Are They Out of Their Minds?
Pediatric Case Studies Distinguishing Encephalitis, Encephalopathy and Delirium
Session #322
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Speaker Introduction
• Jamie Tumulty has been practicing as a PNP in the PICU at University of Maryland Medical Center since 2002. She is author of several book chapters on various topics. She has presented at local and national conferences including NTI and NAPNAP. She has participated in several research studies including two national multicenter studies and was the recipient of AACN funded research grant.
• Dr. Shari Simone is the NP clinical program manager for Women and Children’s Services at the University of Maryland Medical Center. She is also an NP in the PICU and professor at the University of Maryland School of Nursing. Dr. Simone is a frequent national speaker and accomplished author including presentations and publications on pediatric delirium.

Disclosures
We have nothing to disclose

Learning Objectives
• Distinguish between delirium, encephalitis and encephalopathy in the pediatric patient
• Describe evidence-based pharmacologic and other support therapies for the pediatric patient with encephalitis
• Describe evidence-based pharmacologic and other support therapies for the pediatric patient with encephalopathy
• Describe strategies to prevent or minimize delirium in the pediatric patient

Case 1
6 yo male presents with acute AMS and right-sided neurological deficits
- 2-day history of not using right hand
- Decreased activity
- Limp (right side)
- Right eye squint
- Slurred speech

Do you think this patient has encephalopathy or encephalitis?

PMH: previously healthy
HPI:
- Hit his head at school 1 day prior to symptom onset
- No LOC or behavioral changes
- 1-2 weeks of nasal congestion and rhinorrhea
- No seizure, fever, diarrhea, ill contacts, drug exposure, recent travel, tick bites, no signs of trauma
**Definitions**

- **Encephalopathy** – Syndrome of global brain dysfunction associated with alteration in mental status with wide differential (Davies, 2012)
  - May or may not include inflammation
- **Encephalitis** – Condition of neurologic dysfunction due to inflammation of the brain parenchyma. (Messacar, 2018)
- **Delirium** – abrupt change in brain causing mental confusion and emotional disruption (Badii, 2018)

**Etiologies of Encephalopathy**

- Infectious (brain infection, systemic infection)
- Autoimmune (ADEM, anti-NMDA, Hashimoto, Bickerstaff)
- Trauma
- Seizure related
- Toxins (recreational, therapeutic, poisoning)
- Metabolic (IEM, hepatic, renal)
- Hypertensive
- Hypoxic/ischemic
- Hemorrhagic
- Oncologic

**Common Infectious Etiologies of Encephalitis**

<table>
<thead>
<tr>
<th>Virus</th>
<th>Bacteria</th>
<th>Fungi</th>
<th>Protozoa</th>
</tr>
</thead>
<tbody>
<tr>
<td>HSV</td>
<td>Listeria monocytogenes</td>
<td>Cryptococcal neoformans</td>
<td>Naegleria fowleri</td>
</tr>
<tr>
<td>MHH</td>
<td>Mycoplasma pneumoniae</td>
<td>Blastomyces dermatitidis</td>
<td>Balamuthia mandrillaris</td>
</tr>
<tr>
<td>CMV</td>
<td>Chlamydia pneumoniae</td>
<td>Histoplasma capsulatum</td>
<td>Baylisascaris procyonis</td>
</tr>
<tr>
<td>Varicella</td>
<td>Enterovirus</td>
<td>Paracoccidioides brasiliensis</td>
<td>Toxocara canis</td>
</tr>
<tr>
<td>CMV</td>
<td>Enterovirus</td>
<td>Paracoccidioides brasiliensis</td>
<td>Angiostrongylus cantonensis</td>
</tr>
</tbody>
</table>

**Encephalitis**

2013 International Encephalitis Consortium definition

- Major criteria: Altered mental status >24 hours (required)
- Minor criteria: 2=possible, 3=probable

**Delirium – DSM 5 Definition (APA, 2013)**

1. Disturbed Consciousness
   - Alertness/orientation
   - Attention span
   - Focus
2. Change in Cognition
   - Memory deficit
   - Language disturbance
   - Visuospatial disturbance
   - Perceptual disturbances/ Hallucinations
3. Acute Onset, Fluctuating Course
   - Develops in hours-days
   - Severity waxes & wanes
   - Often worse at night
4. Triggered by Serious Condition
   - Medical illness
   - Substance (intoxication or withdrawal)
   - Toxin
Acute Encephalitis Diagnostic Evaluation

