

Carotid

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- 69 y/o gentleman with PMH:
 - CAD (PCI 2010), HTN, HLD, DM
 - CVA (2001) followed by 6 episodes of TIA with residual right sided hemiparesis. Presumably left hemispheric event
- **Presenting complaint:**
 - Slurring of speech and worsening right sided weakness for 45 minutes at OSH
 - Similar episode 3 days ago and was d/c from OSH with antiplatelet regimen.
 - Non-invasive testing showed significant RICA disease. Transferred for higher level of care.

OSH work up

- **CT Brain:** “No acute intracranial abnormality is identified, generalized cortical atrophy, periventricular and subcortical white matter density changes are consistent with but not limited to microvascular ischemia and or demyelination and there is a ***probable lacunar infarct in the left insular cortex measuring approx 4 mm in diameter***”

- **CT angiography of the brain :** ***severe stenosis in the distal vertebral arteries*** in the proximal portions the intradural segments.

- **CT angiogram of neck:**

- critical stenosis at the origin of the ***right internal carotid artery estimated at greater than 95%***
- ***severe/critical stenosis in the distal left vertebral artery estimated at greater than 90%***
- severe stenosis in the distal right vertebral artery estimated ~70%

MRI

- Multiple acute infarcts are seen throughout the right medial and lateral temporal lobe, right occipital lobe, right thalamus, posterior limb of the internal capsule on the right, right caudate, and right frontal and parietal lobes. ***These findings are compatible with embolic infarcts, possibly from a internal carotid artery source.***
- Right frontal parietal and right occipital cortex may represent old infarcts. Old bilateral L basal ganglia, left thalamic, and left pontine old infarcts noted.

MRA

- **Brain:** No evidence of major vessel stenosis or occlusion in the anterior circulation. Irregularity throughout the basilar artery and moderate narrowing in the proximal basilar artery.
- **Neck:** Greater than 70% stenosis at the origin of the right internal carotid artery. Mild narrowing of the origin of the left internal carotid artery.

TTE

- Normal LV size and systolic function with impaired relaxation.
- LA/RA normal size
- No intracardiac source of emboli

