Outline

• Case presentation (Dr. Marshall)
• Psychiatry review of case (Dr. Hilde)
• Neurology overview of autoimmune encephalitis (Dr. Christy)
Case presentation: “M,” a 15 year-old female

**ID:** 15 year-old female with recent diagnosis of psychogenic non-epileptic seizures brought to the OHSU ED by family with new-onset aggressive behavior.
History of Present Illness:

- Sudden unprovoked episode of shouting, kicking, hitting, biting family. Two similar episodes in past month.
- Past 2 months: more isolative, blunted affect, disengaged. Patient denied depression or anxiety.
- Patient awake. No rhythmic jerking, head or eye deviation; prodrome; fevers, nausea/ vomiting, pain, SOB, weakness, numbness, headache.
HPI, continued:

• 7 weeks ago: Convulsive episode of right eye deviation, head shaking lasting 5-10s. Taken to outside ED.
• 6 weeks ago: Recurrent episodes, return to ED
• Normal routine EEG and MRI
• Referred to outpatient neurologist - ? Secondarily generalized epilepsy
• Started on levetiracetam, oxcarbazepine
• 2 weeks prior to presentation: Video EEG
  – EEG w frontal slowing; did not correspond w recorded events
  – Dx psychogenic non-epileptic seizures (PNES)
  – Referred to psychiatry
Initial ED Evaluation

- **PMH:** PNES
- **Meds:** Levetiracetam, Oxcarbazepine
- **Allergies:** NKDA
- **Social Hx:**
  - Lives at home with parents and four younger siblings.
  - Last year, school difficulties. New school in fall but not attending since episodes began.
  - No drug use.
  - Sexual abuse by uncle at 8 y.o.; told teacher in spring
- **Developmental Hx:** Unremarkable
- **Family Hx:** Grandmother with schizophrenia
Initial ED Evaluation

Vitals:
BP 118/67, T 36.8 °C, P 102, Resp 16, SpO2 100 %

Physical Exam:
Unremarkable. Patient described as having “anxious” mood; “occasionally inappropriately giggles describing the events earlier tonight”

Labs:
CMP, TSH, UDS, CBC unremarkable
ED Course

• Psychiatry consult:
  – MSE: “superficial affect;” TP somewhat tangential; oriented; intact concentration.
  – Assessment: Conversion disorder; concern for PTSD vs thought disorder
  – Rec: Intensive community mental health services
• “Stress seizures” → Escalating agitation
• Minimal response to olanzapine, lorazepam, and diphenhydramine
• Placed in four point restraints
• One measured fever of 101.4.
• CK 8052 → 9179
• Mental status notable for being disoriented, actively hallucinating.
• Dx delirium, concern for NMS vs other?
• Abnormal wide-based gait with high-frequency tremor observed
  – Additional history identified regarding other episode of abnormal gait
• Neurology consulted
**HPI:** Possible discrepancy between early episodes and those on VEEG

**Exam:**
- **Mental Status Exam:**
  - Oriented to person and date, not place (somewhere by her house and McDonalds)
  - Attention/Concentration: 1/5 correct on serial 7's
  - Speech fluent, difficulty naming hammock and cactus
  - Memory: 0/3 words at 5 minutes
- **Motor:** Normal tone and strength. Diffuse high frequency tremor
- **Cerebellar:** Finger-to-nose with mild end point high frequency tremor, unable to follow commands for heel-shin
- **Gait:** wide-based, entire body with high frequency tremor
Impression: Acute onset agitation concerning for autoimmune encephalitis

Recommendations:

• Admit
• Full brain MRI wwo
• LP, serum to Mayo Clinic
• Discontinue AEDs
Labs on admission

Abnormal

• CSF positive for oligoclonal bands (3)
• Influenza A H3 positive
• Sed rate 29

Normal

• Immunology panel
• Vitamins B6 and B12
• CRP
• Ammonia
• Infectious panel (with exception of influenza A)
• Copper and ceruloplasmin
• CSF – white count (5) and protein (34)
• **EEG**: frequent slowing of the awake background consistent with at least mild degree of encephalopathy