- Evaluate for infection
- Evaluate for structural abnormalities (imaging)
- Evaluate for toxins or toxic metabolites
- Lumbar puncture
- Neurology consult
- Evaluate for immune mediated process
- EEG

Case 1: Diagnostic Evaluation

<table>
<thead>
<tr>
<th>9.8</th>
<th>11.5</th>
<th>319</th>
<th>35.5</th>
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<tbody>
<tr>
<td>139</td>
<td>101</td>
<td>11</td>
<td>115</td>
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<tr>
<td>15.3</td>
<td>27</td>
<td>3.9</td>
<td>26.0</td>
</tr>
<tr>
<td>0.34</td>
<td>37</td>
<td>8.0</td>
<td>225</td>
</tr>
<tr>
<td>0.9</td>
<td>5.1</td>
<td>0.9</td>
<td>5.1</td>
</tr>
</tbody>
</table>

- Toxicology screen: negative
- Resp viral panel: positive (rhino/enterovirus)
- What additional studies or consults would you obtain?

Case 1: Neurology Consult

- No edema, tenderness, deformity, or signs of trauma; normal muscle tone
- Alert, agitated, disoriented, non-verbal
- Intermitently will follow simple commands
- Normal strength and normal reflexes, no tremor
- Coordination and gait normal, walks without assistance
- Cranial nerves normal

Differential diagnosis:
- ADEM
- Viral encephalitis (less likely)
- Unlikely to be AFM (due to normal strength)

Recommendations:
- MRI
- LP
- Start high dose Methylprednisone
- ID consult

Acute Disseminated Encephalomyelitis (ADEM)

- Neuroinflammatory demyelinating disease of CNS
- Acute onset, single flare up
- Seasonal
  - Winter and Spring
- Symptoms worsen over time, peak at ~4 days
  - Rapid development of high fever
- Autoimmune mechanism
  - 60% associated with MOG antibodies
- Incidence:
  - 1 in every 125,000 - 250,000 individuals/yr
  - Children > adults
  - Boys and girls affected nearly evenly

Pohl (2012), Armangue (2016)

ADEM Diagnostic Criteria

- First episode of inflammatory/demyelinating disease in the CNS
- Acute onset
- Multiple areas of CNS affected/polysymptomatic
- Must include encephalopathy:
  - Acute behavioral change (confusion/irritability) and/or
  - Alteration in consciousness (somnolence/coma)
- Followed by clinical or radiographic improvement
- No other etiologies can explain the event

Pohl (2016)

ADEM Treatment Algorithm

- Some institutions may favor plasma exchange first so as not to remove IVIG

Bensim, Olsen & Gorman, 2017
Case 1: Imaging Results

- Normal
- Normal

Case 1: Acute Behavior Deterioration

- No longer follows commands
- Thrashing, kicking and screaming
- Harmful to self and others, placed in posey bed, sitter utilized
- Psych consulted for behavior management
- Diagnosed with delirium

Delirium Defined as:

- Disturbance of consciousness
- Change in cognition
- Acute onset with fluctuating course
- Triggered by serious condition
- Not explained by pre-existing neurocognitive disorder or coma

Delirium – Clinical Subtypes

- Hyperactive
  - Psychomotor restlessness, agitation, hallucinations
- Hypoactive
  - Diminished responsiveness, decreased movement, apathy
- Mixed
  - Alternating symptoms of both

(Peterson et al, 2006; Smith et al 2009)

Delirium Assessment, Prevention, Treatment Algorithm

- Pain
  - Evaluate for pain
  - Consoling and soothing
  - Provide delirium education
  - Monitor sedation
  - Establish care team – Physician, nurse, family, social worker, dietitian, etc.

