Hyperdense Intracranial Vessels: Eliminating Observational & Interpretive Errors

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Call Readiness in Radiology Residency

Trainee call readiness is important to patient safety

Universal tools for call readiness evaluations
- ACR In-Service Exam
- Simulation Exam

A computer based simulation (SIM) reflecting a typical emergency imaging shift was developed at our institution and administered to
- 103 first (R1) & second (R2) year residents
- From 9 radiology training programs in 2014
# Call Readiness in Radiology Residency

## Comparison of ACR In-Service with SIM exam

<table>
<thead>
<tr>
<th><strong>ACR In-Service Exam</strong></th>
<th><strong>SIM Exam</strong></th>
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<tr>
<td>Covers the entire spectrum of radiology</td>
<td>Presentation of dedicated emergency radiology cases only</td>
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<td>Not specifically designed for assessing “independence”</td>
<td>Cases specifically chosen to assess patient’s safety and resident’s independence</td>
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<td>Only key images are given minimizing observational error</td>
<td>Cases are presented as full DICOM image sets maximizing observational error</td>
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<td>Lack of inclusion of normal studies</td>
<td>Inclusion of normal studies</td>
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<td>Multiple choice questions assess passive knowledge</td>
<td>Free text responses access active knowledge</td>
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<td>Inability to differentiate between observational &amp; cognitive errors</td>
<td>Assessing observational versus cognitive errors</td>
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Case Presentation

Given history: 32 year old female with acute onset of weakness, diplopia and altered mental status. Possible seizure.

Non-contrasted, axial CT image of the SIM case shows markedly hyperdense basilar artery concerning for basilar artery occlusion. Hyperdense vessel(s) are often overlooked on CT especially when midline or symmetric in distribution.
SIM exam results:

- 90% of residents failed to recognize the hyperdense basilar artery = *observational error*
- Only 7% of residents raised the concern for basilar artery thrombosis
- No significant difference in the performance between R1 and R2 residents
- Independent of radiology program

**Conclusion:** Significant observational gaps exist in detection of hyperdense vasculature placing patients at risk for delay in diagnosis and appropriate treatment.
**Purpose**

Purpose of this electronic exhibit is to close this observational gap by:

- Discussing key imaging features in identifying hyperdense vessels = **observational error**
- Reviewing common pitfalls when making this diagnosis = **cognitive error**
- Discussing appropriate action to take after making the correct diagnosis = patient safety issue
Pathophysiology of Hyperdense Vessels

- 40 HU = Hounsfield units for normal flowing blood
- Acute clot measures about 60-80 HU as it is red blood cell rich and poor in serum
- Chronic clot measures about 16-32 HU as it mainly consists of fibrin
- Other causes of elevated Hounsfield units within the vessel lumen
  - Calcium
  - Dilute contrast
  - Elevated hematocrit
Hyperdense Vasculature

- Arterial versus venous
- Single versus multiple hyperdense vessels
- Mimics
Single Hyperdense Arterial Vessel
Non-contrasted, axial CT images of two different patients demonstrate hyperdense proximal MCA and distal MCA related to MCA artery occlusion. These hyperdense vessels are easier to depict than the SIM case as they are asymmetric in distribution.
Notice the relative hyperdensity of the left proximal M1 when compared to the normal right M1 on this non-contrasted CT image. This can be seen with vessel occlusion even prior to loss of the grey-white differentiation or sulcal effacement. The CTA of the same patient shows a vessel cutoff confirming the diagnosis. This patient subsequently underwent thrombectomy.
The non-contrasted, axial CT shows a homogeneously hyperdense right MCA suspicious for acute thrombus despite lack of secondary signs within the brain parenchyma. The subsequently performed MRI reveals restricted diffusion in the right MCA territory and MCA/PCA watershed territory. Advance to the next slide to see additional images.
MRA of the same patient as on prior slide demonstrates **nonopacification of the right MCA** confirming the validity of the hyperdense vessel sign on CT. There is also **occlusion of the right ICA at its origin** on MRA of the neck.
The non-contrast axial CT images show increased density of the **basilar artery at the midbrain level**. This is a subtle finding which becomes more conspicuous when compared to the basilar artery density **at the level of the pons**. The suspected **distal basilar artery occlusion** is confirmed on the sagittal CTA image.
The non-contrasted cervical spine CT in soft tissue algorithm reveals subtle hyperdensity of the V3 & V4 segments of the left vertebral artery. This should be especially concerning if there are secondary findings of cervical spine fracture, seatbelt sign, or significant mechanism of injury as this can denote dissection or intramural hematoma. Advance to the next slide for additional images.
Vertebral Artery Dissection

