS studies

ACS Discussion:

Patient with HTN, T2DM has troponin elevation 0.75. She described upper chest/epigastric discomfort and nausea. Repeat troponin 1 hour later is 1.74. What is your approach?

DDx: Acute coronary syndrome (STEMI, NSTEMI, UA).

Dx	Chest pain	ECG	Biomarkers

Unstable Angina	1. Pain with rest and prolonged, ~ longer than 20 minutes, 2. New onset, or 3. Previously diagnosed angina now more frequent, longer in duration, or occurs with a lower threshold	+/-	negative
NSTEMI	Symptoms consistent with ACS	+/-	positive
STEMI	Symptoms consistent with ACS	ST elevation	positive

Sx: Typical chest pain (all 3: substernal location, provoked by exertion, relief with rest; 2/3 = atypical; 1/3 noncardiac)

Risk stratify: Discuss TIMI, GRACE, HEART Scores – look these in MDCalc

Treatment:

Nitro to relieve ischemic pain

Beta Blocker should be started within 24hrs, but beware in decompensated HF (Level 1A); it decreases HR, BP, contractility, decreasing myocardial O2 demand

High intensity statin - Atorvastatin 80 mg started on admission (Level 1A) for stabilization of atherosclerotic plaque

ASA 325 then 81mg daily; blocks platelet aggregation and endothelial vasoconstriction PLUS

P2Y12 inhibitor (Plavix load - 600mg – or Prasugrel or Ticagrelor)

1. PLATO trial compared Ticagrelor and Clopidogrel, showing 16% reduction in MI, death or stroke with Ticagrelor. Also, fewer cases of in-stent thrombosis, and SAME rate of major bleeding; though other studies suggest higher incidences of bleeding

Anticoagulation: Heparin gtt or Lovenox

1. SYNERGY trial showed Lovenox was non-inferior to heparin but caused more major bleeding; EXTRACT-TIMI trial showed Enoxaparin overall clinical benefit.
2. Lovenox has limitations in renal patients, those going for surgery

Oxygen if O2 <90% (increased mortality with O2 if >90% (DETOX trial)

ACEi – Indicated for all patients in the first 30 days after MI. Most benefit in trials seen in patients with EF < 40% prior to discharge, also indicated if patient has HTN, DM, or CKD (Level 1A)

Other Workup/Orders: 1) **Serial EKGs**, 2) **Trend Troponins**, 3) **Cath lab plans**? 4) **NPO**

What are indications for urgent catheterization in ACS?

- STEMI
- ACS with hemodynamic instability or presentation of cardiac arrest
- ACS with angina refractory to aggressive nitrate therapy

- ACS with ventricular arrhythmias
- ACS with new or worsening valvulopathy

STEMI Cardiac Catheterization / LHC timeline goals:

1) LHC-capable facility: Door to balloon - 90 minutes or less
2) Non-cath capable facility: TPA or no TPA?
    a. If able to transfer and arrive to cath-capable facility within 120 minutes – NO TPA
    b. If transfer to cath facility > 120 minutes – TPA and then transfer

What are absolute contraindications to reperfusion therapy in STEMI? What do you do if they exist?

Absolute Contraindications	Relative Contraindications

• Any ICH previously
• Known AVM
• Ischemic Stroke (within last 3 months)
• Suspected dissection
• Active bleeding
• Bleeding diathesis
• Head/facial Trauma (within last 3 months)
	• Chronic Severe HTN
	• BP >180/100
	• Ischemic Stroke
	  • >3months
	• Traumatic/Prolonged CPR
	  • >10minutes
	• Major Surgery
	  • >3 weeks
	• Recent Internal Bleeding (2-4wks)
	• Pregnancy
	• PUD – active
	• Elevated INR

If contraindications exist, start medical management only and immediately transfer to PCI capable hospital.

You initiated NSTEMI management for this patient with rising troponin. Troponin peaked at 1.74 and began to downtrend.

CTH demonstrated a new low attenuation infarction of unclear etiology.