- Stroke Neurology was consulted

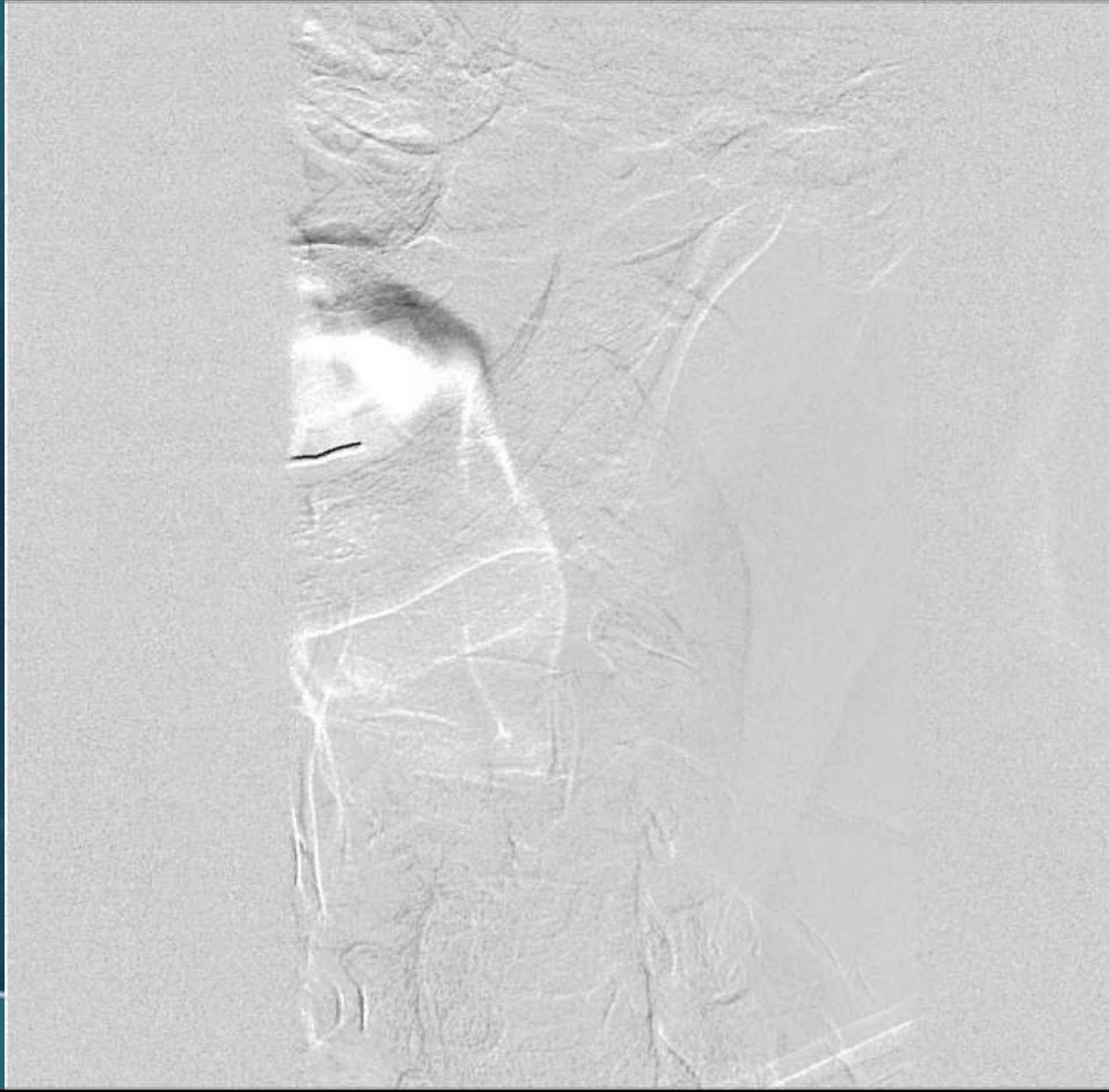
“The pattern of infarcts, involving right middle cerebral artery, PCA and anterior choroidal territories, is consistent with right internal carotid artery stenosis, with right internal carotid artery also supplying PCA through PComm