04/27/21 Neuro Morning Report with @CPSolvers

***Case Presenter**: Kirtan Patolia (@Kirtan Patolia) **Case Discussants**: Valeria Roldan (@valeroldan23) and Marco Malaga(@MarcoMalaga97)*

CC: Behavioral changes

HPI: 16F
2 weeks: Difficulty in voiding urine constipation and back pain. Behavioral changes: Episodes of irrelevant talking, anger, irritability, confusion, temper tantrums, disorientation, and incoherent speech.
Denies: Fever, cough, chills, altralagias, myalgias, rashes, weight loss, dizziness, visual blurring, double vision or hearing loss. Not weakness, and sensory loss.

Vitals: **T**: Nl **HR**: 80-120 **BP**: Nl **RR**: Nl **SpO_2**: Nl
Exam: Not her usual self
Systemic
Neuro
- **Mental Status:** Disoriented to time and place
- **Cranial Nerves: Nl**
- **Motor:** Strength ⅗ in LL, ⅘ UL. Muscle bulk and tone: Nl
- **Reflexes:** Plantar downgoing plantar bilaterally
- **Sensory:** -ve in pain/temp, +ve difuse hyperaesthesia.
- **Cerebellar:** Nl
- **Other:** Meningeal signs -ve.
- **Higher function:** Cognitive impairment with short-term memory loss
- **Psychiatric:** Elevated mood, confusion,disorganized incoherent speech, delusions and hallucinations (> visual)

PMH:
6 months: L sided arm and leg weakness, diagnosed with ADEM. MRI Hyperintensity T2, FAIR in R thalamo-capsular region and cervical spinal cord, medulla, and pons extending caudally to the T1 segment, without enhancement of the lesions. Treated w/ steroids and plasma exchange. 2 w later, she improved but not back to baseline

Meds: None

Fam Hx:
None

Soc Hx:
Denies alcohol, tobacco or illicit drugs.

Health-Related Behaviors:
None

Allergies:
None

Notable Labs & Imaging:
Hematology and chemistry: Nl
TFT, HIV, HbsAg, ANA, ANCA, HCV -ve
Imaging:
MRI brain: Patchy, irregular, confluent T2W/FLAIR hyperintensity involving both caudate nuclei, hippocampus, cerebral peduncles, both thalami, both temporal lobes, midbrain, pons, bilateral periventricular white matter, and corpus callosum. Few lesions show patchy restriction of diffusion. No significant enhancement was noted in the post-contrast study.
MRI spine: Multifocal patchy T2W/STIR hyperintensity in the entire spinal cord extending from cervical segments to the conus medullaris. Lesions predominantly involved the central cord with mild patchy peripheral involvement. There was no significant enhancement in the post-contrast study.
MRA was unrevealing for any regions of dilatation or stenosis in the intracranial vasculature.
CSF: 15 WBC (100% lymph), protein 61 (high), glucose nl, no oligoclonal bands.
CSF +ve Aquaporin- 4 Ig Ab (AQP4- IgG,) and Anti NMDAR encephalitis returned positive. Anti-MOG, ant LGl1 anti caspr2 and anti AMPAR and Anti GABA B -ve
Final Dx: Neuromyelitis optica w/ AQP4 ab with overlap anti-NMDAR encephalitis.
Tx: Improve with plasma exchange and IV Methylprednisolone. Discharged with prednisone, azathioprine, risperidone and melatonin. Recovered and went back to school.

Problem Representation: 16-yo woman presented with subacute episodic behavioral changes with no return to baseline along with lower-limb weakness, difficulty in voiding urine and constipation, and a history of ADEM 6 months ago.

Teaching Points (Sukriti): #EndNeurophobia
Investigating the Sx: Localisation x Time course
Time course in neurology **(3+2); Toxic/metabolic apply to all timeframes: Hyperacute** (instant): Trauma, vascular, seizure; **Acute**: Infection (bacterial, viral), inflammatory; **Subacute**: Infection, inflammatory, neoplasia; **Chronic**: Benign neoplasm, degenerative
Localising behavioural change: **Primary change** in personality (frontal lobe) vs **Neuropsychiatric** of neurological etiology, psychiatric etiology or systemic illness
Layering, Back pain, constipation: Spinal Cord -- intradural (intramedullary, extramedullary), extradural vs Frontal Lobe (loss of sphincter control)
Collecting Clues: Young age, subacute time course of Sx << Demyelinating disorder -- looking for RAPD (NMO vs MS vs Anti-MOG)
ADEM: Acute demyelinating pathology of central nervous system, frequently post-infectious → Self limited, MS, Anti MOG syndrome
Pertinent questions to ask
- Are the background and foreground true true and related (**Recrudescence:** Pre-existing lesion that decompensates in the presence of systemic illness/medication)
- Revisit how a diagnosis was made in the past before incorporating it into the PR as it may falsely alter how you frame a clinical syndrome

Transverse myelitis: Clinical **syndrome** w/ full thickness axial spinal cord involvement (Rem: Not all myelitis is transverse, not all transverse myelitis is AI!)
Longitudinally extensive myelitis: affecting **>3 levels** of spinal cord segments
Myelitis (NMO, Sarcoid), B12 deficiency, dural AV fistulas, radiation induced myelopathy, Inf (mycoplasma)
- NMO/Devic's disease: Aquaporin-4 receptor ab; classically, lesions in **Area postrema**: refractory hiccups, coughing and vomiting

Hyperesthesia: Small fiber neuropathy (normal reflexes, N pain/temp) vs Dejerine Roussy syndrome
Framing the hypothesis**: Take something out and see how the rest of it fits in **-- young woman + rapidly evolving neuropsychiatric changes + autonomic dysfunction: Limbic system = >> NMDA receptor encephalitis** (pred grey matter), **herpes encephalitis