• **Pelvic ultrasound**: unremarkable

• **MRI brain w/wo contrast**: Isolated T2 hyperintense diffusion restricting lesion involving the splenium of the corpus callosum.
Hospital Course

• Mayo Clinic panel for autoimmune encephalitis pending 7-10 days
• Continued insomnia, confusion, hallucinations, nonsensical answers
• Adult neuroimmunology consult HD#6
  – Concern for Mild Encephalopathy with Reversible Splenial Lesion (MERS)
• Decision not to begin immunosuppressives
Hospital Course—Day # 9

• Rapid response called for 30-40 second sudden onset tremulous movement and inability to control oral secretions
• Incontinent, pulling out hair, incoherent screaming.
• Transferred to PICU.
• Agitation continued, not improved with ziprasidone and midazolam.
• Quick brain MRI – increased lesion in splenium of corpus callosum
Hospital Course – PICU

• Decision to treat for inflammation with steroids and IVIG
• IVIG / steroids → more oriented, still intermittent agitation
• Started valproate for agitation and trazodone for sleep
First day of steroids: “Write your name and draw a person.”
Third day of steroids: “Write your name and draw a person.”
Hospital Course

• Mayo panel: anti-NMDA receptor antibodies
• IVIG / steroids completed
• Worsening concentration, orientation, insomnia, convulsive episode
• IV solumedrol burst, started on mycophenolate, oral prednisone
• Sleep, orientation improved, no more agitation
• HD #28: Discharged to inpatient rehab
Follow-up

• Inpatient rehab for 2 weeks – improved physical function and mental status
• Continued on oral immunosuppressives
• Continued on valproate and trazodone
• Followed up with outpatient neurology
Psychiatric Perspective
Epidemiology

- Anti NMDA receptor encephalitis first identified in 2007
- 75% seen by psychiatrist or admitted to psychiatric unit
  - Often misdiagnosed
  - Case report: 27 yo F who spent 3 months in psychiatric hospital prior to re-evaluation and diagnosis (Fisher et al 2017)
Epidemiology

• More common than other paraneoplastic or autoimmune encephalopathies
  – 4% of encephalopathies - based on data from UK (Wingfield et al 2011)
• Children account for 40% of those diagnosed
• Most often affects young women
Epidemiology

  - 314 cases of encephalitis ≤18 years
  - Anti NMDAR encephalitis 24 cases
    - Exceeded all viral etiologies when considered individually
  - Females 2x as likely to be affected
  - Ethnicity: 54% Hispanic / 4% Caucasian / 13% African American, Pacific islander, and Asian
Epidemiology

- 66% presented with psychotic symptoms
  - 92% auditory hallucinations
- 80% agitated
- 60% with personality changes
- 21% admitted to psychiatric unit
  - Seizure activity most often led to additional evaluation
- Those 12 to 18 more likely to present with psychotic symptoms (68% vs 32%)
- 80% with autonomic instability
- **Psychiatric symptoms occurred in all even if not present at onset**
Common Presenting Symptoms

- 70% have viral prodrome
- 60-80% of cases present first with psychiatric symptoms
  - psychomotor agitation, aggressive behavior, impulsivity, mood swings, delusions, paranoia, hallucinations, short term memory loss, personality change
  - Can mimic early onset of schizophrenia, mania or autism
- Neurological signs may or may not be present on initial presentation
  - speech problems, memory and attention deficits, movement disorder, seizures, sleep disturbance, mutism

Pollak et al, Maccaferri et al and Bost et al
Diagnostic Challenges

• Early in course primarily psychotic or mood/anxiety symptoms
• Difficult to distinguish from primary psychiatric disorder
• Progression – can have rapidly changing presentation
  – Viral prodrome > psychiatric and behavioral sx$s$ > neurologic symptoms > autonomic instability
• Early detection important as this impacts outcomes
Initial Differential Diagnosis

- Conversion Disorder *
- Anxiety/PTSD
- Primary psychotic disorder
- Depression w psychotic features
- Catatonia
- Encephalopathy **
  - NMS
Conversion Disorder