and collaterals. There is little flow to either PCA from the basilar artery, in absence of critical basilar stenosis, suggesting that his congenital anatomy is of a small posterior circulation with PComm supply to PCAs”

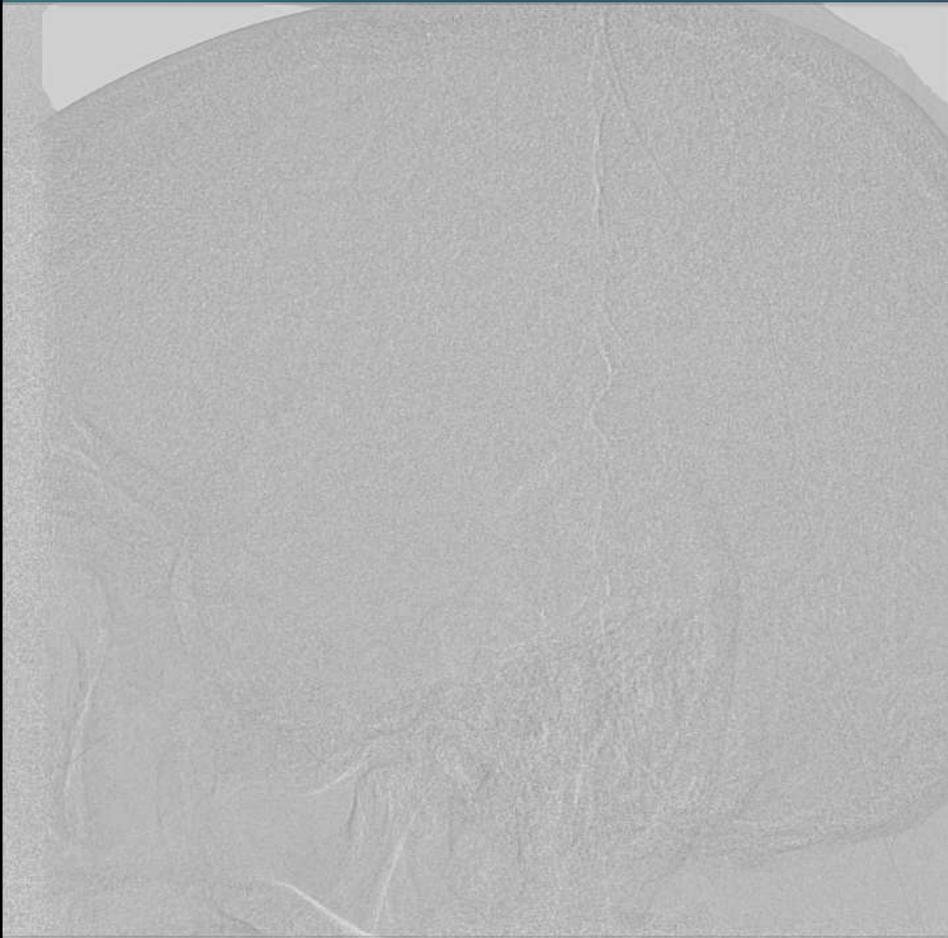
Team decision: intervene on RICA stenosis.

