Case Presentation:

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History of Present Illness:

- 58 y/o left-handed female patient
- Major Depressive Disorder, C-ETOH, cirrhosis, known alcohol withdrawal seizures
- Found on the floor unresponsive, with apparent evidence of LOS control
- Arrived to local hospital, in status epilepticus, GCS 10/15
- Initial HCT performed at local hospital showed traumatic SDH
- Patient transferred to our Hospital for neurosurgery evaluation

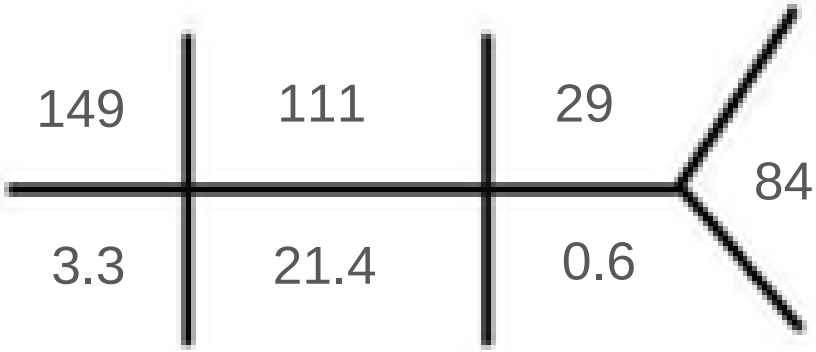
Past Medical History:

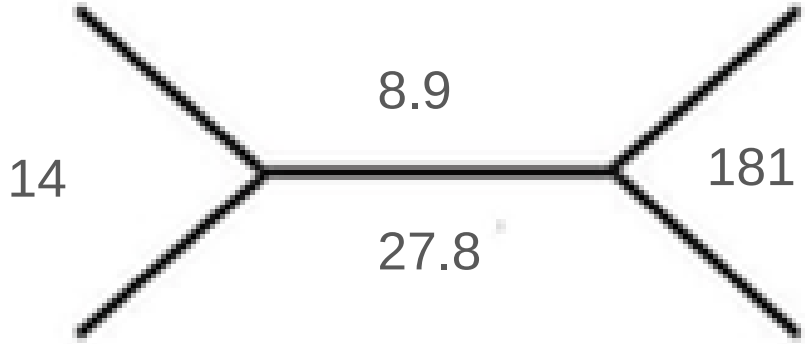
- Allergies: NKDA
- **PMHx: No history of vascular lesions, vasculopathy, coagulopathies, connective tissue disorder**
- Meds: Ativan, antidepressants
- Family Hx: DM, HTN
- Hospitalization Hx: unknown
- Surgical Hx: Hysterectomy, Breast implants, Right foot fracture repair
- Trauma Hx: unknown
- Blood Trans Hx: unknown
- Social/Toxic: Lives alone, C-ETOH

Physical examination:

- Vital Signs: BP 125/75, HR 77, RR 25, T 36.6
- Gen: patient extubated, but not verbalizing, following commands inconsistently, maintains eyes open for several minutes, **left hemineglect**
- SCF: **global aphasia**
- Cranial Nerves II-XII: **right-sided preferential gaze**
- Motor: **profound left hemiparesis**
- Sensory: withdraws LUE to painful stimulation, no withdrawal from RUE, B/L LEs
- Cardiac: No carotid bruits or cardiac murmurs

Laboratories:





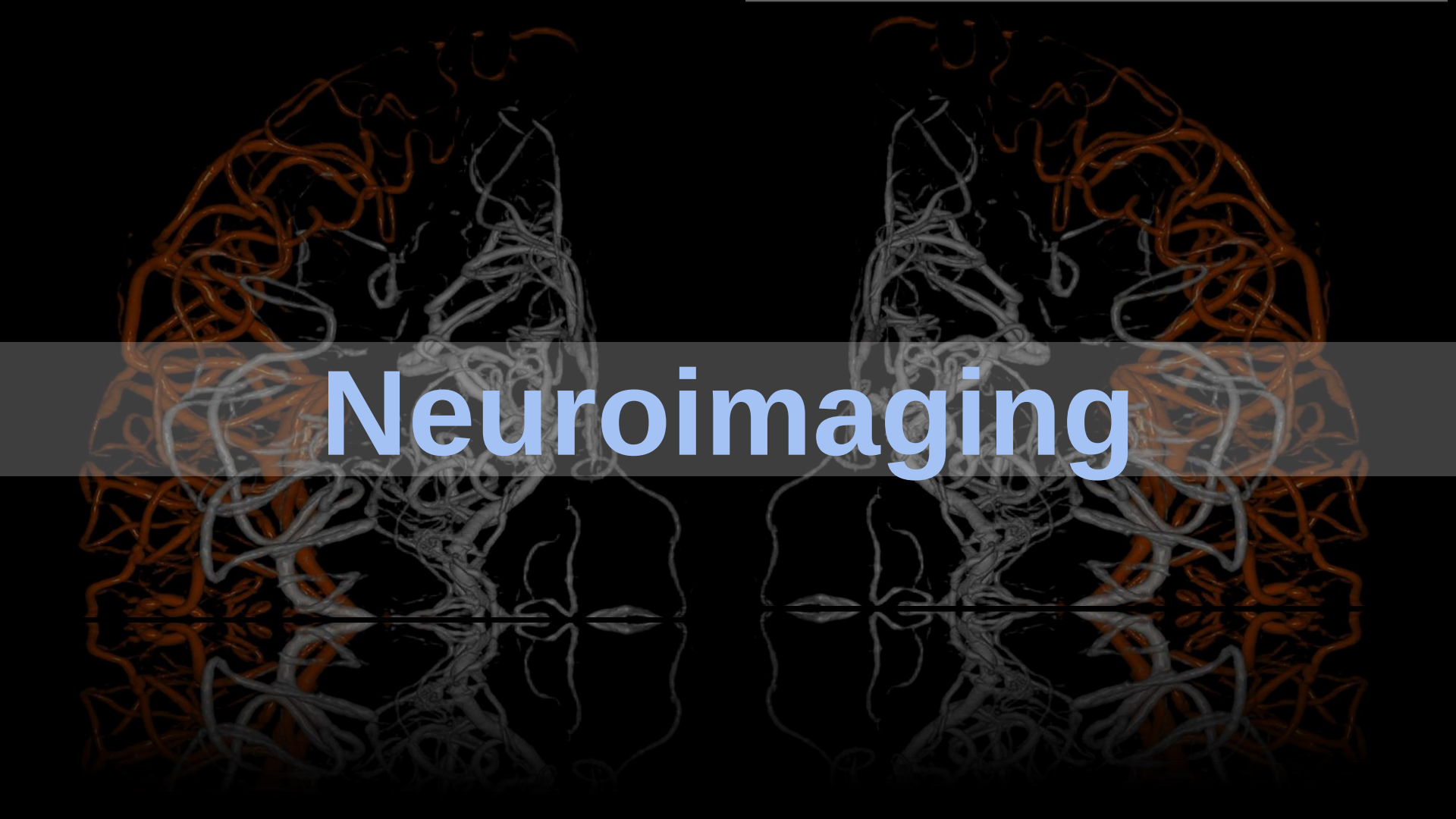
- **AST**: 62
- **ALT**: 35
- **Mg**: 1.7

Tox:

- negative

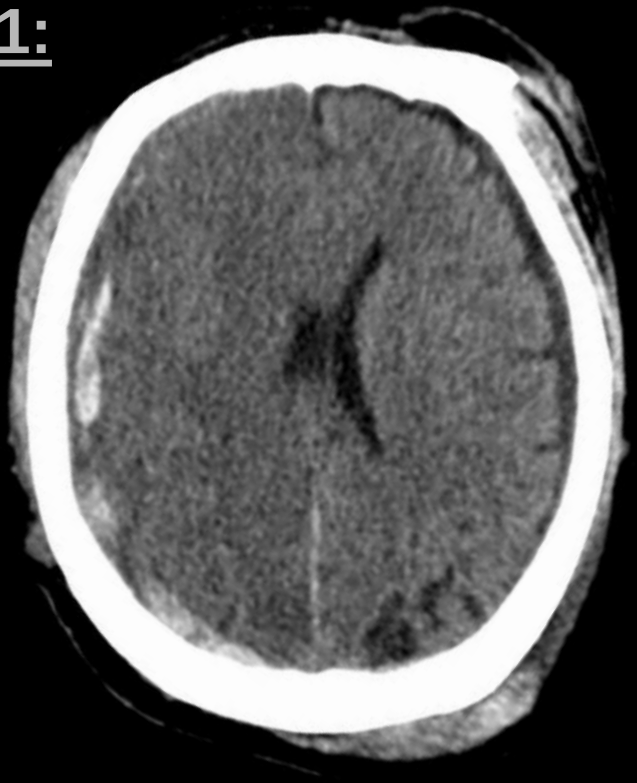
Coag:

- PT:12.1
- PTT: 1.04
- INR:29.4

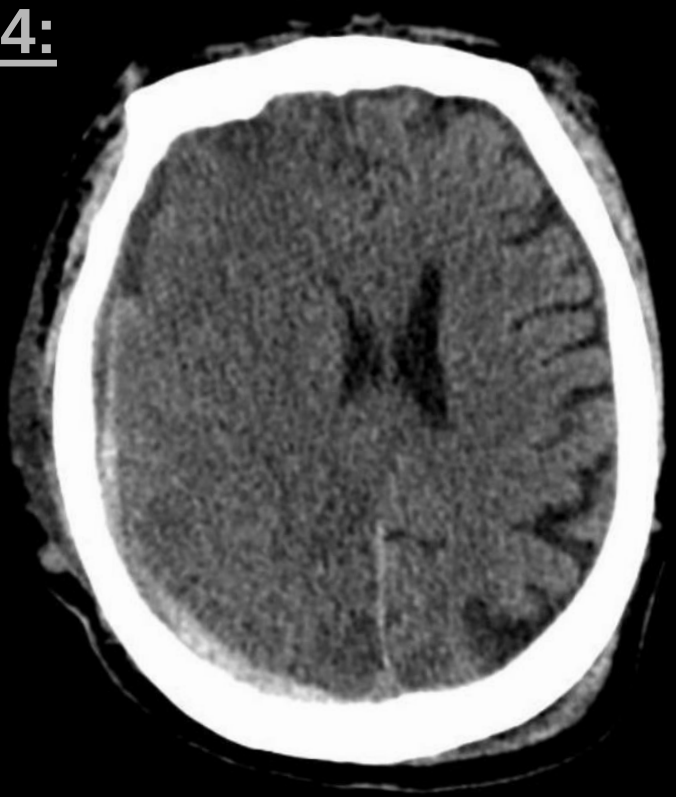

Neuroimaging

HCT without contrast:

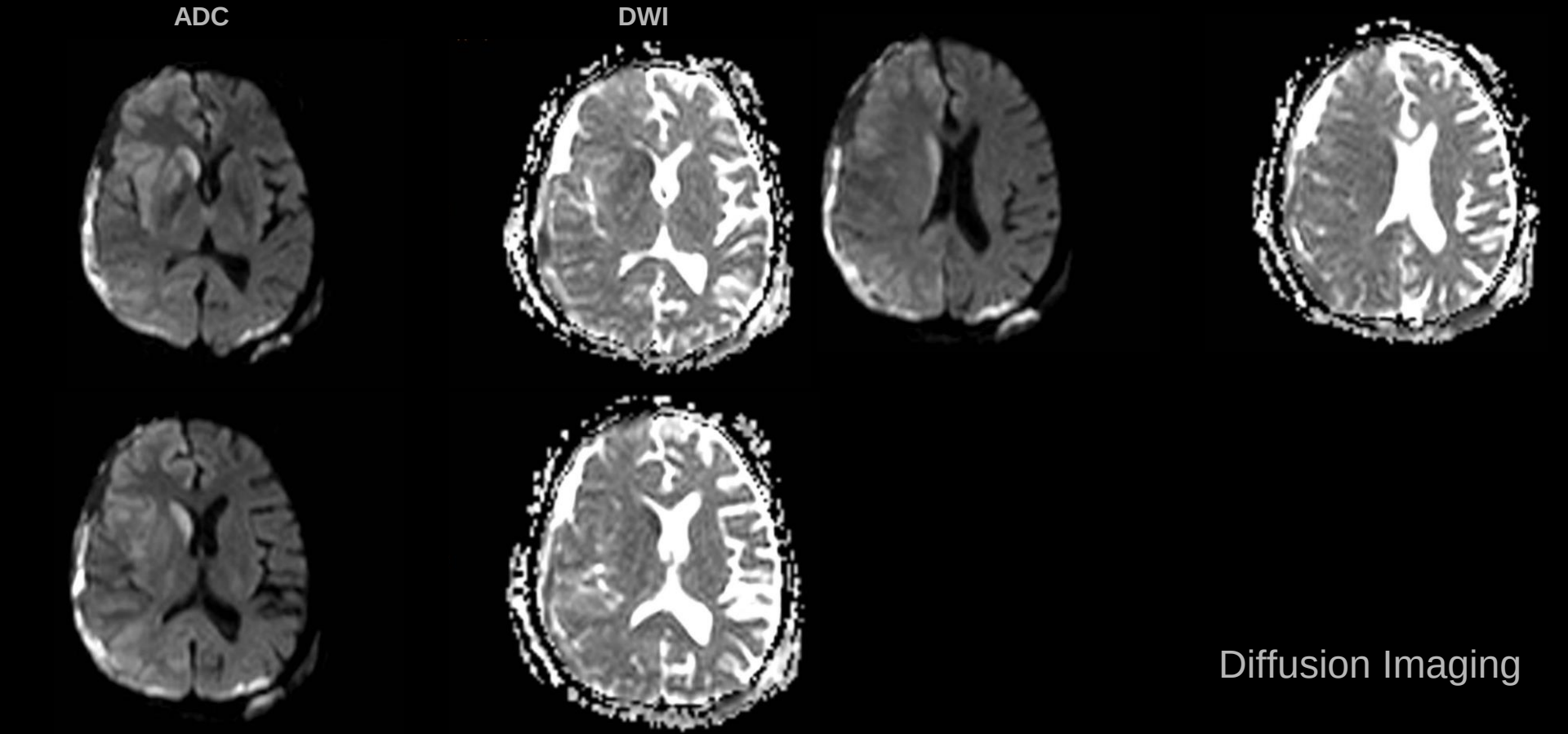
Day 1:



Day 4:

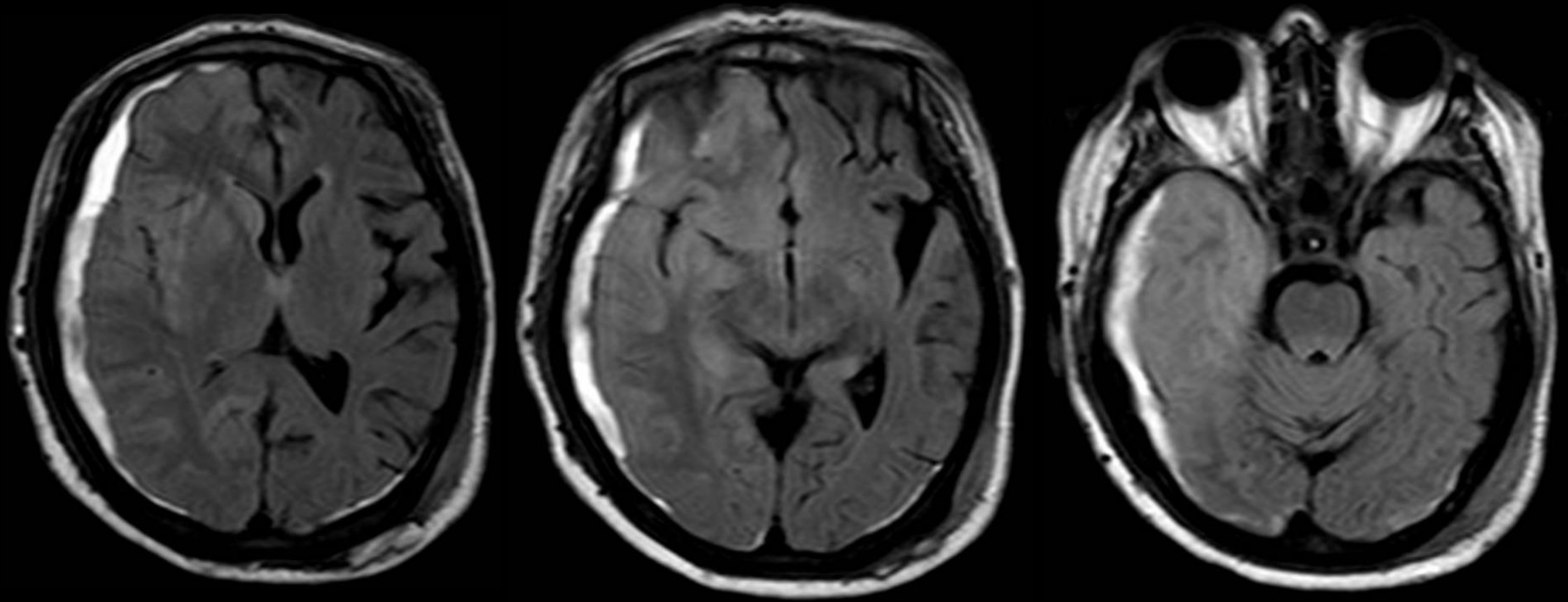


Brain MRI wwo contrast (Day 8):



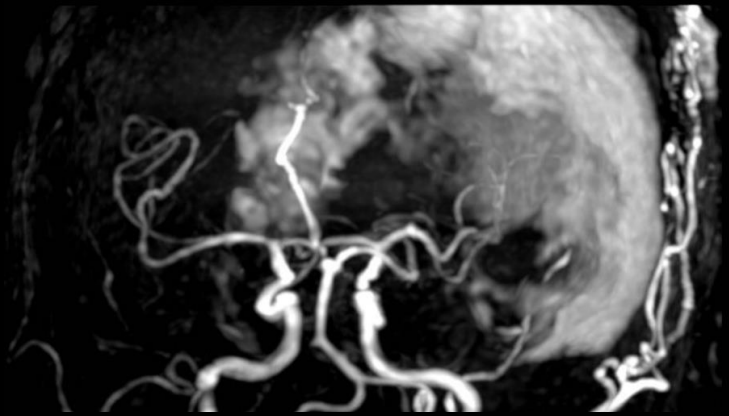
Diffusion Imaging

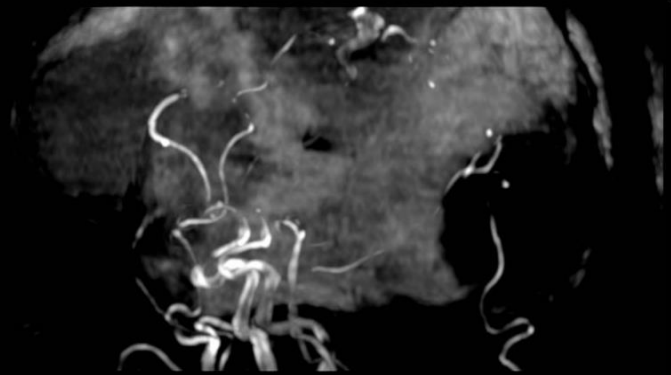
Brain MRI wwo contrast (Day 8):

