60 y/o man with recent onset of left-sided numbness and tingling

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Patient Presentation

• HPI: 60 y/o man with HIV and HLD who awoke from sleep 2 weeks before presentation with numbness and tingling in L torso, which then progressed within 1 week to include his chest, abdomen, back, left arm, and left leg. He did not have any right-sided symptoms.

• ROS: No pain, burning, weakness, diplopia, dizziness, speech difficulty, incoordination, or headache. No fevers, chills, chest pain, SOB, nausea/vomiting, abdominal pain, bowel or bladder issues.

• PMH: HIV (well-controlled), HLD (poorly controlled), OSA.

• PSH: none.

• SH/HRB: no tobacco, occasional EtOH, no drugs.
Objective Data

- Vitals: non-contributory.
- Neuro exam: notable for diminished L hemibody sensation to light touch and vibration, with intact temperature sensation. The precise distribution of the deficit includes the L arm and L leg but spares the neck and head above the collar line. No R-sided sensory deficits. Otherwise AOx3, CN II-XII intact, motor strength globally 5/5, finger to nose intact, normal gait, normal reflexes.
- Labs: cholesterol 268, triglycerides 435, LDL incalculable.
What Imaging Should We Order?
Select the applicable ACR Appropriateness Criteria

<table>
<thead>
<tr>
<th>Variant 4: New focal neurologic defect, fixed or worsening. Longer than 6 hours. Suspected stroke.</th>
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<tbody>
<tr>
<td><strong>Radiologic Procedure</strong></td>
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<tr>
<td>MRI head without IV contrast</td>
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<tr>
<td>MRI head without and with IV contrast</td>
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<tr>
<td>MRA head and neck without IV contrast</td>
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<td>MRA head and neck without and with IV contrast</td>
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<tr>
<td>CT head without IV contrast</td>
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<td>CTA head and neck with IV contrast</td>
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This imaging modality was ordered by the Neurologist.
MRI Head (unlabeled)

DWI (diffusion-weighted imaging)
ADC (apparent diffusion coefficient)
FLAIR (fluid-attenuated inversion recovery)
MRI Head (labeled)

- DWI: mildly reduced diffusion in R thalamus
- ADC: mildly reduced diffusion in R thalamus
- FLAIR: hyperintensity in R thalamus
Final Dx:

Subacute Pure Sensory Stroke of Right Lateral Thalamus
The ventroposterolateral (VPL) nucleus of the thalamus contains the third order neuron cell bodies that relay somatosensory information from the spinothalamic and medial lemniscus pathways to the primary sensory cortex.

On axial images near the level of the anterior and posterior commisures, the VPL nucleus starts laterally in the posterior thalamus near the internal capsule and runs medially in the direction of the posterior commisure (Yamada et al., AJNR 2010).

The ventroposteromedial (VPM) nucleus (not pictured) performs the same function in facial sensation (receiving input from the trigeminal nerve).
Different vascular territories in the thalamus are associated with different nuclei, leading to extremely varied presentations of thalamic stroke.

The arterial supply of the thalamus arises from the Pcomm and the P1 and P2 segments of the PCA, and is divided into four major territories (Schmahmann, *Stroke* 2003):

- **Anterior** and **paramedian** territories: important for arousal, personality, memory, language* (DM and Anterior nuclei)
- **Inferolateral** territory: important for sensation (body- VPL nucleus, face- VPM nucleus)
- **Posterior** territory: important for vision (LGN/Pulvinar)

*Language functions reside in the thalamus ipsilateral to the dominant hemisphere

**Artery abbreviations**
- MCA – Middle cerebral artery
- ICA – Internal carotid artery
- Pcomm – Posterior communicating artery
- PCA – Posterior cerebral artery

**Thalamic nuclei abbreviations**
- DM – Dorsomedial nucleus
- VPM – Ventroposteromedial nucleus
- VPL – Ventroposterolateral nucleus
- LGN – Laternal geniculate nucleus

**Thalamic vascular territories**
- Yellow – Paramedian territory
- Blue – Anterior territory
- Green – Inferolateral territory
- Purple – Posterior territory
Dating Infarcts Using MRI

• In an **acute** infarct, low ATP $\rightarrow$ impaired Na+/K+ ATPase $\rightarrow$ cellular swelling $\rightarrow$ **reduced diffusion** (hyperintense DWI, low ADC)

• These signal changes may persist for 1-3 weeks during the **subacute** phase of the infarct

• In the **chronic** phase, DWI signal is variable (e.g. low in cystic encephalomalacia, but can also be high from T2 shine-through)

<table>
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<tr>
<th>Signal According to Infarct Age</th>
<th>Hyperacute (&lt;6h)</th>
<th>Acute (&lt;1w)</th>
<th>Subacute (1-3w)</th>
<th>Chronic (&gt;3w)</th>
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<tbody>
<tr>
<td>DWI</td>
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<td>(Variable)</td>
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<tr>
<td>ADC</td>
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<td>FLAIR</td>
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References:

