



10/2/25 Morning Report with @CPSolvers

"One life, so many dreams" Case Presenter: (Usha George@) Case Discussants: (Rabih@rabihmgeha) and (Noah@Noah_Nakajima)
<https://clinicalproblemsolving.com/present-a-case/>



<p>Scribing (Seeme)</p> <p>CC and HPI:</p> <p><u>Case 1:</u> 60 y old male with asthma with cough and exacerbation-associated rhinitis, no fever, chest pain or SOB</p> <p><u>Case 2:</u></p> <p>45 y old male with SOB and cough for 1 month, SOB with blood streaks, progressive SOB, rash on fingers and toes, bilateral LL weakness and worsening gait. No fever.</p>		<p><u>Case 1:</u></p> <p>Vitals: T: nl HR: nl BP: nl RR: nl</p> <p>Sat: 93</p> <p>Exam: Gen: cushingoid habitus</p> <p>HEENT: rhinosinusitis</p> <p>CV: wnl</p> <p>Pulm: expiratory wheeze, bronchial hyperreactivity</p> <p>Abd: wnl</p> <p>Neuro:wnl,Extremities/skin:wnl</p>	<p><u>Case 2:</u></p> <p>Vitals: T: 37.2 HR: 112 BP: nl RR:</p> <p>Sat: 92 BMI:</p> <p>Exam: Gen: alert</p> <p>HEENT: no JVD, CV: wnl</p> <p>Pulm: bilateral diffuse crackles</p> <p>Neuro: decreased sensation L below knee, reflexes and CN: nl</p> <p>Extremities/skin: left limb weak, rash on legs</p>	<p>Problem Representation:</p> <p><u>Case 1:</u> 60 y old M with refractory asthma, migratory lung consolidations presented with cough and rhinosinusitis.</p> <p><u>Case 2:</u> 45 y old M presented with SOB, rash and polyneuropathy, was found to have positive p-anca and anti MPO antibodies.</p>
<p>PMH:</p> <p><u>Case 1:</u></p> <p>Asthma (diagnosed 3 y ago)</p> <p><u>Case 2:</u></p> <p>Not significant</p> <p>Meds:</p> <p><u>Case 1:</u></p> <p>Steroids, Triple inhalers, Chinese medication, containing steroids</p> <p><u>Case 2:</u></p> <p>Not significant</p>		<p>Fam Hx:</p> <p>Not significant</p> <p>Social Hx:</p> <p><u>Case 1:</u></p> <p>Retired administrator</p> <p><u>Case 2:</u></p> <p>No smoking or alcohol</p> <p>Health-Related Behaviors: -</p> <p>Allergies:</p> <p>NKDA</p>	<p>Teaching Points (Sarah B):</p> <p><u>Case 1:</u></p> <ol style="list-style-type: none">1) Asthma tends to present early in life due to intrinsic vulnerability coupled with environmental triggers. It is unusual for a patient to have a new diagnosis in their 60s, so they should be fully evaluated with PFTs with methacholine/bronchodilator etc. if not already done.2) Cushingoid habitus tells us that the patient received a lot of steroids. Asthma should be steroid-responsive, so this indicates a misdiagnosis. Alveolar disease also indicates that this was either not asthma at all or had become a complication of steroid treatment (e.g. infection).3) Reasons why an 8am cortisol may show up negative in a patient with cushingoid features: the physical exam is imperfect, intermittent exogenous steroid exposure having cleared the last dose, and taking a steroid not picked up by the lab (e.g. dexamethasone). The eosinophils being normal does not necessarily exclude their role in the disease process—all exogenous steroids should be stopped to prove eosinophilia.4) ABPA and EGPA may present with consolidation and reversible obstruction—negative ANCA makes EGPA less likely. EGPA also causes bronchiectasis. ABPA should be ruled out by antigen testing. Hypereosinophilic syndrome (HES) is challenging to distinguish from EGPA. It is easiest to distinguish these with specific tests like ANCA, biopsy showing granulomatous disease, or specific mutations associated with HES. Chronic eosinophilic pneumonia is a diagnosis of exclusion requiring disease restricted to the respiratory tree.5) Parasitic syndromes would be likely to cause more systemic disease over the course of a 3 year span, especially in the context of exogenous steroid use. <p><u>Case 2:</u></p> <ol style="list-style-type: none">6) Blood streaked sputum may help localize the disease to the lungs. In a patient with blood streaked sputum, chest imaging is often helpful to characterize disease. The rash, weakness, and gait disturbance would then be best framed as systemic manifestations of a pulmonary disease.7) Hypoxia was a clue that there was gas exchange impairment, coupled with crackles on exam, this was most likely an alveolar problem. Purpura with neuropathy and weakness made us concerned that the lung involvement was diffuse alveolar hemorrhage from vessel ischemia—in particular, small vessel vasculitis (MPGA, PAN, ANCA, etc.). In patients with suspected small vessel vasculitis, CT chest, UA, CBC/differential are helpful in characterizing the extent of lung disease, eosinophilia, and GU involvement.8) EGPA develops over years. Patients start with asthma then get extrapulmonary manifestations (GI, cardiac) which are eosinophil driven. The final stage is vasculitic, where patients infarct organs.	
		<p>Notable Labs & Imaging:</p> <p>Hematology:</p> <p><u>Case 1:</u></p> <p>WBC: 6.6 (eosinophils 2%) -> increased eosinophils 12% (on repeat tests) Hgb: 13</p> <p>IgE: raised, Cortisol AM :low ->repeat: nl, ANCA and ANA: nl, IgE aspergillus: negative</p> <p>Imaging:</p> <p>CT scan chest: consolidation present -> repeat CT showed new consolidation in right middle lobe and lingula</p> <p>Patient advised to stop chinese medication and discharged on inhalers, returned with wheeze, started on anti-IL5 receptor.</p> <p><u>Case 2:</u></p> <p>Hematology:</p> <p>WBC: 13 (31% eosinophils absolute eosinophil count: 4, Hb: 12 PLT: 135</p> <p>Cr and urinalysis: nl, sputum AFB: normal, P-anca: positive anti MPO antibodies: positive</p> <p>Imaging:</p> <p>CT scan: bilateral consolidation and ground glass opacities</p> <p>EMG: mixed polyneuropathy</p> <p>Patient started on methylprednisolone, IV cyclophosphamide added, and rituximab added</p> <p>Dx: EGPA</p>		