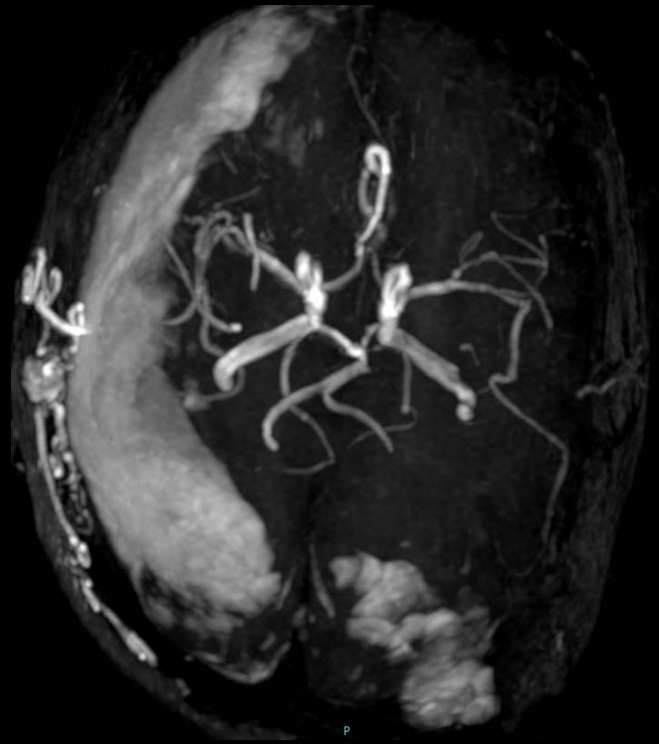


T2 FLAIR

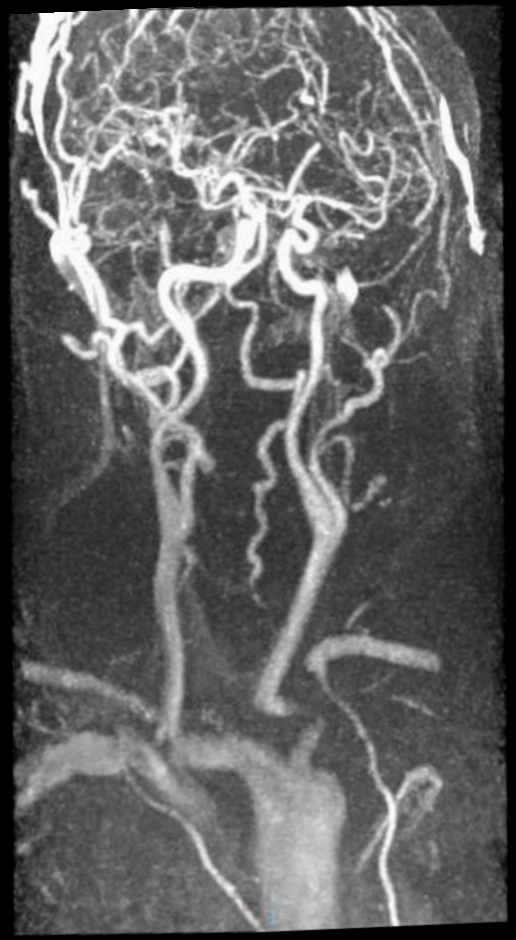
Brain MRA (Day 8):