High Risk Prevention Measures

- Nonpharmacologic
  - Treatable medical conditions
  - Sleep-wake cycle disruption
  - Sedation
  - Pain

Other Prevention Measures

- Preventable medical conditions
  - Infections, sepsis
  - Retained foreign bodies
  - Hypo/hypernatremia
  - Hypo/hyperkalemia
  - Hypoglycemia
  - Hypocalcemia
  - Alkalosis, acidosis
  - Underweight

Differential Diagnoses

<table>
<thead>
<tr>
<th>Clinical Disorder</th>
<th>Considerations</th>
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</thead>
<tbody>
<tr>
<td>Delirium</td>
<td>Likeliest diagnosis for delirium, CPT falls prevalence, absence of other definable cause</td>
</tr>
<tr>
<td>Acute alcoholic delirium</td>
<td>Evidence of alcohol use, delirium typically resolves with alcohol withdrawal</td>
</tr>
<tr>
<td>Lytic</td>
<td>Seizure activity, not usually associated with profound neurocognitive deficit, negative P2R</td>
</tr>
<tr>
<td>Hypothyroid</td>
<td>Lack of findings on imaging and negative P2R</td>
</tr>
<tr>
<td>Other post-infectious cause (CNS or myoclonus)</td>
<td>Negative P2R</td>
</tr>
</tbody>
</table>

Acute delirium – hyperactive

- Delirium with sudden onset, rapidly progresses, delirium typically resolves with alcohol withdrawal

Acute delirium – hypoactive

- Delirium with sudden onset, rapidly progresses, delirium typically resolves with alcohol withdrawal

Acute delirium – mixed

- Delirium with sudden onset, rapidly progresses, delirium typically resolves with alcohol withdrawal

Acute delirium – hypoactive

- Delirium with sudden onset, rapidly progresses, delirium typically resolves with alcohol withdrawal

Acute delirium – mixed

- Delirium with sudden onset, rapidly progresses, delirium typically resolves with alcohol withdrawal
Antipsychotic Therapy

- Serious side effects
  - Prolonged QTC
  - Ventricular arrhythmias
  - Extrapyramidal symptoms
  - Seizures
  - Neuroleptic malignant syndrome
- Milder side effects
  - Sedation
  - Agitation
  - Orthostatic hypotension
  - Anticholinergic symptoms

Not FDA approved for use in children

Kishk, (2019)

Monitoring

Anti-NMDA Receptor Encephalitis

- Most common autoimmune encephalitis in children
- Antecedent viral prodrome
- Psychiatric symptoms
  - bizarre behavior, agitation, temper tantrum, aggression
- Motor dysfunction
  - dyskinesia, choreoathetoid movements, seizures
- Autonomic instability
  - tachycardia, hypertension, hyperthermia, incontinence
- 59% associated with ovarian tumor (primarily teratoma)

Aramangue (2013)

Anti NMDAR Encephalitis Diagnosis

- CSF shows lymphocytic pleocytosis
- Anti NMDA receptor Ab in serum or CSF
- MRI
  - often normal in children (or may be related to timing)
  - may show cortical, subcortical or meningeal enhancement on T2 FLAIR sequences
- EEG
  - diffuse slowing
  - 60% have seizure
  - 30% have extreme delta brush - independent of sedation, dystonia, abnormal movement

Aramangue (2013)
### Positron Emission Tomography CT
- Shows structure and **function**
- PET abnormalities seen earlier
- Solves (2017):
  - Adult retrospective study (n=23)
  - Lobar hypometabolism (most common)
  - Parietal-occipital temporal
- Turpin (2018):
  - Pediatric retrospective study (n=34)
  - Basal ganglia hypermetabolism in 2/3
  - Imaging findings MRI 14/34 vs PET 32/34

**Delay in therapy results in worse outcomes**
**Delay in antibody assay results leads to initiation of therapy before diagnosis known.**

### Anti NMDAR Management
- **First line (fail in 30-40%)**
  - Steroids
  - Plasmapheresis
  - IVIG
- **Second line**
  - Rituximab
  - Cyclophosphamide (used only in refractory cases due to concern of infertility)
- Chronic immune suppression (efficacy unknown)
  - Azathioprine
  - Mycophenolate

### Case 2
- 17 yr with diffuse non B cell non Hodgkin lymphoma dx 3 mo ago
- Completed chemotherapy per ANHL1131 protocol (rituximab, methotrexate, cytarabine) 3 days ago
- Present to heme/onc clinic for final LP
- HA (worse when sitting) (since last LP 3 days ago)
- Photophobia
- Non-bloody non-bilious emesis
- Lethargy
- Chills
- Sick contacts (mother)

**What are your priorities in management? What is in the differential?**

- Admitted to floor and treated with Promethazine, Ondansetron, NS bolus, Hydromorphone, and Cefepime

### Case 2: Acute change in mental status
- Tingling in fingers quickly progressed to unresponsiveness, incontinence and right leg jerking.