Cath lab was activated on Sunday afternoon...

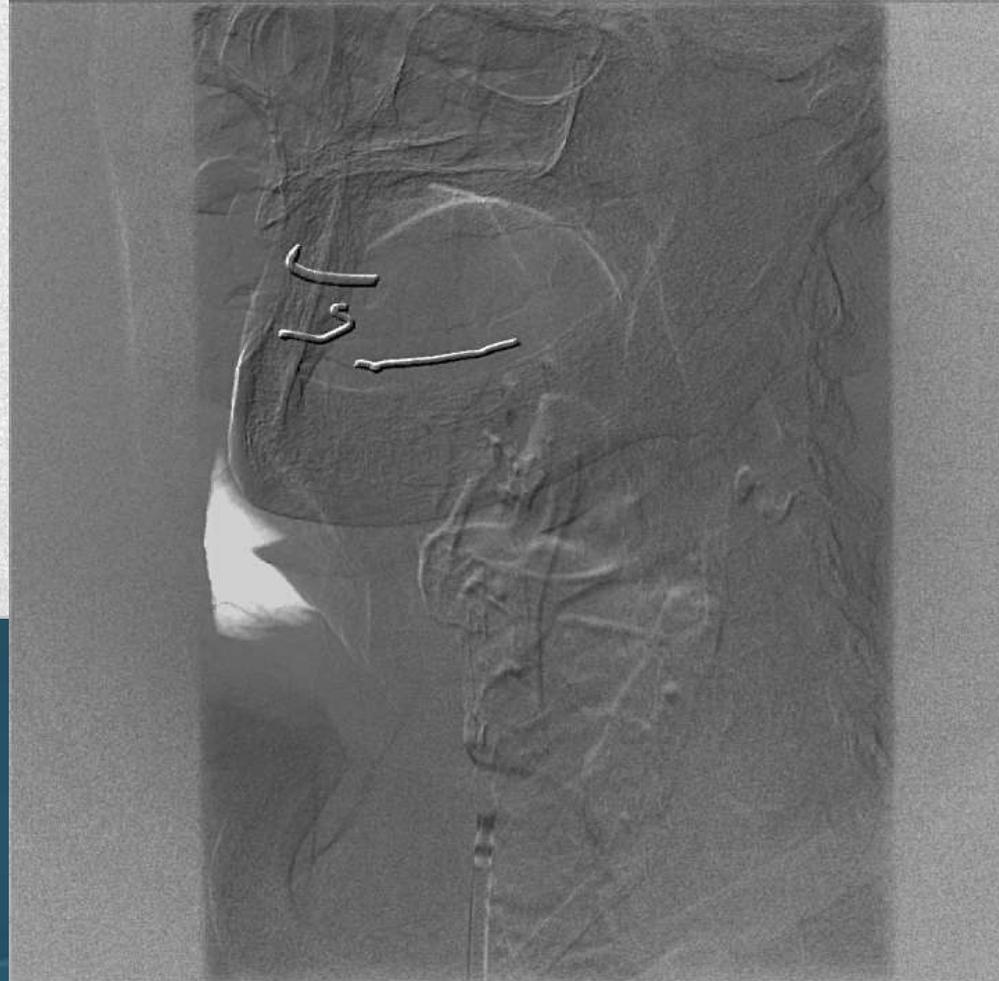
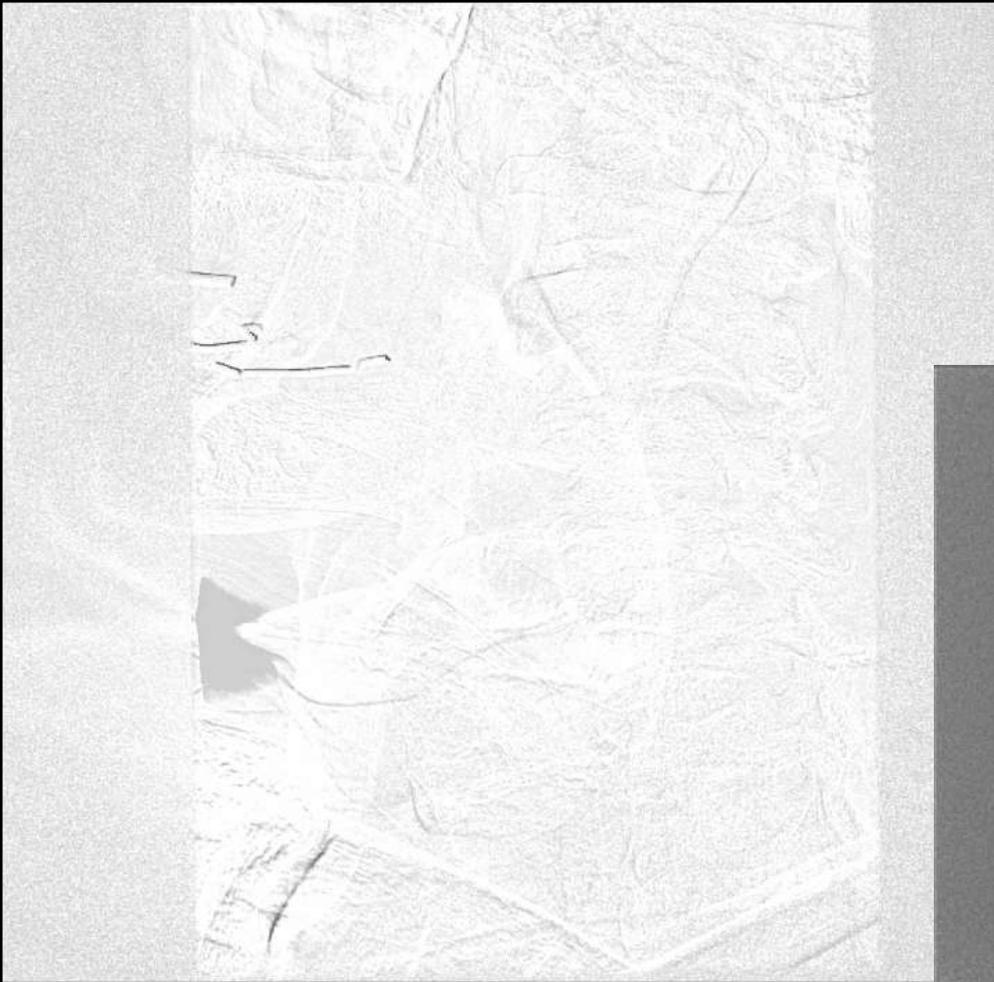
Aortic arch angiogram