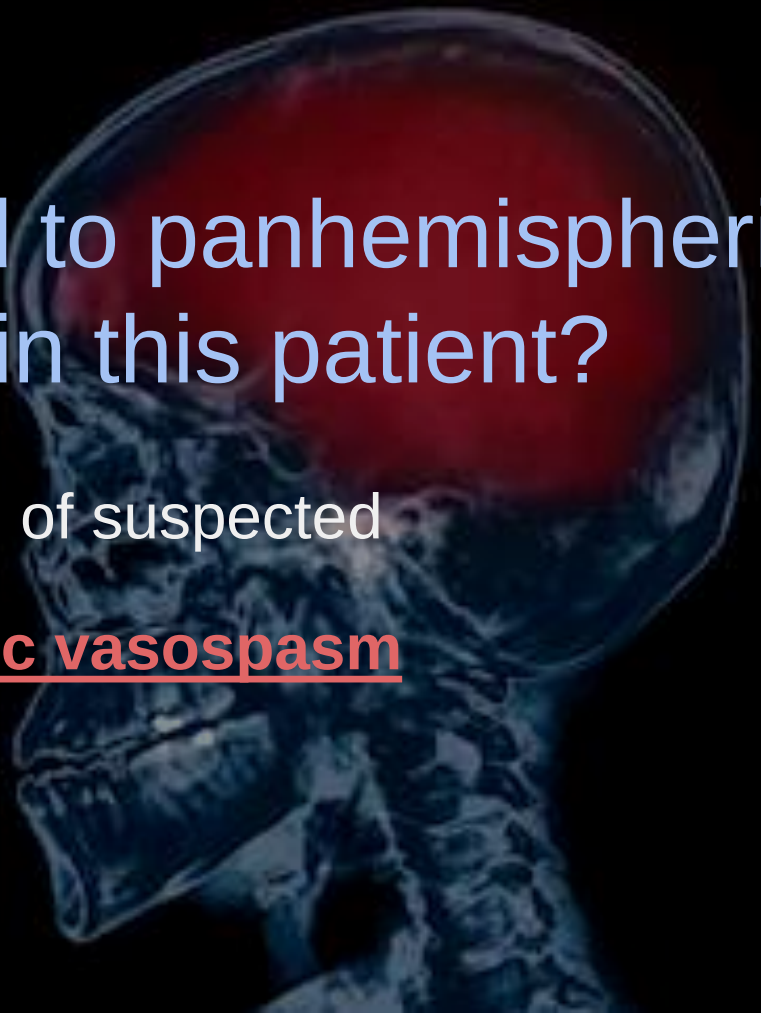
Brain MRA (Day 8):



