

The EM Educator Series

Mini-case #8: When sepsis care becomes not so straightforward

Author: Alex Koefman, MD (@EMHighAK) // Edited by: Brit Long, MD and Manpreet Singh, MD

Mini-Cases:

1: A patient with ESRD on hemodialysis presents with dysuria, frequency, and suprapubic pain with fever. VS include T 38.2C, HR 112, BP 88/48, RR 24, and Sats 98% on RA. UA is significant for large leukocyte esterase, nitrates, and WBCs.

2: A patient with liver transplant presents with fever, cough, chills, and generalized weakness. VS include T 38.4C, BP 95/57, RR 19, Sats 91% on 2L. CXR shows RML infiltrate.

Questions for Learners:

- 1) How do you incorporate sepsis metrics and criteria with the patient in front of you?
- 2) Why is my sepsis patient not improving with resuscitation?
- 3) How does sepsis evaluation and management change with ESRD?
- 4) How does sepsis evaluation and management change with ESLD?
- 5) How does sepsis evaluation and management change with CHF?
- 6) How does sepsis evaluation and management change with pulmonary hypertension?
- 7) How does sepsis evaluation and management change with valvular heart disease?
- 8) How does sepsis evaluation and management change with transplant? Does the specific transplanted organ affect your management?

Suggested Resources:

- <https://emcrit.org/pulmcrit/sepsis-myths/>
- <http://www.emdocs.net/the-sepsis-patient-not-improving-after-iv-fluids-and-resuscitation-what-should-be-considered-how-can-we-improve/>
- <http://rebelem.com/occult-causes-of-non-response-to-vasopressors/>
- <http://www.emdocs.net/the-dangers-of-over-resuscitation-in-sepsis/>
- <http://www.emdocs.net/sepsis-comorbidities-management-strategies/>
- <http://www.emdocs.net/emdocs-cases-sick-esrd-patient/>
- <http://www.emdocs.net/approach-to-the-sick-cirrhotic-patient/>
- <http://www.emdocs.net/ed-management-heart-failure-pearls-pitfalls/>
- <http://www.emdocs.net/the-crashing-pulmonary-hypertension-patient/>
- <https://emergencymedicinescases.com/pulmonary-hypertension/>
- <http://www.emdocs.net/acute-valvular-emergencies-pearls-pitfalls/>
- <http://www.emdocs.net/transplant-emergencies-part-i-infection-rejection-and-medication-effects/>
- <http://www.emdocs.net/transplant-emergencies-part-ii-organ-specific-complications/>

Answers for Learners:

1) How do you incorporate sepsis metrics and criteria with the patient in front of you?

The dosing of intravenous fluids in septic patients should be taken as seriously as any potentially lethal medication. It is essential for physicians to give appropriate doses of intravenous fluids while avoiding fluid overload. Patients' fluid status must be re-evaluated after administration of fluids. Further research must be conducted to **identify the appropriate dosing of intravenous fluid bolus at onset of sepsis and any patient subsets that require different treatment.**

Check out [PulmCrit Six Myths](#) promoted by new surviving sepsis guidelines

2) Why is my sepsis patient not improving with resuscitation?

- Source identification – LUCCASSS.
- Ensure adequate preload, and determine whether further fluid resuscitation warranted.
- Antibiotic regimen coverage and dosage – beware of patient/microbe factors, as well as pharmacodynamics and pharmacokinetic drug effects.
- US for other causes using RUSH – Heart, IVC, FAST, Lungs.
- Balanced resuscitation – early vasopressor provision, which improves vasoconstriction, arterial constriction, positive inotropy, improved cardiac output, and renal perfusion.
- Metabolic/Endocrine concerns – hypocalcemia, adrenal insufficiency, thyroid disease (myxedema coma).
- Abdominal Compartment Syndrome – measure bladder pressure.
- Transfusion of Products – look for bleeding.
- Respiratory Status – if declining, take airway with intubation, place on low tidal volume settings.

occult causes of non-response to vasopressors

vital disclaimer: the cognitive response to hypotension should **not** be reaching for a vasopressor. the primary therapy for hypotension is **treatment of the underlying pathology**

definition: patients who despite substantial vasopressor doses do not show HD parameter improvements. failure of response → **cognitive pause + consideration of reasons**

considerations for non-responders

acidosis	dx: blood gas, basic metabolic panel tx: reverse underlying cause , bicarb gtt unlikely helpful, Continuous Veno-Venous Hemodialysis (CVVHD)
hypothyroid	dx: clinical, TSH (may be false neg or delayed) tx: levothyroxine (consider empiric tx)
anaphylaxis	dx: by history, may present as hypotension alone tx: epinephrine , methylene blue, ECMO
adrenal insuff	dx: clinical, depressed cortisol level, hyperK + hypoNa tx: hydrocortisone 100-200 mg (consider empiric tx)
hypoCa²⁺	dx: ionized Ca²⁺ , prolonged QTc tx: Ca salts (CaCl or CaGluconate)
occult bleeding	occult source: GI, retroperitoneal tx: reverse anticoagulation, transfuse, operative/IR control
toxicologic	occult causes: beta blocker/Ca channel blocker OD, TCA tx: Hyperinsulin Euglycemia Therapy (HIET), ECMO, Bicarb (for TCA)
2nd cause shock	patients can have multiple concurrent causes of shock . look for second cause RUSH protocol extremely useful when considering multiple causes

thanks to @emupdates, @criticalcarenow, @srrezaie

3) How does sepsis evaluation and management change with ESRD?

4) How does sepsis evaluation and management change with ESLD?

5) How does sepsis evaluation and management change with CHF?

- Despite comorbidities, sepsis is the primary threat to the life of the patient and must be treated
- SBP should not be overlooked in cirrhotic, and valuable culture data can be gleaned from inoculating culture bottles immediately after sample collection
- Early vasopressor usage in pulmonary hypertension and avoidance of positive pressure ventilation can preserve compromised right ventricular function
- Congestive heart failure patients, conversely, may benefit from positive pressure ventilation used judiciously during resuscitation

6) How does sepsis evaluation and management change with pulmonary hypertension?

- Patients with PH quickly decompensate – worry about obstructive and cardiogenic shock.
- Bedside echo is your #1 tool in undiagnosed PH – Assess the RV, is there hypertrophy? What is the RV:LV ratio? Is it >1? Is there septal bowing? What does the IVC tell you about the patient's intravascular volume status?
- Get a troponin – patients with severe PH often suffer from coronary ischemia secondary to RCA perfusion deficits.
- Your goals as an EM physician should be to:
- Reverse Underlying Causes When Possible
- Optimize RV Preload
 - Use your knowledge gained from the bedside echo to help you. REMEMBER: RV failure and hypotension are MORE COMMONLY the result of increased RV afterload and hypervolemia.
 - Intravascular hypovolemia: 250-500cc crystalloid boluses
 - Intravascular hypervolemia: controlled diuresis
- Support Systolic Function
 - Dobutamine is FIRST LINE: 2mcg/kg/min-10mcg/kg/min
 - Milrinone is SECOND LINE: 0.375mcg/kg/min-0.75mcg/kg/min
 - HYPOTENSION = Norepi
- Maintain RCA Perfusion
 - Norepi: Start at 0.05mcg/kg/min but caution with high doses = worsens pulmonary vasoconstriction
 - No Phenylephrine: worsens RV systolic function
- Reduce RV Afterload
 - SpO₂ > 90%
 - AVOID PPV in hypotensive patients
 - Try to avoid intubation and mechanical ventilation at all costs, but if you need to intubate: etomidate, lung-protective ventilator settings, and avoid permissive hypercapnea
- Rate Control Dysrhythmias
 - Rate control is much more important than rhythm control – PH patients are dependent upon the atrial kick to maintain CO
 - Cardiovert when indicated

7) How does sepsis evaluation and management change with valvular heart disease?

- Definitive management is surgery more often than not, so get consultants on board early.
- If you have a sick patient with a native valve emergency consider nitroprusside +/- dobutamine.
- If present, don't forget to treat the underlying cause of aortic regurgitation (aortic dissection, endocarditis), mitral regurgitation (ischemia, endocarditis), or prosthetic valve emergency (endocarditis, thrombosis).

8) How does sepsis evaluation and management change with transplant? Does the specific transplanted organ affect your management?

- Transplant patients possess significant variation in anatomy and physiology based on the surgical procedure and medications.
- Infection is the most frequent complication, classified by several different periods: within one month of transplant, one to six months, and after six months.
- Each time period is associated with different infections.
- Rejection occurs in several phases including hyperacute, acute, and chronic. Patient symptoms differ based on the specific organ.
- Immunosuppressive medication effects are common and have significant interactions with other medications commonly used in the ED.
- All transplant patients require consultation with the transplant team. Surgical consultation may also be required.
- Beware the transplant patient: involve the transplant physician, transplant surgeon, and specific organ consultant for evaluation and management.
- Renal complications post-transplant include infection, vascular obstruction, hematoma, ureteral obstruction, and lymphocele. Patients often present with edema, decreased urine output, and increased serum Cr.
- Liver transplant complications include vascular obstruction, biliary conditions (stricture, leak, biliary stone, fluid collection), biloma, and stricture.
- Cardiac transplant is associated with denervation, thus patients often do not present with chest pain, but symptoms of CHF including edema. Complications include vasculopathy with accelerated atherosclerosis and cardiac dysrhythmia.
- Lung transplant requires three anastomotic connections. Patients may experience obstruction, vascular complications, phrenic nerve dysfunction, and pleural lining complications.