Ongoing Meningitis discussion:

9.) Your lumbar puncture returns with the following:

OP: High

CSF Color: colorless

Clarity: slighly hazy

TNC: 796

RBC: 296

Neut %: 94

Glucose: 240

Total Protein: 83

Interpret these results

Table 3: CSF profiles

Cerebrospinal Fluid Profiles					
Investigation	**Normal**	**Bacterial**	**Viral**	**Tuberculosis**	**Fungal**
Opening pressure	10-20 cm (50-180 mm H_2O)	High	Normal/high	High	High/very high
Color	Clear	Cloudy	Clear/cloudy	Cloudy/yellow	Clear/cloudy
Cells	< 5 mm^3	1,000-50,000 mm^3	50-1,000 mm^3	50-500 mm^3	0-1,000 mm^3
Differential	Mononuclear	Neutrophilic	Lymphocytic	Mononuclear	Mononuclear
Glucose	> 45 mg/dL (2.5 mmol/L)	< 40 mg/dL (2.2 mmol/L)	> 45 mg/dL (2.5 mmol/L)	< 45 mg/dL (2.5 mmol/L)	> 45 mg/dL (2.5 mmol/L)
Protein	< 45 mg/dL	100-500mg/dL	< 200 mg/dL =	50-300 mg/dL	> 45 mg/dL

Gram stain returns with Gram positive Cocci in pairs and clusters

---> speciates as MRSA

In addition, BCx x 2 return for GPC in clusters --> MRSA.

The patient's antibiotics are narrowed to IV Vancomycin.

10.) What is your next step in management?

ENDOCARDITIS DISCUSSION

MRSA bacteremia requires TTE for assessment of Infective Endocarditis. Can review Duke's Criteria here (at end of packet).

TTE reveals 9.2 x 7.9 mm mass on anterior chord of mitral valve.

What are High Risk Echocardiogram features?
- Large (>10mm in diameter) or mobile vegetations
- Severe valvular insufficiency
- Abscess cavities or pseudoaneurysms
- Valvular perforation or dehiscence
- Evidence of decompensated heart failure

When should a left sided IE patient undergo surgical treatment?:
- Valve dysfunction with signs/sx of heart failure
- IE complicated by heart block, annular or aortic abscess, or destructive penetrating lesions
- Evidence of persistent bacteremia (or fever) lasting > 5-7 days and other sources excluded
- Early surgery considered when caused by fungi or resistant organisms
- Early surgery reasonable for:

  - Recurrent emboli despite appropriate antibiotic therapy >7 days
  - Mobile vegetation > 10 mm and severe valve regurg
- Early surgery may be considered if mobile vegetation > 10 mm

Will this patient need IE prophylaxis in the future? Indications for antibiotic prophylaxis:
- Prosthetic cardiac valve or prosthetic material used for valve repair
- Previous infective endocarditis
- Some congenital heart disease
  - o Unrepaired cyanotic CHD
  - o Completely repaired CHD with prosthetic material/device during first 6 months after procedure
  - o Cardiac transplantation recipients who develop cardiac valvulopathy

Mini Cases

Syncope:

1. A 57 year old man presents to the emergency department after passing out while standing at church. After passing out he had shaking of both his arms and legs but he recovered completely within a couple of minutes. He does not recall the event, and does not know if he had symptoms before. He has a history of type 2 diabetes, hypertension, hypercholesterolemia, and hypothyroidism. His current medications are metformin, glyburide, hydrochlorothiazide, hydralazine, lisinopril, atorvastatin, and levothyroxine. His doctor recently increased hydralazine to better control his elevated blood pressure. Vitals pulse is 92 beats per minute, respirations 16/minute, blood pressure 132/76 mmHg, oxygen saturations 96% on room air. Physical exam is normal. Blood glucose is 104 mg/dl. Complete blood count, basic metabolic panel and urinalysis are all normal. Electrocardiogram is normal. Which of the following is the best next step in management?
    a. IV Normal Saline
    b. Trp and TTE
    c. Admission and tele
    d. EEG
    e. Orthostatic Vital Signs

Orthostatic Vitals: The correct answer is orthostatic vital signs. This patient's history is very consistent with orthostatic hypotension and is most likely due to his recent increase in hydralazine. Remember with standing that sympathetic system is activated to vasoconstrict and speed the heart rate up to continue perfusion to the brain. In a patient with recent increase in hydralazine it is difficult for him to peripherally vasoconstrict. Positive orthostatic vitals will show a drop of SBP by >20 or DBP >10 within 3 minutes of standing. It is very common for patients with syncope to have convulsions but the lack of posturing and quick recovery make seizure very unlikely. In a patient at high risk of cardiogenic syncope it is not unrealistic to consider admission or an ECHO but both would follow the completion of initial evaluation with orthostatic vitals.