**What are your priorities in management?**
- Lorazepam 0.1 mg/kg, CR monitor, oxygen

**What diagnostic studies would you order?**
- Head CT (normal, no bleed, mass, infarction, or midline shift)
- Neurology consult, EEG – no seizure
- LP normal, anti NMDA negative

### Case 2: Diagnosis and Management

#### Chemotherapy (methotrexate) Neurotoxicity
- Methotrexate blocks nucleic acid synthesis by inhibiting dihydrofolate reductase (reduce folate needed for purine synthesis)
- Associated with elevated CSF adenosine
- Increased risk
  - High dose methotrexate
  - Simultaneous IP methotrexate
  - Pt >10 yr
  - High MTX: folinic acid ratio
- Supportive Treatment
  - Stop methotrexate
  - Symptom management (AEDs, Gabapentin)
  - Steroids
  - Folinic acid
  - Aminophylline
  - Neuro rehab

**Our pt also received IVIG**

### Case 2: Acute change in mental status
- Case courtesy of Assoc Prof Frank Sokoloff, Radiopaedia.org, ID: 4438
Folinic Acid and Aminophylline

- **Folinic acid**
  - Active form of folic acid, allows nucleic acid synthesis
- **Aminophylline**
  - A methylxanthine
  - Acts as a competitive adenosine antagonist
- Case report 20 yr college student (Jaksic, 2004)
  - Folinic acid 2,500 mg IV over 24 h followed by taper over 6 weeks
  - Aminophylline 145 mg IV daily for 7 days
  - Gradual symptom resolution: MRI normal at 1 mo, return to college at 6 mo
- Case report 24 yr college student (Ganesan, 2014)
  - Dexamethasone and Folinic acid (doses not specified)
  - Aminophylline 100 mg IV q 6 x 5 days (Dc’d due to side effects/tachycardia)
  - Able to talk and eat by 72 h, able to ambulate by 1 mo
  - MRI at 1 mo improved diffusion, but residual deficits on T2

Comparison of Encephalitis, Encephalopathy, and Delirium

**Case 3**

- 6 yr female with cough congestion and headache x 5 days, afebrile
- Sick contacts at home
- Sent to the nurse’s office for vomiting
- Awake but not responding to school staff, confused
- Observed in ED, then discharged at neurologic baseline
- Developed headache \(\rightarrow\) vomited with ibuprofen
- Left arm shaking \(\rightarrow\) progressed to GTCS and loss of consciousness

What are your priorities in management?

Seizure stopped with lorazepam, loaded with fosphenytoin

**Case 3: Diagnostic Evaluation**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Reference</th>
<th>Albumin (mg/dL)</th>
<th>BUN/CREA (mg/dL)</th>
<th>WBC</th>
<th>Hct</th>
<th>Hgb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
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<tr>
<td>Followup</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Respiratory viral panel: adenovirus, coronavirus and RSV

**Case 3: Diagnosis & Management**

- ADEM initially favored in diagnosis as obvious antecedent viral infection RSV, coronavirus and adenovirus
- Steroids initiated methylprednisolone 500 mg bid x 5 day (~18 mg/kg)
- Acyclovir x 21 days
- Test of cure LP negative
- Fosphenytoin 10 mg/kg load then 75 mg bid (2.7 mg/kg)
- Keppra 500 mg bid
- Intubated for respiratory failure
- N acetyl cysteine
Case 3: Imaging Results

- Repeat MRI evolution of encephalitis

N Acetyl Cysteine

- Antidote in Acetaminophen toxicity, emerging as a potential encephalopathy therapy
- Mechanism of action: precursor to the antioxidant glutathione, modulates glutamatergic, neurotrophic, and inflammatory pathways
  - Free radical scavenger
  - Decreases inflammation
  - Provides cysteine moiety for production of glutathione
  - Facilitates production of nitric oxide thus causing vasodilatation (may have side effect of hypotension)
- Moss (2017) neonatal HIE study (n=30)
  - Hypothermia 33 C x 72 h → rewarmed 0.2 C/h x 16 h
  - NAC calcitriol 25-40 mg/kg q 12 on DOL 0-10
  - MR spec of glutathione on DOL 5-6 pre and post NAC dose
  - Pre 1.64 ± 0.20 mM
  - Post 1.90 ± 0.22 mM (p < 0.0001).
  - Healthy term neonates 2.5 ± 0.8