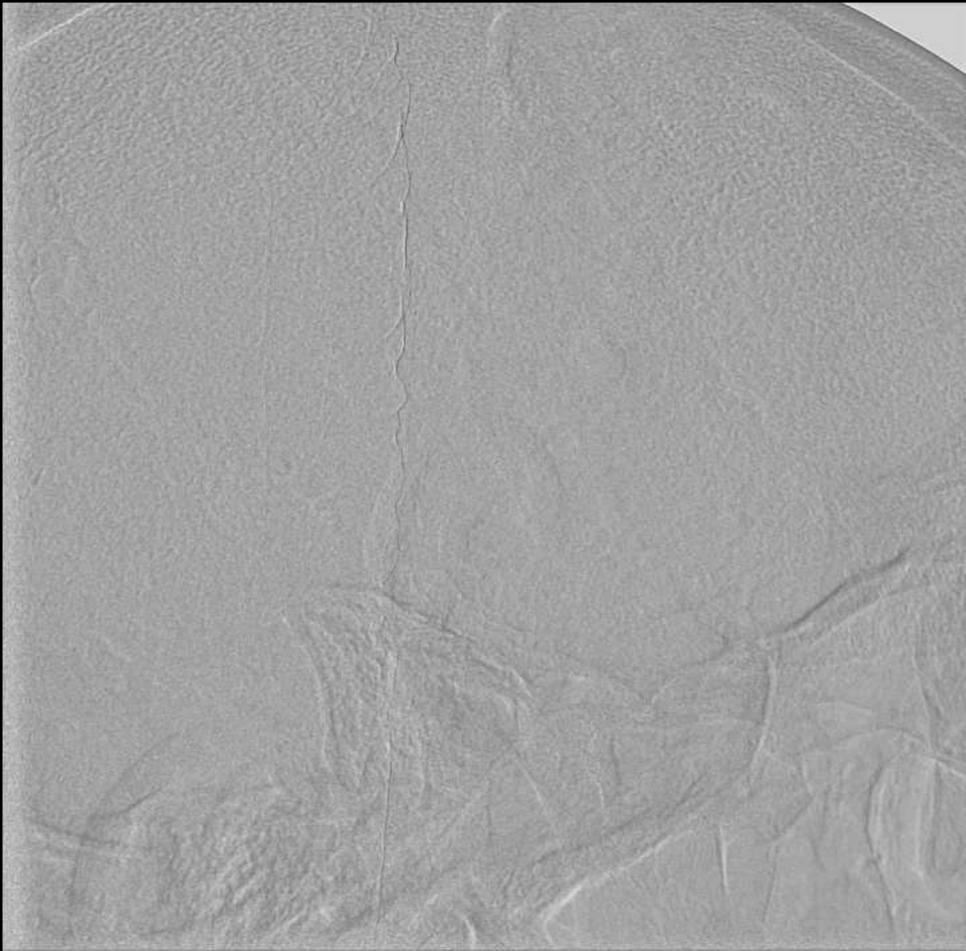


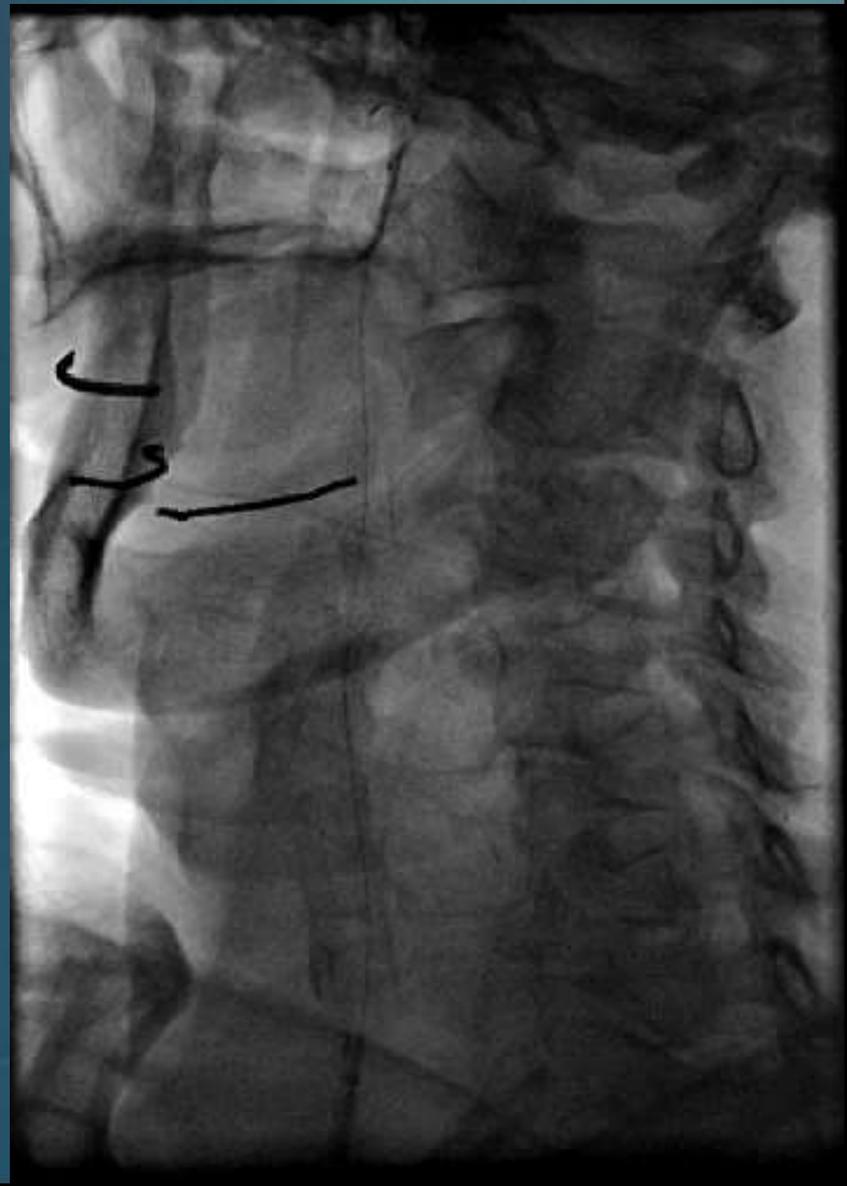