- Neurological sx involving motor or sensory function
- Cannot be explained by neurologic or medical condition
  - Must definitively rule out medical d/o
- Associated with psychological conflict or stress
- Common symptoms:
  - blindness, paralysis, mutism, dystonia, PNES, anesthesia, swallowing difficulties, motor tics, difficulty walking, hallucinations
- Factors to consider:
  - Onset of symptoms - acute
  - Recent stressors – linked to sx onset
  - Presence of dissociate disorder (50% will have dissociative features)
  - Lack of concern by patient
  - Absence of injury, falls, bladder control loss

Nicholson et al and Ali et al
Conversion Disorder

Evidence for:  

Evidence against:
Conversion Disorder

Evidence for:

- H/o trauma
- Diagnosed with PNES
- Dissociative symptoms?
- At times appeared to be indifferent to situation
- Negative initial medical w/u

Evidence against:

- Stressor was not recent or correlated with symptom onset
- Fall with walking
- Serious self injury
- Behavior change
- Waxing and waning
- Memory problems
Anxiety/PTSD

• Evidence for:

• Evidence against:
Anxiety/PTSD

• **Evidence for:**
  – Endorsed stress and seizures related to stress
  – History of abuse
  – Social isolation
  – Dysregulated

• **Evidence against:**
  – Denied PTSD symptoms
  – Denied GAD symptoms
  – Did not appear anxious
Primary Thought Disorder

• Evidence for:  

• Evidence against:
Primary Thought Disorder

• Evidence for:
  – Recent presentation of negative symptoms
  – Cognitive slowing
  – Aggressive agitation
  – FH
  – Neg initial medical workup

• Evidence against:
  – Following outbursts returns to “baseline”
  – No decrease in aggression/agitation with antipsychotic medication
  – Does not endorse depression or paranoia
Psychosis

- Many underlying causes
  - Psychiatric: schizophrenia, bipolar, depression, OCD, PTSD, ASD)
  - Medical: delirium, dementia, infection, endocrine, autoimmune, malignancy, substances/toxin

- Fairly common in pediatric populations and can be transitory
  - Simon et al found that in teens presenting with hallucinations after one year, 50% had no symptoms and many had reduced sx$s
Psychosis

• Future considerations
  – Link between schizophrenia and NMDA-R antibodies
  – Steiner et al: Case control study (230 controls, cases - 121 schizophrenia, 70 MDD, 38 Borderline PD)
    • NMDA-R antibodies identified in 15 subjects
      – 9.9 % Schizophrenia diagnosis
      – 2.8% MDD
      – 0 % BPD
      – 0.4% Controls
Diagnostically Challenging

• Did not fit into diagnostic categories
• Need for more extensive evaluation
  – Waxing and waning level of consciousness
  – Visual hallucinations
  – Memory deficits
  – Tremor
  – Wide based gait
  – Insomnia
  – Bizarre behavior change
Treatment Considerations

- Neurological sx's often improve acutely; may be little to no impact on psychotic symptoms
- Agitation can be severe (93% of pediatric cases)
- First and second generation antipsychotics
  - Pts can develop fever, rigidity, autonomic instability and movement d/o in absence of antipsychotics
  - 50% of pts treated with neuroleptics develop symptoms consistent with NMS and or EPS
    - Hard to distinguish if NMS, EPS or disease progression
    - Can exacerbate symptoms

Maccaferri et al, Bost et al, Mohommad et al, Schumacher et al and Haung et al
Treatment Recommendations

• Agitation
  – Antipsychotics: Olanzapine and quetiapine
    • Use those less likely to cause NMS and EPS
      – Low binding profile for D2
      – Higher affinity for H1 (ie sedating effects)
      – Quetiapine: SGA least likely to cause EPS (Schumacher et al 2016)
  – Mood stabilizers: Depakote and Lithium
    • Limited data; may help w mood and impulsiveness
Treatment Recommendations

– Benzodiazepines: not particularly helpful
  • High risk of paradoxical agitation particularly with pediatric population

