



6/22/26 Mainstream Monday with @CPSolvers

"One life, so many dreams" Case Presenter: Hafsa Saeed (@) Case Discussants: Youssef (@saklawiMD) & Manaswini (@manaswiniikeshav)
<https://clinicalproblemsolving.com/present-a-case/>



Scribing (Varsha) CC: 61Y/M with weakness in all 4 extremities and tongue x 2 weeks HPI: Had flu like symptoms 2 weeks ago, self resolved. Metoprolol was increased from 25 to 50mg, and symptoms started after this change. The higher dose metoprolol was then stopped and switched to diltiazem. Weakness started in arms and tongue, then spread to legs. He also has SOB x 1 week. He then developed gait changes for 2 days ROS: no back pain, no headache, vision change or nausea/vomiting		Vitals: T: 97 HR: 67 bpm BP:138/87 mmHg RR:16 Sat: 99% BMI: Exam: Gen: well appearing, alert and conversant HEENT: facial flushing CV: normal Pulm: normal Abd: normal Neuro: Alert and oriented, GCS 15, higher ft, CN, visual field intact, 4/5 R hip flexion, all others 5/5, areflexia, decreased pinprick in stocking and glove distribution, diminished vibration in B/L toes, + romberg, wide unsteady gait, other cerebellar function normal	Problem Representation: 61Y/M presented with weakness, SOB and gait abnormality with recent h/o self resolving flu like symptoms. Neurological exam revealed areflexia, abnormal sensory system exam and + romberg sign. CSF analysis was positive for albuminocytological dissociation and he was diagnosed with AIDP
PMH: Depression HTN Obesity Atrial fibrillation Meds: Diltiazem Carvedilol Eliquis	Fam Hx: - Social Hx: - Health-Related Behaviors: Alcohol use Allergies: -	Notable Labs & Imaging: Hematology: WBC: 10 Hgb:17.4 Hct: 48.7 Plt: 322: Chemistry: Na:136 K: Cl: 98 HCO3: 21 Cr: 0.84 BUN: 17 Glucose: 130 Ca: 9.6 Mg: AST: ALT: Alk-P: 96 Bili: Albumin: Total Protein: ESR: CRP: LDH: TSH 3.2 HbA1c: 5.5 COVID: negative Tick panel and HIV, B12 and folate : normal CSF: WBC 6, Protein 142, RBC 2000, glucose 83, negative cytology and culture. Imaging: Echo: LVEF 55%, unremarkable MRI: orbit, face, neck C spine: Inflammatory Vs infectious polyradiculopathy, confederation for GBS, less likely inflammatory/infectious arachnoiditis, lyme disease, sarcoidosis, leptomenigeal carcinomatosis MRI T-spine and L- spine: Normal Dx: Acute inflammatory demyelinating Polyneuropathy Patient received IVIG x 5 days, PT & OT, gradually improved and discharged.	Teaching Points (Seeme) Approach to weakness: -muscle / neuromuscular junction/ nerves/ brainstem/brain, it important to localize the problem -widespread involvement makes us think about electrolytes/muscles as well Approach to HPI: - Weakness from arms to legs shows a descending pattern. -Flu or campylobacter history can make us think about GBS -We can look for signs of UMN vs LMN signs -No cognition deficits and CN involvement takes us away from UMN lesion, lack of sensory involvement makes us think about motor neurons or NMJ or myopathy. -Descending pattern makes us think about GBS variants(Miller fisher), myasthenia gravis (evolves slowly-fluctuating), botulism (evolves in hours) -Eyes are involved in botulism and myasthenia gravis Approach to Exam Findings and Labs: -Decreased sensation and areflexia could be indicative of polyneuropathy -Polyneuropathy differentials :ABCT- A(AIDP), B(B vitamin deficiency), C(cauda equina), T(toxins) -Romberg test indicates proprioception, peripheral nerves involvement can cause positive romberg test as well. Approach to imaging and LP analysis: Albuminocytological dissociation can be seen in Guillain Barre Syndrome