Case 4

- 8 yr male with Wilson's disease now POD # 11 s/p liver transplant
- Immunosuppression: prednisone, mycophenolate, tacrolimus
- Infection prophylaxis: sulfamethoxazole/trimethoprim, valganciclovir, fluconazole
- Develops HA, abdominal discomfort, tingling of extremities
- HR 165, BP 131/94, RR 28, 96%, temp 37.1
- Speech becomes incoherent, staring
- Lip twitching, R gaze deviation, left leg shaking, becomes verbally unresponsive
- Progresses to seizure, pupils unequal, right fixed 4 mm
- Repeat VS: HR 176, BP 157/66, RR 24, SpO2 96%, temp 36.7
- What are management priorities?
  - Stop seizure, stabilize ABCs (lorazepam administered, placed on NRB with 100% oxygen)

Case 4: Differential Diagnosis

- Venous sinus thrombosis
- Cerebral hemorrhage
  - subdural, intracerebral, or subarachnoid
- Ischemic stroke
  - Posterior circulation stroke, Basilar artery thrombosis
- Infective encephalitis or meningitis
- Autoimmune encephalitis
- Metabolic encephalopathies
  - Na or glucose derangements, uremia, or drug toxicity

Case 4: Diagnostic Evaluation

- CT: no mass, bleed or infarct
- MRI: signal abnormality in bilateral posterior parietal lobes consistent with PRES
- EEG
  - Focal slowing.
  - Excess beta activity – (meds like benzodiazepines or barbiturates)
  - Focal sharps
- 28.8 \[\text{10.8} \quad 419\] CRP 0.7
- Blood culture – negative
- 141 105 113 \[\text{223}\] Mg 1.5 ALT 302 bil 2.0 albumin 4.5
- Phos 4.1
- LP – not done in this pt

Hobson (2012)

Posterior Reversible Encephalopathy Syndrome

- Rapid onset of symptoms
  - Headache, seizures, altered consciousness, visual disturbance
  - Often associated with hypertension
  - Less common nausea, vomiting
- Strong association with
  - Renal disease, vascular disease, Autoimmune disease
  - Immunosuppressive drugs, organ transplantation
- Mechanism
  - Thought to be related to vasogenic edema and endothelial dysfunction

Management

- Supportive Care/ Symptom management
  - Rapid withdrawal of trigger or offending medication
  - Blood pressure management
  - Antiepileptics to control seizures
  - Airway protection (intubation/ventilation) in obtunded patients
  - No evidence for steroids in PRES
  - No clinical trials of PRES management

- Held tacrolimus and prednisone for 2 days – received 1 dose methylprednisolone
- Arterial line for BP monitoring
- Nicardipine gtt (goal SBP 90-120)
- Transitioned to amlodipine
- Levetiracetam
- Remained stable without intubation
- Repeat MRI demonstrated resolution at 1 week

Case 5

- 6 mo old male with bronchiolitis presents from an OSH in respiratory distress
  - HPI:
    - 4-day H/O of URI symptoms seen twice at OSH. DOA with increased WOB & fever to 38.5 C
    - Upon transfer was placed on HFNC at 15 L & 100% FiO2 & admitted to PICU
  - PICU:
    - CXR revealed mild cardiomegaly and bilateral perihilar opacities, & small pleural effusions. ECHO obtained & revealed pericardial effusion.
    - Pt was intubated & pericardiocentesis performed.
  - PMH:
    - FT, SVD infant
    - No previous hospitalizations, no medications, normal development
    - No significant social or family history

Case 5: Hospital Course

- Resp/CV:
  - Developed significant hypoxia & ↓ lung compliance requiring increased mechanical ventilation support & cardiac insufficiency requiring vasopressor/inotopic support
  - RD:
    - MSSA pericarditis & bacteremia, & bilateral effusions
  - Neuro:
    - On Fentanyl & Dexmedetomidine infusions
    - On HD # 4 develops intermittent, sudden episodes of agitation & restlessness, CAP-D scores 14-15
    - Tachycardic, tachypneic, dysynchronous with mechanical ventilation
    - Inconsolable despite nonpharmacologic measures
    - Minimal improvement in agitation with extra doses of analgesic/sedatives

Pathophysiology of Delirium

- Reduced Acetylcholine
  - Memory & attention
  - Motor & learning
  - Sleepwake cycle

- Excess in excitotoxic & inhibitory NTs
  - Dopamine & serotonin
  - Norepinephrine & ß-adrenergic
  - Glutamate & GABA
  - Serotonin & GABA

- IFN; TNF-α; IL-1, 2, 6 & 8; CRP , procalcitonin
- Stress response/cortisol
- 2nd Messenger systems
  - Blood Brain Barrier, free radicals

- Cognitive Sx
  - Agitation/irritability
  - Hallucinations

- Excitotoxic or oversedation

- ???