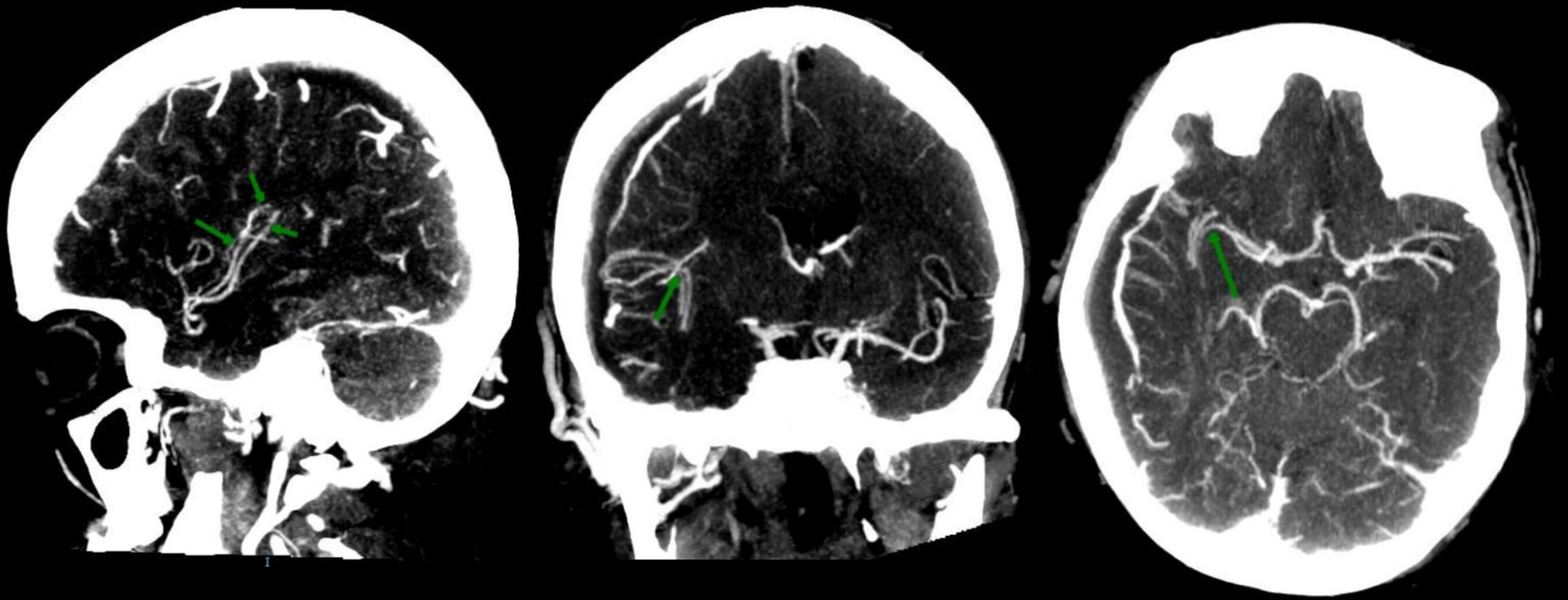
What may have lead to panhemispheric unilateral CVA in this patient?

This is a case of suspected

post-traumatic vasospasm



H&N CTA (Day 14)



Post-traumatic Cerebral Vasospasm

- Severe vasospasm after TBI with minimal, focal tSAH is rare and often forgotten
- Pathophysiology remains elusive
- Mechanical stretching, inflammation, calcium dysregulation, endotelin, contractile proteins, products of cerebral metabolism and cortical spreading depolarization have been involved in PTV pathophysiology
- Distal regions of vasospasm may have been missed on the initial MRA
- They may have been possibly detected with an initial CTA instead

Post-traumatic Cerebral Vasospasm

TABLE IV.—*Time course and prognostic of post-traumatic vasospasm.*[2, 10, 48, 49, 75]

Time course	Onset	Duration	SAH	Prognosis
Early	Within 24 hours	<14 days	Possibly absent	Poor
Late	After 48 hours	3 days to 3 weeks	Present	Variable

TABLE III.—*Risk factors of post-traumatic cerebral vasospasm.*

Risk factor of PTV	Authors
Severity of TBI with low GCS (< 9) on admission	Lee 1997,[62] Oertel 2005 [6]
Young age (< 30)	Oertel 2005 [6]
Traumatic subarachnoid haemorrhage	Abraszko 1996,[50] Zubkov 2000,[68] Oertel 2005,[6] Armin 2008 [65] Aminmansour 2009 [67]

References:

- https://www.cambridge.org/core/journals/canadian-journal-of-neurological-sciences/article/severe-cerebral-vasospasm-and-infarction-after-minor-head-trauma/C90F3297B5941336B1DFEAAE7ACE5D3A/core-reader
- https://www.sciencedirect.com/science/article/pii/S096758689890028 5
- https://www.minervamedica.it/en/getfreepdf/oj4ypqH4Rfdwp3%252BnCDOpk557TyuxV5UV96eUuhHy%252FiAXoULk8FyF75pi%252BYWMZBpDOJlcrlY%252Fxp5Ht8Cs3nloTA%253D%253D/R02Y2015N11A1219.pdf
- http://www.rimed.org/rimedicaljournal/2014/07/2014-07-45-case-fehnel.pdf


Thank you!