2. In a patient that presents with syncope what is the most high yield initial test after performing performing a thorough history and physical?
    a. Echocardiogram
    b. Carotid ultrasound

c. **Electrocardiogram**
d. CBC and Renal panel
e. CT head

The correct answer EKG History and physical can make the diagnosis in 50% of cases. EKG makes the diagnosis in 5%. Basic lab work makes the diagnosis in 2-3%. Echocardiogram is only helpful if suggested by history. Carotid ultrasound is usually not helpful.

3. 42 year old woman is brought to the urgent care after she developed lightheadedness, diaphoresis and nausea in front of a large group while giving a presentation She reports her vision "blacked-out" and she collapsed to the ground. Her co-workers report that she recovered within 1 minute, had no confusion but did have jerking of her right arm after collapsing. She denies any chest pain or palpitations prior or during the episode. She has a history of hypertension and type 2 diabetes. Vitals signs: pulse 86 beats per minute, blood pressure 125/82 mmHg in both arms and negative orthostatic changes. Physical examination is normal. Electrocardiogram is obtained which is normal sinus rhythm with no other abnormalities noted. What is the next appropriate step?
    a. Electroencephalogram (EEG)
    b. **Discharge home with no additional work-up**
    c. Bilateral carotid Doppler
    d. 30-day event monitor
    e. Continuous Holter Monitor

This woman had neurocardiogenic syncope after standing for a prolonged period of time during a stressful event (presenting in front of several people). She had stimuli sent to her medulla from both cardiac C fibers (decreased venous return) and cerebral cortex (fear) which resulted in inappropriate increase in parasympathetic tone and decrease sympathetic tone. She has no risk factors for cardiogenic syncope; age less than 45, no cardiac disease, chest pain or dyspnea during the episode, normal ECG, and she was not exerting herself during the episode.

For episodes of syncope that are clearly classified as Neurocardiogenic syncope without any risk factors for cardiogenic syncope and normal ECG, no further work-up is needed. An EEG would be inappropriate this presentation is not consistent with seizure. If she had risk factors for cardiogenic syncope that are concerning for obstruction (i.e. syncope on exertion, chest pain or family history of sudden cardiac death) then an echocardiogram would be indicated. There is no indication for bilateral carotid dopplers in the work-up for syncope, if there was concern for stroke or TIA then it would be an appropriate test. If there was concern for cardiogenic syncope due to arrhythmia then a 30-day event monitor would be an appropriate work-up.

4. A 72 year old man presents to clinic with loss of consciousness. He has a history of ischemic cardiomyopathy, (ejection fraction 40%) hypertension and hyperlipidemia. He's had 3 similar syncopal episodes in the past 2 weeks. Two of the episodes occurred while he was seated at home, the third episode while mowing the lawn. In each case, he had palpitations immediately preceding the loss of consciousness but he denies diaphoresis, nausea, or shortness of breath. Vitals are temperature 98.8 F, pulse 75 bpm, respirations 12/minute, blood pressure 140/75 mmHg, pulse oximetry 100% on room air. Orthostatic blood pressure and heart rate in the office are normal. Physical examination shows normal S1, S2 without murmurs, rubs or gallops. Electrocardiogram shows sinus rhythm with no ST or T wave changes. Which of the following is the best next step?
    a. Electroencephalography (EEG)

b. Outpatient echocardiogram
c. Outpatient cardiac stress
d. Reassurance
e. **Hospital admission with telemetry**

The correct answer is hospital admission with telemetry. The clinical scenario is very concerning for a cardiac cause for the syncopal episodes. Cardiomyopathy increases the risk for life threatening arrhythmias. Furthermore, palpitations prior to syncope suggests an arrhythmia. Syncope during exertion also supports a cardiac cause. The patient requires admission with telemetry.