(Zaal & Slooter, 2012)

What is your diagnosis?
Risk Factors for Delirium
(Smith et al 2009)

Delirium Screening & Prevention

- Assessment: pain & sedation q 2, delirium q shift
- Prevention Strategies:
  - Multidisciplinary rounds discussion with parental participation
  - SAFE‐T Checklist
    - S: Sedation/Sleep/Analgesia/Delirium
    - A: Access, Assess lab frequency
    - F: Food/Family
    - E: Enteral meds/Early mobilization
    - T: Thromboembolism, Review Protocols, Family presence/needs
  - Nonpharmacologic strategies
    - Parental presence
    - Create familiar environment
    - Promote wake‐sleep cycle
    - Noise reduction
    - Minimize restraints
    - Early mobilization
    - Early PT/OT consults

Pharmacologic Treatment of Delirium

Hypoactive Delirium: Diminished responsiveness, decreased movement, apathy, difficult to engage, quiet confusion

Hyperactive Delirium: Agitation, hallucinations, pulling out lines & tubes, unsafe behavior

Mixed Delirium: Fluctuating symptoms of agitation, restlessness, hallucinations with decreased responsiveness, or movement or confusion

Consider ATC
- Risperidone (Risperdal)
  - < 10 kg: initial dose 0.1mg @ qhs‐q12hr
- Quetiapine (Seroquel)
  - 10‐15 kg: 6.25 mg q 12 hr
  - >15‐25 kg: 6.25 mg q 8hr
  - >25‐50 kg: 12.5 mg q 8 hr
  - > 50 kg: 25 mg q 8 hr
  - Max daily dose: 100 mg

Consider ATC 1st Quetiapine: hypoactive dosing based on weight
  - If need IV dosing: Consider PRN Chlorpromazine
    - 0.4 mg/kg IV q 6 hr prn, for max 72 hrs
    - Max daily dosing 50 mg

Case 5: Hospital Course

- Risperidone (0.1mg NG q pm) started D#6
- Baseline EKG QTc: 0.39sec
- QTc after tx initiated: 0.44sec
- Daily EKG
- Monitoring for side effects
- Intubated for 10 days
- Treated with vancomycin & gentamycin→ transitioned to nafcillin
- Received 28 days of antibiotic coverage for MSSA pericarditis & sepsis
- Sedation while mechanically ventilated
- Fentanyl & dexmedetomidine infusions
- Transitioned to methadone
- Weaned off prior to DC without withdrawal symptoms
- Risperidone weaned by 50% on HD #11 & Dc’d on HD14

Antipsychotic Therapy
Comparison of Encephalitis, Encephalopathy, and Delirium

<table>
<thead>
<tr>
<th>Encephalitis</th>
<th>Encephalopathy</th>
<th>Delirium</th>
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<tbody>
<tr>
<td>Definition</td>
<td>Acute neurologic dysfunction due to inflammation of the brain</td>
<td>Global brain dysfunction with AMS</td>
</tr>
<tr>
<td>Presentation</td>
<td>Acute onset of fever, NA, &amp; AMS</td>
<td>Personality/behavioral change</td>
</tr>
<tr>
<td>Diagnostic markers</td>
<td>Seizures, focal neuro deficits, or focal or systemic signs</td>
<td>CT scan, ERAS</td>
</tr>
<tr>
<td>General Management</td>
<td>Supportive care: i.e., control seizures, therapy for common treatable causes: i.e., amygdala</td>
<td>Prompt treatment of treatable causes: i.e., use of anticonvulsants, hyperosmolar encephalopathy: CRRT; i.e., treatment of treatable causes in the brain</td>
</tr>
</tbody>
</table>

Summary

- Overlap between clinical exam findings in encephalitis, encephalopathy, and delirium
- Major etiology categories include infection, autoimmune, toxic/metabolic
- Management:
  - Priority is to identify and manage emergencies (seizure, respiratory failure)
  - Targeted therapy based on diagnosis

References


Questions?

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