

# 5/09/25 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter: Jerome Case Discussants: Reza (@DxRxEdu) and Rabih (@rabihmgeha)

<p>Scribing (Zakariyya G.)  <b>CC:</b> 81 yo male now with <b>progressive confusion</b> and abnormal behavior  <b>HPI:</b>  <b>Acutely</b> developed progressive AMS with fecal incontinence and loss of appetite.  <b>No focal deficits noted on exam.</b>          Not conversing.          No constitutional symptoms or narcotic use  <b>CTB + CXR negative</b>          Past 3 months: <u>Repeated admissions for severe PAD.</u>          Right foot toe Osteo due <i>Candida &amp; Pseudomonas</i> after surgery. On cefepime and micafungin  <b>1 month prior:</b> Multiple debridements. Developed DiHS/Dress. On 60mg prednisone daily.</p>	<p><b>Vitals:</b> <b>Afebrile, BP 135/68, pulse 84 O2 98 on RA. RR 17 : Baseline:</b>  <b>Normal neuro exam previously</b>  <b>Exam:</b> <b>Gen:</b> Uncomfortable <b>HEENT:</b> Normal  <b>CVS:</b> Normal  <b>Abd:</b> Slightly distended. No ascites. Mild Lower quadrant tenderness  <b>Neuro:</b> Alert. Able to track movement. Grunts and make "uhm" sounds.  <b>Clonus</b> of upper extremities. Lower: No clonus. Normal reflexes. Able to move extremities against gravity  <b>MSK:</b> Right toe surgical site: clean dressing</p>	<p><b>Problem Representation:</b> Elderly male with metabolic syndrome and cognitive impairment with acute AMS after being on Cefepime for Osteomyelitis. New AKI - found to have bilateral arm clonus on exam and abnormal speech, with cefepime levels confirming neurotoxicity</p>	
<p><b>PMH:</b>          Admitted to rehab facility one week prior  <b>Baseline:</b> Converses fully. Mild cognitive impairment but independent with ADLs          Vascular dementia - some evidence on imaging.          Metabolic syndrome          Diabetic Neuropathy          CKD GDR in 50s          Low visual acuity</p>	<p><b>Past Medical History</b>          Vascular dementia - some evidence on imaging.          Metabolic syndrome          Diabetic Neuropathy          CKD GDR in 50s          Low visual acuity  <b>Meds</b>          Cefepime, Micafungin, Losartan, SGLT-2, metformin, Clopidogrel, Aspirin. Prednisone 60 mg  <b>Allergies:</b> Clindamycin + Ciprofloxacin</p>	<p><b>Notable Labs &amp; Imaging:</b>  <b>Hematology</b>  <b>CBC:</b> Hb 10 (at baseline), <b>WCC 11 neutrophilic, 7.1% eos, Plate 260</b>  <b>Chemistry</b>  <b>Creatinine 4.06 (1.2 one week prior) BUN 49 (21 one week prior.) Bicarb 13 anion gap 15.</b> Normal sodium and potassium  <b>Tbili 3, indirect.</b> Normal liver enzymes. <b>Albumin 3.</b>  <b>VBG:</b> pH 7.26 PCO2 38, normal lactate, mildly raised Beta-hydroxybutyrate          Urine and blood cultures negative  <b>Urinalysis:</b> <b>UPCR 32.2!</b> Urine Na 65, 5-10 Eos on high power field. No casts. Some white cells. 1+ blood  <b>CT abdo/pelvis:</b> No hydro. Stable 12 cm cyst on right kidney. Moderate stool burden. No stercoral colitis  <b>ECG: RBBB</b>  <b>EEG: Negative</b>          Further history: given 2l RL. Nephro: Either AIN or ATN started on steroids          Renal biopsy done: Diabetic nephropathy. Tubulointerstitial changes.  <i>Admitted to ICU for dialysis after cefepime level found to be 45. Marked improvement within days</i></p> <p><b>Final Diagnosis: Cefepime Induced Neurotoxicity</b></p>	<p><b>Teaching Points (Seeme):</b>  <b>Approach to Cognitive impairment:</b>          We can ask what happened before, during and after the event. Important to know the baseline. Patients with dementia are more vulnerable. History is important. Mini Cog test can be done in which we ask patient to repeat three words.  <b>Approach to Confusion:</b>          When patient's attention and focus is oscillating, delirium is likely. Delirium is seen in vulnerable patients having a mild trigger i.e UTI, medication change, daylight pattern changes.  <b>Approach to past medical history:</b>          Background can be related to current condition. Patients with atherosclerotic disease can develop vascular dementia. Osteomyelitis is common in diabetic patients secondary to immune compromise and neuropathy. Source control is important. Bacteremia or medications can cause confusion.  <b>Approach to Clonus and Aphasia:</b>          Patients is wide awake but not converting properly. Clonus is marker of upper motor neuron sign. We can check for spasticity and other markers of upper motor neuron signs. We can do MIST workup (metabolic, infection, structural, toxins) and imaging.  <b>Approach to Kidney disease and medications:</b>          Eosinophils can be elevated by parasitic infections. SGLT2 inhibitors can cause DKA. Brain and kidney can be affected together by anything in blood stream i.e. infections and medications. Uremia can also cause confusion. Cefepime and cephalosporins can cause neurotoxicity.. Speech deficits are characteristic of cefepime induced neurotoxicity. Dialysis can help reverse some medication toxicities.</p>