5. A 65 year old man presents to the emergency department after an episode of syncope at work. He has never had a history of syncope. He denies dyspnea on exertion, palpitations or chest pain. His family history is unremarkable for any sudden death or collapse. Physical examination reveals a III/VI crescendo-decrescendo systolic murmur at the right upper sternal border with radiation into the carotid arteries. Pulses are weak and have a slow upstroke. After confirming severe aortic stenosis with echocardiogram what is the next best step?
    a. Echo every 6-12mo
    b. Echo every 1-2 yrs
    c. Echo every 3-5yrs
    d. **Aortic valve replacement**
    e. Aortic valve balloon dilation

Severe aortic stenosis that is causing symptoms (angina, dyspnea on exertion, syncope) is an indication for surgical aortic valve replacement. Balloon valvotomy has high rate of complications and restenosis and should only be used for stabilization and a bridge to surgery.

This patient is presenting with symptomatic aortic stenosis after a syncopal episode and therefore is a candidate for surgical aortic valve replacement. The other indications for aortic valve replacement include; 1) asymptomatic aortic stenosis with left ventricular dysfunction (ejection fraction <50%) 2) very severe aortic stenosis (Vmax >5m/sec) and low surgical risk 3) patients with decreased exercise tolerance 4) moderate aortic stenosis who are undergoing other cardiac surgery 5) asymptomatic severe aortic stenosis with rapid progression. Transcatheter aortic valve replacement (TAVR) may be considered in patients who have high surgical risk.

****** Valve review:**

Stenosis – Treat for Symptoms

- AS – syncope, angina or heart failure --> aortic replacement
- MS – Afib or heart failure --> percutaneous mitral balloon valvotomy

Regurgitation – Follow the Rules

- MR
    - Asymptomatic MR < 60% EF or ≥40 mm end systolic dimension; new afib is strong modifier and may consider even if > 60%. This is VALVULAR Afib!
        - ---> mitral valve repair is preferred to valve replacement
- AR
    - Asymptomatic = < 50% EF or > 50% but LVESD > 55 mm or LVEDD > 75 mm

- --> aortic replacement

Pneumonia

1. 44 year old man with type 2 diabetes, COPD, and alcohol abuse presents to your office with cough for 3 days and subjective fevers at home. His cough is productive of green sputum. He is tolerating fluids. He did receive azithromycin 3 weeks prior and prednisone for a possible COPD exacerbation. His vitals are within normal limits. Physical examination shows right lower lobe crackles but no wheezes. You diagnose the patient with pneumonia. Which of the following is the best antibiotic regimen?
    a. **Levofloxacin 750 mg daily**
    b. Amoxicillin-clavulanate 2 grams BID
    c. Amoxicillin-clavulanate 2 grams BID and azithromycin 500 mg daily
    d. Doxycycline 100 mg BID

a) Level I evidence for patients with risk factors for drug resistant s. pneumoniae (alcohol use, DM, COPD). The patient is at risk for drug resistant S. pneumoniae given his history of DM, COPD, and alcohol use. He has recently been on a macrolide which increases the risk of resistance and thus is not an appropriate choice for this reason as well. The recommended antibiotic choice according to the IDSA guidelines for a patient who meets criteria to be treated as an outpatient with risk factors for drug resistant S. pneumoniae is a flouroquinolone (moxifloxacin, gemifloxacin, or levofloxacin) Level I evidence.

b) Can be used WITH a macrolide per guidelines

c) This is an appropriate option for someone who had not recently had a macrolide. Our patient was given a macrolide in the past month which makes this inaccurate.

d) This is not an appropriate option for a patient with risk factors for drug resistant s. pneumoniae.

2. 44-year-old man presents to your office with worsening productive cough over 4 days and fever to 102 F. He has been unable to go to work due to malaise. He denies rhinorrhea, headache, nausea, vomiting, or diarrhea. He is a non-smoker and has no past medical history.
Vitals temperature 102.4 F, pulse 96 beats/min, respiratory rate 18/min, blood pressure 110/70 mmHg, oxygen saturations 96% on room air. Physical exam shows an ill but nontoxic appearing male. He is breathing comfortably with crackles noted over the right lung base with deep inspiration. There is no egophony or increased fremitus. What is the most appropriate treatment plan for this patient?"

