



# 4/30/20 Morning Report with @CPSolvers



Case Presenter: Mike Rose (@MikeRoseMDMPH) Case Discussants: Imran Nizamuddin (@INizamuddinMD) & Lindsay Haselden

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| <p><b>CC:</b> found down (AMS)</p> <p><b>HPI:</b> 50ish male presents to ED <b>found down</b> on porch.</p> <ul style="list-style-type: none"> <li>- Audibly snoring (improves with jaw thrust)</li> <li>- No change in mental status after 2 amps of D50, fingerstick glucose remains &lt; 10</li> <li>- Naloxone given, some improvement</li> <li>- Reports he “snorted something”</li> <li>- Says he does not have diabetes</li> <li>- Reports a history of “problems with my heart”</li> </ul> <p>Improvements in UOP and mental status after diuretics</p> <p>ROS: diarrhea</p> | <p><b>Vitals:</b> T: 35 C HR: 113 BP: 182/131 RR: 16 SpO<sub>2</sub>: 92% (on RA)</p> <p><b>Exam:</b></p> <p><b>Gen:</b> initially not responding to sternal rub, moaning after naloxone</p> <p><b>CV:</b> JVD, no murmurs, rubs, gallops, muffled heart sounds. Tachycardic</p> <p><b>Pulm:</b> no crackles</p> <p><b>Abd:</b> diffusely tender after naloxone, no guarding</p> <p><b>Neuro:</b> pinpoint pupils</p> <p><b>Extremities/Skin:</b> cool to touch, delayed capillary refill</p> | <p><b>Problem Representation:</b> Middle age man with HFrEF and substance use disorder p/w AMS found to have cardiogenic shock, an osmolal gap, and toxicology consistent with cocaine induced hyperacute heart failure exacerbation and heroin intoxication</p>  |  |
| <p><b>PMH:</b><br/>Cocaine-induced cardiomyopathy (EF 10-15%)<br/>Substance use disorder<br/>CKD<br/>HTN</p> <p><b>Meds:</b><br/>Heart med<br/>BP med</p>  | <p><b>Fam Hx:</b></p> <p><b>Soc Hx:</b> lives with mom, usual state of health until morning of presenting</p> <p><b>Health-Related Behaviors:</b><br/>Heroin, Cocaine use</p> <p><b>Allergies:</b></p>  | <p><b>Notable Labs &amp; Imaging:</b></p> <p><b>Hematology:</b><br/>WBC: 17.3 (92% neutrophils), Hgb: 8.6 (nl MCV), Plt: 261</p> <p><b>Chemistry:</b><br/>Fingerstick glucose &lt; 10<br/>Na: 138 K: 5.4 Cl: 100 CO<sub>2</sub>: 8, AG: 30 BUN: 20 Cr: 1.7 (bl 1.6), glucose &lt; 2 → from upper arm IV similar BMP, <u>except glucose 247</u><br/>AST: 914 ALT: 221 Alk-P: 171 T. Bili: 3.2 Direct 2.9<br/>Troponin 0.76, lactate: 16<br/>VBG: pH 7.15/27/9<br/>SOsm: 300 (nl 285-295) Osm gap: 17, CK: 1092, proBNP: 127,000</p> <p><b>Imaging:</b><br/>EKG: normal sinus rhythm, no ST abnormalities<br/>CXR: clear lung fields, enlarged cardiac silhouette<br/>Echo: no pericardial effusion, no tamponade, grossly dilated IVC w/o respiratory variation. Greatly reduced EF<br/>Urine tox: positive for cocaine &amp; heroin</p> | <p><b>Teaching Points (Jack):</b><br/><b>Translate Found Down → AMS; Translate signs of hypoperfusion and JVD → Cardiogenic shock:</b> In any clinical case, we translate individual clinical findings into an entity that allows us to deploy a schema.<br/><b>Normal or elevated BP does not rule out shock.</b> Shock is a state of systemic hypoperfusion, which often happens with hypotension, but can also occur in normo- or hypertension.<br/><b>Rx before Dx in a patient found down:</b> ABCs and emergent interventions (D50, Naloxone, Intubation, +/- Thiamine).<br/><b>An initial approach to hypoglycemia</b> involves differentiating between <b>true hypoglycemia</b> or <b>pseudo/peripheral hypoglycemia</b> (peripheral vasoconstriction, delayed sampling, or leuko/erythrocytosis), especially in a patient with <b>hypoglycemia out of proportion with their MS.</b></p> <ul style="list-style-type: none"> <li>- In this case, the combination of D50-refractory hypoglycemia, poor cap refill, and elevated JVP suggest cardiogenic shock even in the <i>absence</i> of hypotension.</li> </ul> <p><b>Signs of a Cardiomyopathy + Clear Lungs?</b> Chronic HF → increase in capacity of pulmonary lymphatics vs. pericardial disease.<br/><b>Acute CHF:</b> Endocardial causes (acute MR), myocardial causes (ACS, Takotsubo, myocarditis) and pericardial causes (tamponade). However, we also have to consider a mimic which is compensated CHF with a severe acute trigger (e.g., infection or acute ingestion, as in this case).</p> |