



11/8/24 Morning Report with @CPSolvers



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

CC: 51 year old male presents w/right eye pain & blurred vision for 3 days

HPI: ICU consulted due to Afib w/ hypotension

- Further history: Associated with visual loss. Eye found to be red, proptotic & swollen with confirmed fevers
- No pain with eye movement.
- No trauma.
- No sick contacts, no med changes.
- No prior history of eye issues

ROS: Headaches (mild), palpitations, light-headed. Diarrhea & nausea (profound).

PMH:

DM2 on insulin
HPT
Dyslipidemia
OSA
Ulcerative Colitis (negative recent colonoscopy)

Meds:

Vedolizumab
Alegra
Apixaban
Mesalamine
Insulin Glargine
Atorvastatin
Fexofenadine

Fam Hx: None

Social History:

Retired Navy.
Worked in IT

Health-Related Behaviors:

Tobacco.
No drugs/alcohol

Allergies:

Shell-Fish

Further history

Had 2 x metoprolol pushes and BPs responded to leg raise. Started on ABx

Vitals: T:101F, HR: 135, BP: 95/55, RR: 18, O2 98% on room air

Exam: General: Notably Diaphoretic.

HEENT: Normal eye muscle movement. Mild pain on right lateral gaze.

Purulent discharge & conjunctival erythema. Decreased vision. Left eye normal
CVS: Tachycardic, irregular, no murmur, no JVD
Pulmonary: Clear lungs and no respiratory distress

Abdominal : SNT, no organomegaly

Neurological: Withdrawn but fully oriented. Normal pupils. No focal deficits noted

Dermatological: Warm, well perfused. No edema. No rash

Notable Labs & Imaging:

Hematology:

WCC 1.3 (lymphocytic predominant) 3% basophils (elevated). Absolute neutrophil count: ZERO! Hb 8 with normal Reticulocyte count, Plat 370

Chemistry: Na 136, K3.8, Bicarb 22, BUN 17, glucose: 199 TBili 1.5

Normal AST, ALT. Total protein 5.3 and Albumin: 2

INR 1.45 **TSH: undetectable w/ T3 5 (markedly elevated) and mildly raised T4**
Troponin: 100 (120 1h later) **Urinalysis** negative.

Imaging: CT head: unremarkable CXR: mild infiltrate, mild cardiomegaly

CT orbit: No post septal pathology. Preseptal cellulitis, right proptosis > left. No fracture. Ethmoid thickening.

Echo: N EF. Diastolic dysfunction. No WMAs or thrombus

On Further history: Recent hospital admission prior after URI: Afib since w/markedly low TSH, elevated T3 & both **Thyroid receptor antibody and TSH antibodies positive.** He had been started on methimazole & eliquis.

Final Diagnosis: Grave Disease with methimazole-induced agranulocytosis

Problem Representation: 52 yo male with UC & DM2 presents w/acute, inflammatory red eye & Afib. Imaging showed preseptal cellulitis and labs notable for severe agranulocytosis and confirmed antibody positive Grave Disease

Teaching Points(Khashayar)::

Rx first

Atrial Fibrillation ->conceptualize Afib as a compensatory tachy arrhythmia mechanism vs a sinister rhythm as primary pathology -> As such we don't expect it to trigger the hypotension so we put our focus on the hypotension and start fluids

Hypotension -> Hypovolemic vs cardiogenic vs obstructive vs distributive -> inflammatory state can produce many of these states approach all of them despite the distributive being more likely

Immunocompromised patient in the context of chronic disease -> Not only are they immunocompromised they are compromised in multiple arms of the system -> Increased risk of common infections & other atypical infections -> Empiric antimicrobial therapy and maybe even considering fungal therapy considering the immunocompromised state (although because fungi dont typically have a rapidly progressive infection you dont usually use empiric therapy)

Approaching the ocular features -> verify everything in exam

Usually happen in isolation

Eye pain -> locate where the pain is coming from -> pain in the eye itself (conjunctival injection) vs somewhere else(many patients with primary headaches)

Red Eye -> inflammation(allergic,infection) vs venous engorgement

localization -> the surface(conjunctivitis, uveitis, etc) vs the globe(orbital melanoma,GPA, etc) vs the surface around the eye

Proptotic eye -> pushing the eye outside of the orbit -> concerning for problem in the globe(while unlikely in this context consider thyrotoxicosis) -> how to check for it? -> Looking to see if the cornea exists in a more anterior position compared to the lower lead -> stand to the side of patient

Pus in the eye -> pus cannot leak out of the eye without it rupturing -> there is either pus leaking around the eye or pus is invading the eye -> graves doesn't cause pus

This can also be coming from the eyelid -> periorbital infection due the eyes being open for so long -> ask the wife , how was the disease progression ->

Pain with eye movement -> the globe itself is bumping into the something -> staph and strep prioritization -> pain in lateral gaze -> hypertrophy of eye muscles in graves disease is the cause of proptosis -> the proptosis happens in this order -> I(inferior) aM(medial) S(Superior)L(Lateral)O(Oblique)

Orbital disease -> isolated vs presentation of systemic disease vs presentation of local disease (invasive sinus disease)

Lid Lag -> increased sympathetic tone in the eye because of the excess in thyroid hormone

Approaching the Labs

WBC & Hgb down -> neutropenic fever

- etiology? -> Sepsis through cytokine release causing marrow suppression(high PLT favors this) or infiltration of the marrow(infection vs blood cancer) -> PBS showing the marrow crying will tell us about infiltration -> tear drop cells

- Treatment -> broad broad spectrum antimicrobial therapy

Undetectable TSH -> points to T3 predominant thyrotoxicosis -> excessive synthesis => the most common cause is graves when there is iodine supplementation in the population -> The only factor that causes proptotic eye -> only graves causes this -> anemia & leukopenia can be explained by thyroid storm in case the progression is too fast
T4 predominant thyrotoxicosis -> thyroiditis predominant picture

Miscellaneous

There is no evidence that allergy to shellfish alters the risk of reaction to intravenous contrast more than any other allergy