A. Observation and close follow up
B. Outpatient treatment, Azithromycin
C. Outpatient treatment, Amoxicillin plus Azithromycin

D. Outpatient treatment, Levofloxacin
E. Inpatient treatment, Ceftriaxone plus Azithromycin
F. Inpatient treatment, Piperacillin-tazobactam plus Vancomycin

The correct answer is outpatient treatment and azithromycin. The guidelines for outpatient treatment of community acquired pneumonia with no comorbidities or risk factors for drug resistant strep pneumoniae is a macrolide (level 1 evidence) or doxycycline (level 3). This patient has no comorbidities. If the patient were to have risk factors for drug resistant S. pneumonia or comorbidities such as heart, lung, liver or renal disease, diabetes, alcoholism, asplenia and malignancy then the treatment would be a respiratory fluoroquinolone or B-lactam + macrolide. Regarding inpatient or outpatient treatment, remember to use **CURB-65** to help decide the need for admission. He is alert (C=confusion), respiratory rate (R = respiratory rate >30), blood pressure is normal (Systolic BP <90 mmHg or diastolic <60mmHg) and he is young (age >65). Even if labs reveal a BUN >19 (U in CURB-65= uremia) he still only has 1 point which does not require hospitalization.

location	Previously healthy and no risk factor(s) for drug-resistant *Streptococcus pneumoniae*	Risk factor(s) for ...
Outpatient	Macrolide or Doxy	...for drug-resistant *S. pneumoniae* or underlying comorbidities Resp FQ or b-lactam + macrolide or doxy
Inpatient (non-ICU)	Resp FQ or b-lactam + macrolide or doxy	…for *Pseudomonas aeruginosa* or gram-negative rods on sputum Gram stain Antipseudomonal β-lactam with pneumococcal coverage (cefepime, imipenem, meropenem, or piperacillin-tazobactam) + RFQ ….For + CA-MRSA, cavitary infiltrates, or compatible sputum Gram stain + Vanc

Comorbidities: (chronic heart, lung, liver, or kidney disease; diabetes mellitus; alcoholism; malignancies; asplenia; and immunosuppressive conditions or use of immunosuppressive drugs), recent (within 3 months) antimicrobial use, or residence in regions with a high rate (>25%) of infection with high-level (minimum inhibitory concentration ≥16 µg/mL) macrolide-resistant *S. Pneumoniae.*

3. 68-year-old homeless man presents to the emergency department with 3 days of right-sided pleuritic chest pain, subjective fevers, and productive cough. His past medical history includes hypertension, hyperlipidemia, and alcohol abuse. Vital signs are 100.7 F, blood pressure 118/78 mmHg, pulse 98/min, respiratory rate 24/min, oxygen saturation 92% on 2L. Physical examination reveals decreased breath sounds and dullness to percussion in lower third of the right lung. Chest x-ray shows right basilar consolidation and a right-sided pleural effusion. A diagnostic thoracentesis is performed, results are as noted. What is the most likely cause of this pleural effusion?

Serum	**Pleural Fluid**	
Protein 7.1 g/dL LDH 165 IU/L (normal 140 - 200)	**Protein** 4.7 g/dL **LDH** 146 IU **Glucose** 20 mg/dL **pH** 7.24 **Red Blood Cells** 564	**Total Nucleated Cells** 12,568 Neutrophils 85% Lymphocytes 10% Monocytes 5% Gram Stain none

A. Heart failure
B. Tuberculosis
C. Small cell lung cancer
D. Alcoholic cirrhosis
E. Bacterial pneumonia

This patient has an uncomplicated parapneumonic effusion due to bacterial pneumonia. Parapneumonic effusions are pleural effusions that are secondary to pneumonia. This patient is presenting with signs and symptoms of a bacterial pneumonia, additionally his pleural effusion is exudative based on **Light's Criteria (TP eff/TP serum >0.5 or LDH eff/LDH serum >0.6 or LDH eff >2/3 ULN of LDH serum**), Total neutrophil count >10,000 with a neutrophil predominance. Heart failure and cirrhosis can result in a transudative pleural effusion. While tuberculosis and small cell lung cancer result in an exudative pleural effusion, they typically are lymphocyte predominant. It is not uncommon to have a negative gram stain in a parapneumonic effusion. **The definition of a complicated parapneumonic effusion includes the presence of pus OR gram stain positive pleural fluid OR a pleural fluid pH <7.2, gluc <40, LDH >1000.**

<u>Liver Disease</u>

1. A 52-year-old man comes to the emergency department because of abdominal pain and fever for the past 2 days. Past medical history is significant for chronic hepatitis C and cirrhosis. His temperature is 38 °C (100.4 °F), pulse is 95/min, respirations are 16/min and blood pressure is 98/60 mmHg. Physical examination shows spider angiomata, palmar erythema, and sclera icterus. He has a distended

abdomen with a positive fluid wave and diffuse tenderness to palpation. Mental status examination is normal. Laboratory tests are shown below;

Serum	**Ascites**
White blood cells - 14,000 cells/mircoL (**AST**/SGOT) 70 Units/L (**ALT**/SGPT) 79 Units/L **Albumin** 2.8 g/dL **Total bilirubin** 5 mg/dL International normalized ratio (**INR**) 1.9 **Creatinine** 1.2 mg/dL	Total cell count 600 cells/mm3 Neutrophils 50 % Albumin <1.5 g/dL

Which of the following is indicated at this time?

A. Prednisone
B. Cefotaxime plus albumin
C. Lactulose
D. Furosemide
E. Propanolol

The correct answer is **Cefotxime and albumin**. This patient has spontaneous bacterial peritonitis (SBP). He presents with typical symptoms of fever and abdominal pain. The diagnosis is confirmed because he has greater than 250 polymorphonuclear cells (PMNs) in the ascitic fluid (600 cells x 50% neutrophils = 300 PMNs). Antibiotic treatment is indicated with a third generation cephalosporin such as cefotaxime or ceftriaxone. Albumin infusion is also indicated for the prevention of renal failure in patients diagnosed with spontaneous bacterial peritonitis when the creatinine is > 1 mg/dL, the blood urea nitrogen is > 30 mg/dL, or the total bilirubin is > 4 mg/dL. Treatment is initiated within 6 hours of diagnosis (1.5 g/kg) and is repeated on day 3 (1.0 g/kg).

Prednisone is given in patients with suspected alcoholic hepatitis when their calculated discriminant function is greater than or equal to 32. This patient does not have a history of alcohol use, and liver function tests do not suggest alcoholic hepatitis (AST 2x > ALT). In addition, the patient has spontaneous bacterial peritonitis, so steroids should be avoided.

Although **lactulose** is often indicated for patients with cirrhosis and evidence of hepatic encephalopathy, this patient has a normal mental status examination. Therefore, lactulose is not indicated at this time.

Diuretic therapy is often indicated for extravascular volume overload in patients with cirrhosis. However, this patient has signs of sepsis secondary to spontaneous bacterial peritonitis. Adding a diuretic in this setting is not the next best step.

Patients with **spontaneous bacterial peritonitis** do much worse when treated with a beta blocker. They are used often for prevention of variceal bleeding but once SBP develops they should be permanently discontinued.

2. A 40 year old man presents to the emergency department with ascites. A diagnostic paracentesis is performed and they are now calling for admission. The results are the following. Which of the following is the most likely etiology of this patient's ascites?
Serum: Albumin 3.2 g/dL, Protein: 5.6 g/dL
Ascitic fluid: Albumin 1.0 g/dL, Protein: 2.0 g/dL

A. Cardiac ascites
B. Tuberculous ascites
C. Peritoneal carcinomatosis
D. Nephrotic syndrome
E. Alcoholic cirrhosis

The correct answer is alcoholic cirrhosis. The SAAG (SAAG = serum albumin - ascitic fluid albumin) helps distinguish ascites caused by portal hypertension (SAAG >1.1) and other disease processes (SAAG <1.1). Diseases that cause SAAG <1.1 g/dL include peritoneal tuberculosis, peritoneal carcinomatosis, nephrotic syndrome, and pancreatitis ascites. Heart failure or liver cirrhosis both cause elevated portal hypertension and a SAAG gradient >1.1g/dL. These can be differentiated by the ascitic fluid total protein. Total protein <2.5 g/dL is consistent with ascites from cirrhosis; a high ascitic fluid protein >2.5 g/dL is consistent with cardiac ascites.

3.A Fourth Year Medical Student presents to his PCP office for new jaundice. He denies recent illness, pruritis, confusion, travel, or substance abuse. He states that he has recently completed a strenuous surgical acting internship. Exam shows scleral icterus with faint jaundiced skin. No HSM. Labs reveal: Hgb 14, PLT 150, AST 21, ALT 19, AP 45, T.Bili 2.1, indirect 1.8, Albumin 3.5, INR 1.0.

