RADY 403
Neonatal Cerebral Infarction
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Focused Patient History and Workup

• JK is a 3-day-old male born at 40w3d by spontaneous vaginal delivery to a febrile, Group B Strep + mother (appropriate abx ppx) but with good APGARs; blood cultures drawn from infant showed no growth.

• Brought to OSH ED after grandma noticed foot twitching, then extremities rhythmic jerking, then a 15-second episode of whole-body jerking. Initially in ED, reassuring exam, no fever, emesis, or difficulty feeding or voiding. No LOC or apnea.

• OSH ED obtained CT, evidence of ischemia/infarct. Pt began to have apneic episodes with cyanosis, reviving after stimulation by nurse.

• Transferred to UNC PICU, where a sepsis workup (LP) was continued and abx started, and pt was intubated, started on IVF, and loaded with Keppra
Neonatal Cerebral Infarction: Differential Dx

- Most common Ischemic Causes: Cardiac Disorders and Hemoglobinopathies (i.e. SCD) \(^1\)

Other causes include \(^2\):
- Bacterial Meningitis
- Coagulopathies
- Trauma
- Hypoxia/Ischemia
- Blood vessel anomalies (more likely hemorrhagic)
List of Imaging Studies

- Non-contrast Head CT (at OSH)
- Portable CXR (on admission)
- Echocardiogram (on admission)
- MRI Head wo Contrast (on admission)
- MRA Head wo Contrast (on admission)
- MRI Head wo Contrast (Hospital Day 3)
- MRA Head wo Contrast (Hospital Day 3)
- MRA Neck wo Contrast (Hospital Day 3)
Non-contrast Head CT (at OSH)

Findings axial: “Subacute large left MCA territory infarct in left frontal and left parietal lobes.”

Findings sagittal: “Hyperdense appearance of the superior sagittal sinus, internal cerebral veins, vein of Galen, straight sinus, torcula, and cavernous sinus.” Initially questioned extensive thrombus, but over-read as more likely “related to high hemoglobin/hematocrit level relative to the edematous brain.”
Portable CXR (on admission)

Findings: ET Tube appropriately positioned above the level of the carina. Enteric tube in place in stomach. Normal cardiac silhouette, bones intact. No increased pulmonary vasculature or opacities.
Echocardiogram (on admission)

**Findings:** PFO, with left to right shunting of blood. Overall, ventricular size was normal, no concern for congenital malformations leading to hypoxia/ischemia.
Findings: Left hypointensity on T1 axial, hyperintensity on T2 axial, and hyperintensity on diffusion-weighted imaging all consistent with findings on CT of late acute/early subacute left MCA infarct.
**Findings:** TOF sequence images; no abnormal findings, vasculature is patent, no evidence of stenotic vessels, occluded vessels, or aneurysms. Apparent vascular cutoff more likely related to artifact and non-concerning on source images.
MRI Head w/wo Contrast (Hospital Day 3)

Findings: Similar localization as initial findings, now hyperintensity in MCA region on T1 and T2 weighted images.

Findings: New finding of restricted diffusion (hyperintense) along left lateral corticospinal tract, evidence of Wallerian Degeneration.
Findings: TOF sequence images; as on Hospital Day 1 there are no abnormal findings. No stenosis, occlusions, or aneurysms. Again, cutoff is most likely artifact.
Findings: TOF sequence images; No stenosis, occlusions, or aneurysms. Areas that appear stenotic here were normal on source images.
Patient Outcome

• Infectious workup (including HSV/CMV/Entero/VZV PCR, CSF culture) not concerning for meningitis or encephalitis, urine and blood cultures negative at 48 hours

• CBC w/ diff, CMP, and extensive workup for other congenital disorders did not demonstrate any coagulopathies or genetic abnormalities

• Most likely etiology considered to be birth trauma/perinatal event

• Pt was stabilized and managed on multiple anti-epileptics, now receiving PT and works with Children’s Developmental Services, “doing well”
## ACR Criteria

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Procedure</th>
<th>Adult RRL</th>
<th>Peds RRL</th>
<th>Appropriateness Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke suspected, acute, emergent intervention contraindicated, initial imaging</td>
<td>MRI head without IV contrast</td>
<td>0 mSv</td>
<td>0 mSv [ped]</td>
<td>Usually appropriate</td>
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<td>CT head without IV contrast</td>
<td>1-10 mSv</td>
<td>0.3-3 mSv [ped]</td>
<td>Usually appropriate</td>
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<td></td>
<td>MRA head without IV contrast</td>
<td>0 mSv</td>
<td>0 mSv [ped]</td>
<td>Usually appropriate</td>
</tr>
<tr>
<td></td>
<td>MRI head perfusion with IV contrast</td>
<td>0 mSv</td>
<td>0 mSv [ped]</td>
<td>May be appropriate</td>
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<tr>
<td></td>
<td>CTA head with IV contrast</td>
<td>1-10 mSv</td>
<td>3-10 mSv [ped]</td>
<td>May be appropriate</td>
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<tr>
<td></td>
<td>MRI head perfusion without IV contrast</td>
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<td>0 mSv [ped]</td>
<td>May be appropriate</td>
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<tr>
<td></td>
<td>MRI head without and with IV contrast</td>
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<td>0 mSv [ped]</td>
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<td></td>
<td>MRA head with IV contrast</td>
<td>0 mSv</td>
<td>0 mSv [ped]</td>
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<td>0 mSv [ped]</td>
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<tr>
<td></td>
<td>US duplex Doppler head</td>
<td>0 mSv</td>
<td>0 mSv [ped]</td>
<td>Usually not appropriate</td>
</tr>
</tbody>
</table>
Features of Ischemia on CT

Acute
- Cytotoxic Edema (more accurately, ionic edema)
- Ischemic region more dense than CSF
- ± Mass Effect (volume gain, with possible herniation, midline shift, or ventricular effacement)  

Chronic
- Encephalomalacia, Wallerian Degeneration
- Ischemic region shares similar density as CSF
- ± Mass Effect (volume loss, with possible widened sulci, or ex vacuo dilatation of ipsilateral ventricle)  

Ionic Edema: Uniform low attenuation, blurred gray-white matter junction, cortical ribbon swells

Insular Ribbon Sign (blue arrow): Early sign of MCA infarct, loss of gray-white distinction
Features of Early Ischemia on MRI

• T1: Hypointensity shows after ~16 hours and remains, due to cortical laminar necrosis

• T2: High signal appears after ~6 hours, increasing over 1-2 days (more apparent on FLAIR sequence)

• DWI: Increased signal within minutes

DWI measures the diffusion of water molecule in multiple directions to create a 3-dimensional “diffusion tensor”, where areas with more diffusing molecules appear darker. Thus, ischemic areas where diffusion is low are hyperintense.
Features of Late Ischemia on MRI

• T1: Remains low, can be high if cortical necrosis is present.\(^7\)

• T2: Remains high, cortical contrast enhancement remains \(~2\text{-}4\) months. *If parenchymal enhancement remains after 12 weeks, consider underlying lesion.*\(^7\)

• DWI: As time passes, the signal intensity will continue to decrease.\(^7\)
Take-home Points

• Broad differential for ischemic infarcts in neonates, but most common presenting symptom is contralateral focal seizure

• MRI w/ DWI is most sensitive, although non-contrast head CT is appropriate for initial imaging

• Various signs on CT and MRI demonstrate the acuteness of the infarct (cytotoxic edema, Wallerian degeneration, insular ribbon sign)

• Patient outcomes are variable, but often children will regain full motor function\(^2\)
References