The non-contrasted cervical spine CT in bone algorithm better delineates the fractures of the C1 ring which were associated with a fracture of the dens (not pictured). The subsequently performed T2 images confirm abnormal flow in the left vertebral artery reflected as **hyperintense signal** in contrast to the **preserved normal flow void** in the right vertebral artery.
Single Hyperdense Arterial Vessel Mimics
**Single Hyperdense Arterial Vessel Mimics**

- **Atherosclerosis - MCA**

Non-contrasted CT reveals **hyperdense right MCA** when compared to the **normal left MCA**. Note that the hyperdensity is more patchy than confluent as in the previous case. This is more characteristic for calcium deposition due to atherosclerotic disease. The **patency of the right MCA** was confirmed with CTA.
This non-contrast CT image at the level of the foramen magnum was interpreted as hyperdense right vertebral artery due to thrombus or atherosclerosis. Subsequent MRI of the brain demonstrated patency of the right vertebral artery on post contrasted T1 without diffusion restriction. The T2 shows irregularity within the vessel lumen more consistent with atherosclerosis.
As in the SIM case, the **basilar artery** is **hyperdense** on this non-contrast CT image concerning for acute thrombus. However, the follow up MRI reveals no restricted diffusion and preservation of the **flow void** on T2 & MRA MIP images consistent with patent **basilar artery**.
Single Hyperdense Venous Vessel
Non-contrast CT image reveals an expanded and hyperdense right transverse sinus consistent with transverse sinus thrombosis. The non-contrast CT image in a different patient shows a more subtle hyperdensity of the right transverse sinus that was confirmed to represent thrombosis on the CTV.
Non-contrasted, axial CT images show a markedly hyperdense superior sagittal sinus. This is harder to depict than the transverse sinus thrombosis on the prior slide due to the symmetric nature of the involvement. Sagittal T1 image confirms the lack of flow void consistent with posterior superior sagittal sinus thrombosis.
Multiple Hyperdense Venous Vessels
Note the hyperdense appearance of the transverse sinuses bilaterally with extension into the left sigmoid sinus, and inferior & superior sagittal sinuses. Due to the symmetric involvement, one could overlook this abnormality or consider it to be caused by a hyperosmotic state. However, note there is no corresponding increased density of the basilar artery to support the latter. On corresponding CTV the transverse sinuses do not opacify with contrast confirming the diagnosis of sinus thrombosis. Notice the marked enhancement in the collateral dural veins.
Extensive venous sinus thrombosis manifesting as distention and increased density of the right transverse sinus, right sigmoid sinus, superior sagittal sinus, sinus rectus & internal cerebral vein. The diagnosis was confirmed with MRV that shows patency of small, primarily cortical veins only.
Multiple Hyperdense Vessel Mimics
Polycythemia vera versus retained contrast

Non-contrasted, axial CT images of two different patients reveal bilateral hyperdense transverse sinuses. Such finding can be caused by bilateral transverse sinus thrombosis, elevated hematocrit due to dehydration or polycythemia vera and retained contrast agent in a chronic renal disease patient. Bilateral transverse sinus thrombosis usually manifests with significant neurological symptoms in contrast to the other patients that are typically neurologically intact.
Non-contrasted, axial CT images show diffusely hyperdense vessels including the **basilar artery, MCA’s, transverse sinuses** and **superior sagittal sinus**. This is caused by retained contrast that was given to this patient with end stage renal disease without a focal neurologic deficit for an abdominal CT 4 hours prior to the head CT study.
Hyperosmolarity secondary to inebriation

Non-contrasted, axial CT images reveal a hyperdense basilar artery similar to the SIM case presented earlier. In this patient, however, there is also hyperdensity of the MCA bilaterally and superior sagittal sinus. The diffuse nature of the vascular hyperdensity should trigger the correct diagnosis of elevated serum osmolality in this patient with alcohol intoxication.
Non-contrast CT shows diffuse hyperdense arterial & venous vessels. As in the prior two cases, this might be related to retained contrast or elevated hematocrit. In this patient however, this is caused by diffuse edema manifesting as **loss of grey white differentiation & effacement of the sulci** due to hypoxic brain injury. The hyperdense **tentorium & falx** provides an easily detectable hint to the correct diagnosis.
Non-contrast CT shows diffuse hyperdense arterial & venous vessels. In this patient however, this is caused by pathologically elevated hematocrit due to polycythemia vera mimicking "red thrombus" (acute thrombus). Of note, polycythemia vera patients are more prone to infarction(s). Therefore, careful attention must be paid to clinical history & secondary signs of infarctions in each of these patients.
Hyperdense vessel sign = commonly missed finding

Radiologists can minimize their risk of missing the hyperdense vessel sign by:

- Specifically noting the density of arteries and veins
- Observing whether it is diffuse or focal
- Investigating the clinical history
  - Neurologic deficits
  - Comorbid conditions (ie ESRD, Polycythemia vera, dehydration)
  - History of recent contrast administration

If a hyperdense vessel sign is suspected, this requires a phone call to the ordering physician.
References