– Diphenhydramine: not particularly helpful
  • Anticholinergic effects can worsen delirium

• Insomnia:
  – Depakote, Trazodone, and Quetiapine
Autoimmune encephalitis: Keep it in mind

• Diagnostic clues
  – Symptom changes and progression
  – Fever prodrome followed by psychiatric sx
  – Atypical psychiatric presentation
  – Hallucinations - visual more common than auditory
  – Personality changes
  – Impulsive behavior that may require restraint
  – Young women with first psychiatric episode
  – Memory deficits (80%), often short term
  – Seizure (87%)
  – Movement disorders
Autoimmune Encephalitis
History of autoimmune encephalitis

- 1968: Paraneoplastic limbic encephalitis

“Limbic Encephalitis” and Its Association with Carcinoma

By

J. A. N. Corsellis, G. J. Goldberg, and A. R. Norton

Department of Neuropathology, Runwell Hospital, Wickford, Essex and the Department of Neuropathology, Institute of Psychiatry, London

There are several kinds of neurological disorder which may develop in patients with carcinoma, even though no manifest spread of tumour cells to the nervous system has occurred. In many of these patients
History of autoimmune encephalitis

• Anti-neuronal antibodies
  – Sydenham’s chorea
  – Neuropsychiatric lupus
  – anti-Hu and other onco-neuronal antibodies against intracellular proteins
1991: Hashimoto’s encephalopathy

Hashimoto’s encephalopathy:  
A steroid-responsive disorder associated with high anti-thyroid antibody titers—report of 5 cases

P.J. Shaw, MD; T.J. Walls, MD; P.K. Newman, MB, ChB; P.G. Cleland, MB, BChir; N.E.F. Cartlidge, MB, BS

- Coma, seizures, stroke-like episodes, movement disorders
- High titers of antithyroid antibodies (anti-TPO and anti-TG)
- Very steroid-responsive
History of autoimmune encephalitis

• 2005: 4 young women with teratoma and encephalopathy

Paraneoplastic encephalitis, psychiatric symptoms, and hypoventilation in ovarian teratoma

Roberta Vitaliani MD, Warren Mason MD, Beau Ances MD, PhD, Theodore Zwerdling MD, Zhilong Jiang PhD, Josep Dalmau MD, PhD

First published: 21 September 2005  Full publication history
• Seizures, psychosis, abnormal movements, autonomic symptoms, hypoventilation
• 3 recovered after surgery and immunosuppression
• 1 died
Antibodies to cell-surface antigens

NMDA (extracellular)

Hu (intracellular)
NMDA receptor encephalitis and other antibody-mediated disorders of the synapse

Josep Dalmau, MD, PhD

Neurology December 6, 2016 vol. 87 no. 23 2471-2482
Patients’ antibodies bind, crosslink and internalize NMDA receptors.

Normal | Abs to NMDA receptors | Internalization of NMDA receptors

Cellular and synaptic mechanisms of anti-NMDA receptor encephalitis.
Hughes, et al. *Journal of Neuroscience*, 2010
• Movement
• Memory
• Psychosis
Autoimmune encephalitis
Autoimmune encephalitis

• Can be devastating and have high morbidity
• Can be treatable
• We believe that treating early can affect outcomes
• Paraneoplastic panels take a long time to result
Encephalopathy Autoimmune Evaluation Algorithm, Spinal Fluid

**ENCEC / Encephalopathy, Autoimmune Evaluation, Spinal Fluid**

The following tests are always performed:

- Radioimmunoprecipitation Assay (RIA)
  - Glutamic Acid Decarboxylase (GAD65) Antibody Assay, Spinal Fluid
  - Neuronal Voltage-Gated Potassium Channel-Complex (VGKC) Autoantibody, Spinal Fluid

- Immunofluorescence Assay (tissue IFA)
  - Antineuronal Nuclear Antibody-Type 1 (ANNA-1), Spinal Fluid
  - Antineuronal Nuclear Antibody-Type 2 (ANNA-2), Spinal Fluid
  - Antineuronal Nuclear Antibody-Type 3 (ANNA-3), Spinal Fluid
  - Purkinje Cell Cytoplasmic Antibody, Type 1 (PCA-1), Spinal Fluid
  - Purkinje Cell Cytoplasmic Antibody, Type 2 (PCA-2), Spinal Fluid
  - Purkinje Cell Cytoplasmic Antibody, Type Tr (PCA-Tr), Spinal Fluid
  - Amphiphysin Antibody Assay, Spinal Fluid
  - collapsin Response-Mediator Protein-5 Neuronal (CRMP-5-IgG), Spinal Fluid
  - Anti-Gliial / Neuronal Nuclear Antibody-Type 1 (AGNA-1), Spinal Fluid

- Immunofluorescence Assay (cell binding; CBA)
  - NMDA-Receptor Antibody by CBA, Spinal Fluid
  - AMPA-Receptor Antibody by CBA, Spinal Fluid
  - GABA-B-Receptor Antibody by CBA, Spinal Fluid

**Decision Tree**

- If IFA suggests ANNA-1, ANNA-2, PCA-1, PCA-2, or CRMP-5 IgG or if IFA pattern is indeterminate:
  - Paraneoplastic Autoantibody, Western Blot Confirmation, Spinal Fluid

- If IFA pattern suggests CRMP-5 IgG:
  - collapsin Response-Mediator Protein-5 (CRMP-5-IgG) Western Blot, Spinal Fluid

- If IFA pattern suggests Amphiphysin Antibody:
  - Amphiphysin Antibody Western Blot, Spinal Fluid

- If IFA pattern suggests NMO / Aquaporin-4-IgG:
  - Neuromyelitis Optica (NMO)/ Aquaporin-4-IgG Cell-Binding Assay, CSF

- If pattern suggests AMPA-Receptor Antibody and AMPA-Receptor Antibody, CBA is positive:
  - AMPA-Receptor Antibody IF titer assay

- If pattern suggests NMDA-Receptor Antibody and NMDA-Receptor Antibody, CBA is positive:
  - NMDA-Receptor Antibody IF titer assay

- If pattern suggests GABA-B-Receptor Antibody and GABA-B-Receptor Antibody, CBA is positive:
  - GABA-B-Receptor Antibody IF titer assay
Mayo Clinic: serum panel

For NMDA: CSF is more sensitive
LAB OTHER: Mayo Clinic Autoimmune Encephalopathy Panel (Test ID: ENCES)

Priority: Routine, Routine, Urgent

Frequency: ONCE, Collect Now, X1, Tomorrow AM, Next Draw, X1

Starting: 1/23/2017, Today, Tomorrow, At: 1115

First Occurrence: Today 1115
Scheduled Times: Hide Schedule
1/23/17 1115

Comments (F6): Click to add text
Specimen Src: Cerebrospir

Specimen Type: Cerebrospir

Test Name: Mayo Clinic Autoimmune Encephalopathy Panel

Test ID: ENCES

Additional Test Information:
How do we diagnose a patient before the labs come back?
Panel 1: Diagnostic criteria for possible autoimmune encephalitis

Diagnosis can be made when all three of the following criteria have been met:

1. Subacute onset (rapid progression of less than 3 months) of working memory deficits (short-term memory loss), altered mental status*, or psychiatric symptoms
2. At least one of the following:
   - New focal CNS findings
   - Seizures not explained by a previously known seizure disorder
   - CSF pleocytosis (white blood cell count of more than five cells per mm$^3$)
   - MRI features suggestive of encephalitis†
3. Reasonable exclusion of alternative causes (appendix)

*Altered mental status defined as decreased or altered level of consciousness, lethargy, or personality change. †Brain MRI hyperintense signal on T2-weighted fluid-attenuated inversion recovery sequences highly restricted to one or both medial temporal lobes (limbic encephalitis), or in multifocal areas involving grey matter, white matter, or both compatible with demyelination or inflammation.
Panel 4: Diagnostic criteria for anti-NMDA receptor encephalitis

Probable anti-NMDA receptor encephalitis*
Diagnosis can be made when all three of the following criteria have been met:

1. Rapid onset (less than 3 months) of at least four of the six following major groups of symptoms:
   - Abnormal (psychiatric) behaviour or cognitive dysfunction
   - Speech dysfunction (pressured speech, verbal reduction, mutism)
   - Seizures
   - Movement disorder, dyskinesias, or rigidity/abnormal postures
   - Decreased level of consciousness
   - Autonomic dysfunction or central hypoventilation

2. At least one of the following laboratory study results:
   - Abnormal EEG (focal or diffuse slow or disorganised activity, epileptic activity, or extreme delta brush)
   - CSF with pleocytosis or oligoclonal bands

3. Reasonable exclusion of other disorders (appendix)

Diagnosis can also be made in the presence of three of the above groups of symptoms accompanied by a systemic teratoma

Definite anti-NMDA receptor encephalitis*
Diagnosis can be made in the presence of one or more of the six major groups of symptoms and IgG anti-GluN1 antibodies, † after reasonable exclusion of other disorders (appendix)

*Patients with a history of herpes simplex virus encephalitis in the previous weeks might have relapsing immune-mediated neurological symptoms (post-herpes simplex virus encephalitis). †Antibody testing should include testing of CSF. If only serum is available, confirmatory tests should be included (e.g., live neurons or tissue immunohistochemistry, in addition to cell-based assay).
Anti-NMDAR encephalitis: gender and tumor association in 577 patients

Titulaer et al., Lancet Neurol 2013;12:157-65
Panel 7: Criteria for autoantibody-negative but probable autoimmune encephalitis

Diagnosis can be made when all four of the following criteria have been met:
1. Rapid progression (less than 3 months) of working memory deficits (short-term memory loss), altered mental status, or psychiatric symptoms
2. Exclusion of well-defined syndromes of autoimmune encephalitis (e.g., typical limbic encephalitis, Bickerstaff's brainstem encephalitis, acute disseminated encephalomyelitis)
3. Absence of well-characterised autoantibodies in serum and CSF, and at least two of the following criteria:
   - MRI abnormalities suggestive of autoimmune encephalitis*
   - CSF pleocytosis, CSF-specific oligoclonal bands or elevated CSF IgG index, or both*
   - Brain biopsy showing inflammatory infiltrates and excluding other disorders (e.g., tumour)
4. Reasonable exclusion of alternative causes

*Some inherited mitochondrial and metabolic disorders can present with symmetric or asymmetric MRI abnormalities and CSF inflammatory changes resembling an acquired autoimmune disorder.107
From Josep Dalmau’s presentation at the American Academy of Neurology annual conference, April 2016
Different providers treat differently

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<th>First line treatment with immunotherapy</th>
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<td>IVIG</td>
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<th>Length of immunotherapy with first line treatment if return to baseline</th>
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<th>Time of initiation of second line disease-modifying therapy</th>
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<td>At diagnosis</td>
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<th>Factors influencing use of a disease-modifying agent?</th>
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<th>First disease-modifying agent if failure of immunotherapy</th>
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**Anti-\(\text{N}-\text{Methyl-d-Aspartate (NMDA)}\)**
**Receptor Encephalitis: A Survey of Treatment Progress and Prospects From Pediatric Neurologists**

Ilana Kahn, MD\(^1\), Guy Helman, BS\(^{1,2}\), Adeline Vanderver, MD\(^{1,2}\), and Elizabeth Wells, MD\(^1\)
Take home points

- If acute onset of seizures, psychosis, and movement disorders, consider autoimmune encephalitis
- Send autoimmune encephalopathy panel (CSF) or paraneoplastic panel (serum) to Mayo
- Consider possible teratoma
- We recommend involving a multi-disciplinary team for these very complicated patients
Thank you!

- Pediatric Neurology
  - Alison Christy
  - Juan Piantino
  - Jae Cho
  - Ittai Bushlin
  - Tracy Bazan

- Child & Adolescent Psychiatry
  - Rebecca Marshall
  - Anandam Hilde
  - Jennifer Chaffin
  - John Sheridan
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