15 year old girl with altered mental status

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HPI

- 15 yo F with h/o Graves disease s/p subtotal thyroidectomy with recurrent hyperthyroidism admitted 6/10/13 with altered mental status
- Developed severe headaches unresponsive to NSAIDs after in MVA 2/2013
- TFTs 2/11/13: TSH 0.01, FT4 1.26, T3 168->MMI 2.5 to 5 daily
- Admitted to neurology service 3/3013 for Depakote load to break headaches
- Discharged on amitriptylline
HPI continued

• TFTs 5/3: TSH <0.01, FT4 2.09, T3 257-> MMI 10 daily

• Seen in ER 5/7/13 for 2.5 weeks of being slow to respond, staring spells at school
  – Noted to be alert but staring blankly, slow to respond, HR 122, otherwise nl exam
  – Workup negative, d/c with neuro f/u

• TFTs 5/7/13: TSH <0.01, FT4 2.40->MMI 10 bid
HPI continued

• Returned to ER 5/11/13 for complaints of shaking episodes x 5 days
  – Seen by neurology earlier in the day and instructed to decrease amitriptylline and start Neurontin
  – Shaking of head, arms, legs, increasing duration, associated with dizziness, also c/o diffuse pain
  – Seen by neurology who suspected depression

• Endo visit 5/20: At neuro baseline, no tremors
  – TSH <0.01, FT4 1.23, T3 180
  – HR 106-> started atenolol
HPI continued

• Acting herself for ~ 2 weeks
• Transferred from outside ER 6/10 after presented with progressive MS changes x 5 d
• Slow to respond, forgetful, wandering at night rather than sleeping, tremulousness, dizziness, continued headache
• Mother questioned if caused by Neurontin and/or atenolol
• Endo consult to comment on thyroid status and beta blocker discontinuation
PMH

• Graves disease
  – Diagnosed 2002 (age 4)
  – Thyroid storm 2004
  – Subtotal thyroidectomy 2004
  – Required LT4 until 2008->euthyroid off replacement
    2008-2009, MMI restarted 2009, but weaned off by
    3/2012, restarted 7/2012

• Seizure disorder
  – Seizures associated with fever and thyroid storm,
    none since <age 10

• Asthma
Medications on Admission

- Neurontin 300 mg q12h
- Atenolol 25 mg daily
- Methimazole 10 mg bid
- Flovent
- Albuterol prn
Family and Social History

• Family History
  – Graves disease: 11 relatives including cousins, aunts, and an uncle
  – No other known autoimmune diseases
  – No known neurologic or psychiatric disorders

• Social history
  – Lives with mother, only child, father in prison
  – 8\textsuperscript{th} grade, declining school performance x ~ 2 yrs, now has IEP
  – Bullied at school
Physical Exam

- T 37, P120, R24, BP 134/81, Wt 69.85 kg (91%), Ht 162.6 cm (54%), BMI 26.4 (92%)
- Constitutional: Well-developed and well-nourished. No distress.
- HEENT: Normocephalic and atraumatic. Oropharynx is clear and moist.
- Eyes: No scleral icterus. Bilateral exophthalmos
- Neck: Thyroid palpable but not enlarged.
- Cardiovascular: Tachycardic, regular rhythm. No murmurs. No edema.
- Pulmonary/Chest: Effort normal and breath sounds normal.
- Abdominal: Soft. BS nl, NT/ND.
- Neurological: CN 2-12 intact. NI strength and sensation. 2+ reflexes throughout. No tremor or dystonia. NI gait and station.
- Skin: Skin is warm and dry. +acanthosis
- Psychiatric: Alert, oriented to person and place, not time. Cooperative but confused, restless, distractable, coherent speech but significant delay in response, poor insight, impaired memory and attention
Can encephalopathy be associated with Graves disease?

Table 3: Comparison between Hashimoto’s thyroiditis and Graves’ disease patients with encephalopathy associated with autoimmune thyroid disease.

<table>
<thead>
<tr>
<th></th>
<th>HT patients (n = 20)</th>
<th>GD patients (n = 14)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ serum anti-TPO Abs</td>
<td>100%</td>
<td>100%</td>
<td>1</td>
</tr>
<tr>
<td>+ serum anti-TG Abs</td>
<td>60% (n = 10)</td>
<td>58% (n = 12)</td>
<td>1</td>
</tr>
<tr>
<td>CSF protein concentration</td>
<td>85%</td>
<td>64% (n = 11)</td>
<td>0.2</td>
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<tr>
<td>CSF leukocytes count</td>
<td>25%</td>
<td>45% (n = 11)</td>
<td>0.4</td>
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<tr>
<td>normal MRI</td>
<td>74% (n = 19)</td>
<td>62% (n = 13)</td>
<td>0.7</td>
</tr>
<tr>
<td>abnormal EEG</td>
<td>95%</td>
<td>92% (n = 13)</td>
<td>1</td>
</tr>
</tbody>
</table>
SPECT imaging

Study of controls vs. patients with AIT without encephalopathy
Patients had higher scores on SAS and SDS (psychiatric testing)
Patients had more areas of hypoperfusion
But no correlation between scores and hypoperfusion
Hypothesize this means AIT associated with cerebral hypoperfusion
?SPECT really meaningful in SREAIT
Would treatment with steroids prevent further episodes?

- Majority of cases present with relapsing-remitting episodes
- Episodes can resolve spontaneously
- High dose short term steroids may shorten episodes but don’t seem to prevent further episodes
- If episodes continue to recur, may require longer term steroid taper or other immunomodulatory agents for long term remission