Next step in management?

Peripheral blood smear
Osmotic fragility test
Annual follow up
G6PD testing in 4-6 weeks

None; annual follow up would be sufficient for health care maintenance. This is Gilbert Syndrome. It is defined as benign, familial, mild, unconjugated hyperbilirubinemia related to deficiency in bilirubin UGT activity. The condition may be diagnosed incidentally at a routine medical examination or when blood being examined for another reason, for instance, viral hepatitis. Jaundice is mild and intermittent. Bilirubin levels are most often <3 mg/dl5. Jaundice may follow an intercurrent infection, fasting/dehydration, or stressor.

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Can you answer all of these learning objectives? MEGACASE covers Topics 1-6. The Mini-Q's cover 7-9. Your team has the option of self-directed learning for Topics 10-14. Any learning objective you can't teach to your friend deserves some extra TLC.

Topic	Learning Objectives
1. Acid Base	Review Acid Base Calculations Understand indications for toxic alcohol work up
2. ACS	Define typical chest pain Differentiate forms of ACS Utilize resource to risk stratify patients Initiate treatment of ACS Define contraindications and goals for early reperfusion therapy Define indications for urgent catheterization
3. Sepsis	Define sepsis Use qSOFA to screen for sepsis Review the golden hour of sepsis Initiate adequate fluid resuscitation
4. Endocarditis	Review Duke's Criteria for IE Initiate empiric treatment for suspected IE Know the high-risk features of IE on echo Indications for surgical intervention on left sided IE Indications for IE prophylaxis
5. Meningitis	Discuss the prognostic utility of Kernig and Brudzinski. Indications for CT prior to LP Know the time loss of obtaining CT prior to LP Interpret CSF studies Initiate empiric treatment for meningitis. Indications for dexamethasone in meningitis treatment
6. Diabetes	Review diagnostic criteria for diabetes Review types of insulin Initiate insulin regimen based on total daily dose Titrate insulin with 50/30 rule to goal (what is goal?) Differentiate DKA v HHS Initiate treatment of DKA/HHS Treat hypoglycemia
7. **Syncope**	State the definition of syncope State the 3 classifications of syncope Identify key clinical features that separate syncope from seizure Identify signs and symptoms that make a patient higher risk for Cardiogenic Syncope
8. **Pneumonia**	Define pneumonia based on the IDSA definition Explain the difference between CAP, HAP, and VAP. Use clinical decision tools to determine treatment location.

	Choose the appropriate initial work-up and antibiotic regimen for community acquired pneumonia based on patient risk factors and clinical setting.
9. **Liver**	Initiate work up of abnormal LFTs Diagnose Gilbert syndrome Diagnose and manage alcoholic hepatitis Recognize acute liver failure Manage chronic liver disease Diagnose and treat SBP Manage hepatic encephalopathy
10. Heart Failure	Manage Acute Decompensated Heart Failure Practice Guideline Directed Therapy for Chronic Heart Failure
11. Hem/Onc Emergencies, Liquid Tumors, Anemia	Define and treat neutropenic fever. Recognize the presentation of Tumor Lysis Syndrome. Understand indications for allopurinol, rasburicase, and dialysis in management of TLS. Recognize and treat acute cord compression. Work up hypercalcemia. Manage acute symptomatic hypercalcemia. Recognize and manage acute SVC syndrome. Recognize and treat leukostasis. Develop a differential work up for microcytic and macrocytic anemias. Prescribe iron replacement in IDA. Recognize and manage TTP. Develop a differential work up for thrombocytosis
12. COPD	Identify an obstructive and restrictive pattern on a pulmonary function test Define the diagnostic criteria for COPD Apply GOLD Assessment tool to initiate treatment for stable COPD patients Manage an acute COPD exacerbation
13. AKI	Discuss the diagnostic criteria for AKI based on KDIGO Explain three mechanisms of acute kidney injury Develop initial diagnostic and treatment plans for acute kidney injury
14. Hyponatremia	Initiate a systematic work-up of hyponatremia Recognize when hypertonic saline is indicated Understand brisk correction complications – cerebral edema and CPM. Explain the different treatment options for SIADH