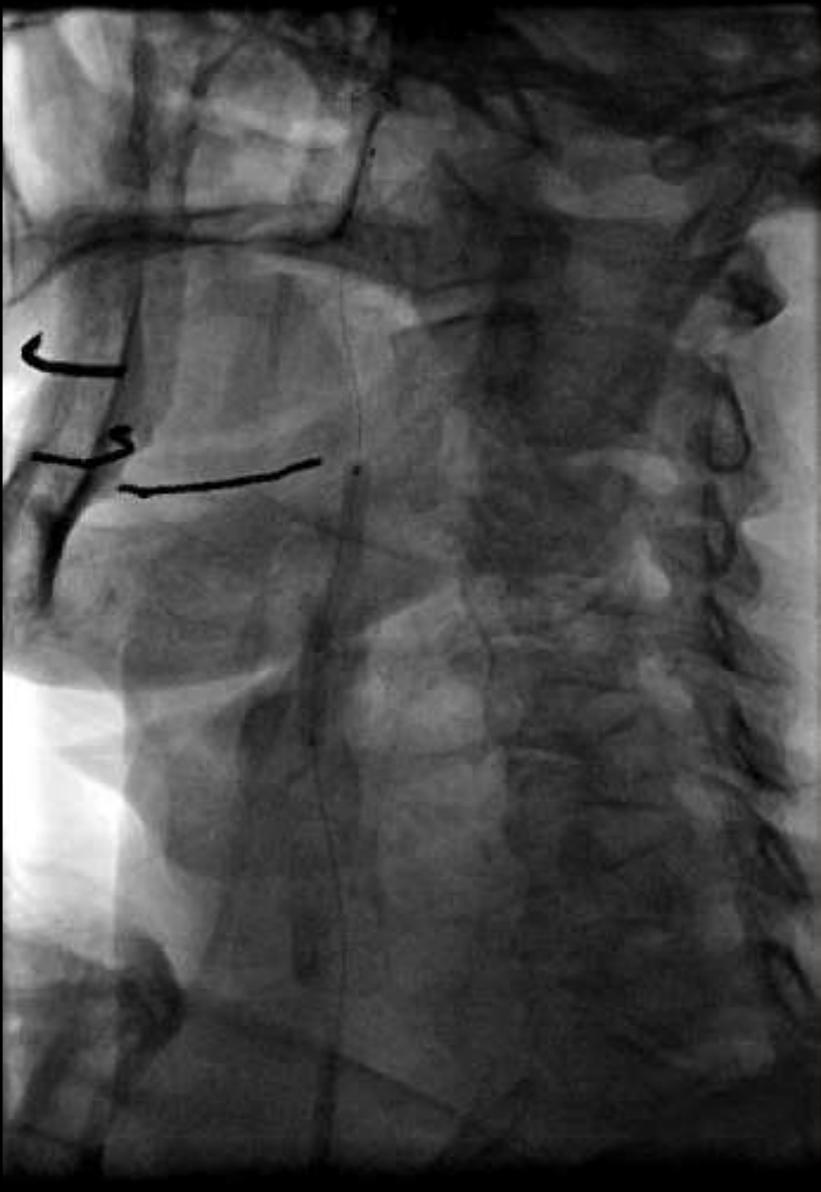


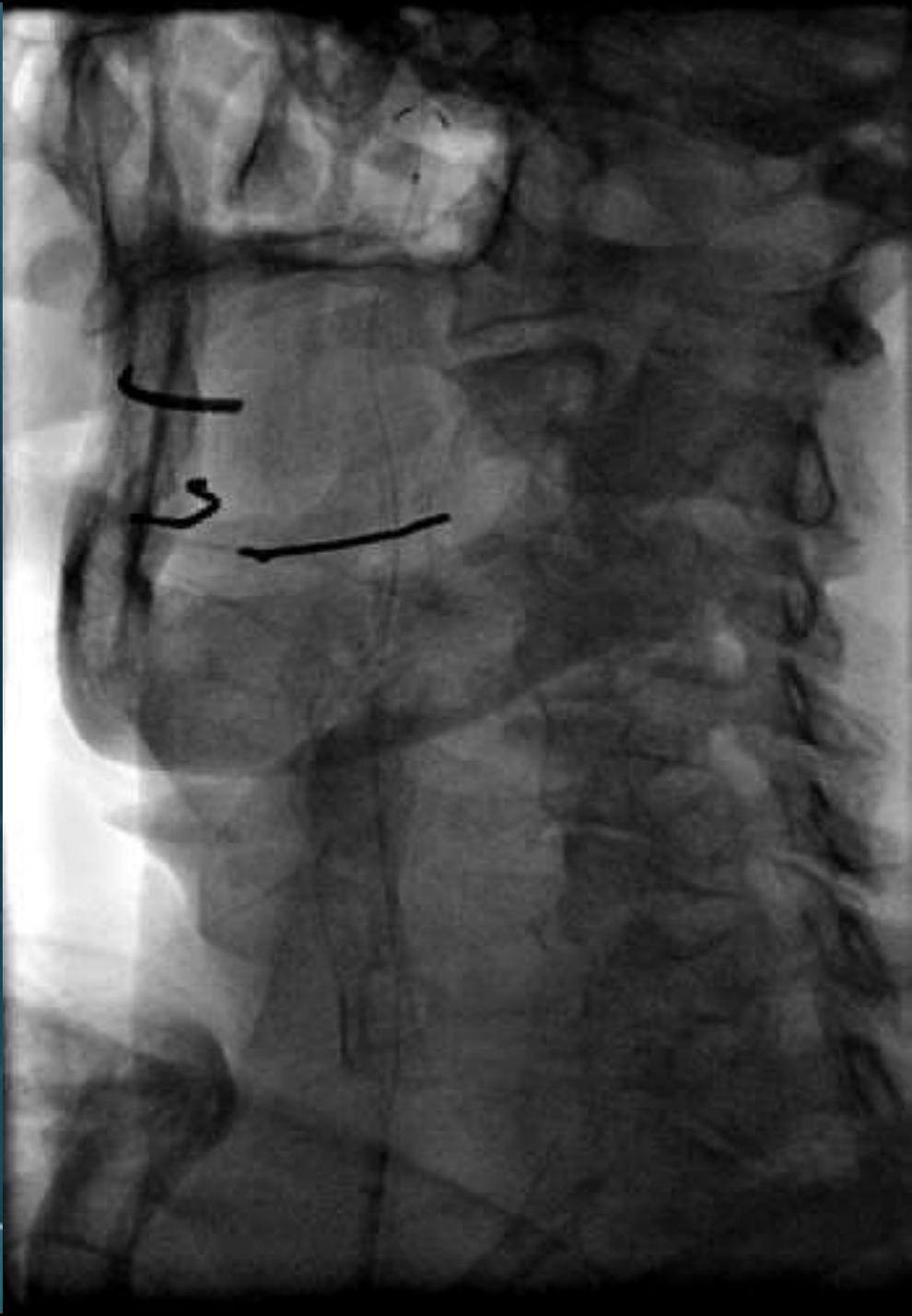


Right Carotid



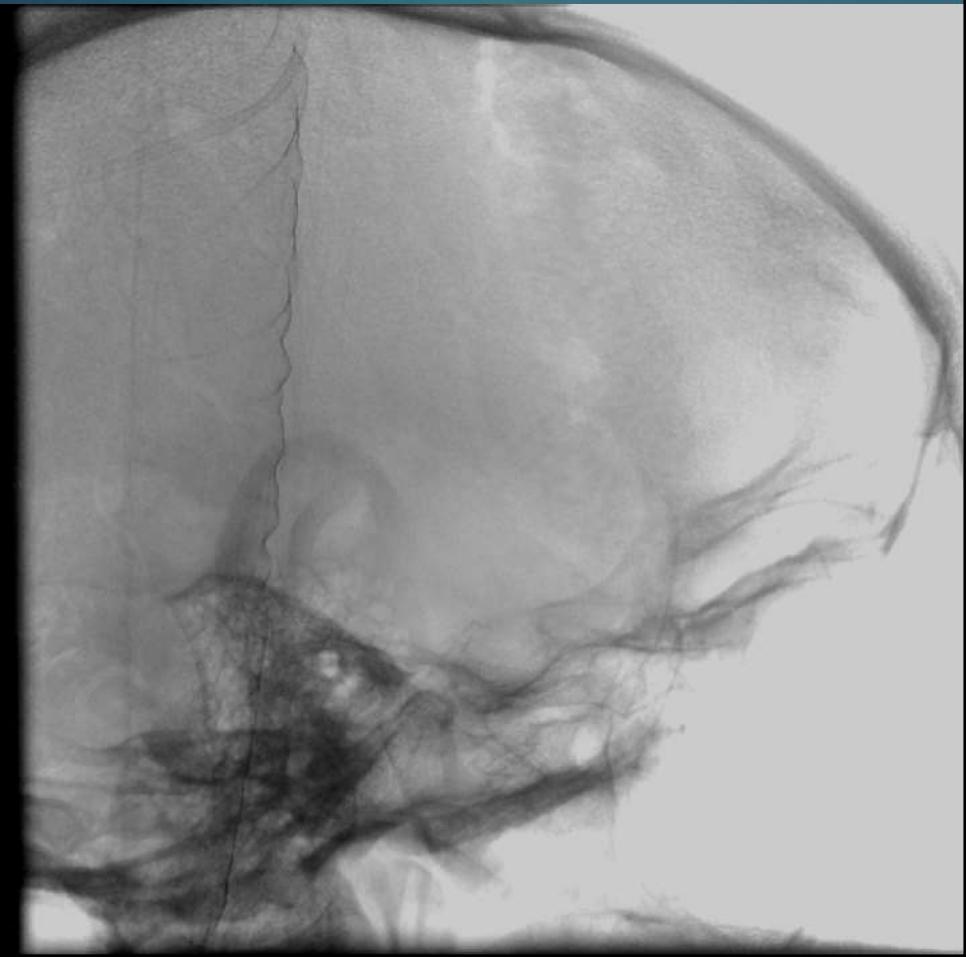






Final Pictures





Post Procedure

- Tolerated the procedure well. No hypotensive or bradycardic episodes.
- Transferred back to the floor.
- ASA/Plavix/Statin therapy

48 hours after

- Right arm started shaking and he suddenly stopped speaking. became tachycardic to 120-130's, hypertensive to 190/85, diaphoretic, aphasic, dysphasic and unable to follow commands, though he remained awake and alert.
- Per chart pt began speaking again after 30 min, but on exam has continued dysarthria, word finding difficulties, and telephoning speech.
- Transferred to Neurology Service. Their differential Diagnosis:
 - recurrent stroke,
 - seizures/status epilepticus,
 - “hyperperfusion syndrome”

- Started on Keppra
- **Repeat Carotid Doppler:** patent RICA stent, 1-39% LICA.
- **EEG:** No active seizures, possible epileptogenic potential, right hemispheric slow and attenuation.
- **MRI:** Tiny foci new DWI in right cerebellum and left frontal subcortical, but ***no major new infarction*** in right middle cerebral artery territory and no evidence edema to suggest hyperperfusion syndrome.
- Waxing and waning mental status for next 48 hours---probably post-ictal

● **Lessons Learned**

- **This was ultimately a CAS in a clinically asymptomatic patient**
 - **At high risk for event given asymptomatic embolic signature on brain imaging?**
- **Need for acute intervention not clear in retrospect**
- **Neurology probably should have elevated seizure diagnosis**

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