Educational Exhibit: Young Adult Stroke

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Learning Objectives

1. Define and recognize young adult stroke and challenges associated with this diagnosis
2. Recognize imaging findings in some of the etiologies of young adult stroke which are over represented relative to etiologies of stroke in the older adult population.
3. Select the appropriate follow-up and confirmatory imaging to establish diagnosis and etiology of young-adult stroke.

Young adult stroke definition

- Stroke clinical diagnosis: episode of acute neurological dysfunction presumably caused by ischemia or hemorrhage, lasting for at least 24 hours
- No official definition of young age group: most papers say < 45, < 55, or < 65 years of age
- Most studies suggest that the incidence of stroke in young adults is increasing
- Increase in traditional risk factors for stroke, e.g. hypertension, diabetes, hypercholesterolemia, obesity in young adults
- Hemorrhagic stroke: intraparenchymal hemorrhage, subarachnoid hemorrhage, etc.
- Ischemic stroke: large artery atherosclerosis, cardioembolic, small vessel occlusion, stroke of other determined cause, stroke of undetermined cause

Young adult stroke versus older adult stroke

- Young adult strokes account for 10-15% of all ischemic strokes
- Etiology for young adult strokes different, more likely to be thromboembolic, less likely to be large vessel atherosclerosis
- More likely to be stroke of other determined cause including unusual causes such as dissection, genetic diseases, hematologic disorders, inflammatory processes, drug use, and hypercoagulable states
- Peripartum state is also associated with increased risk of thrombotic events and stroke

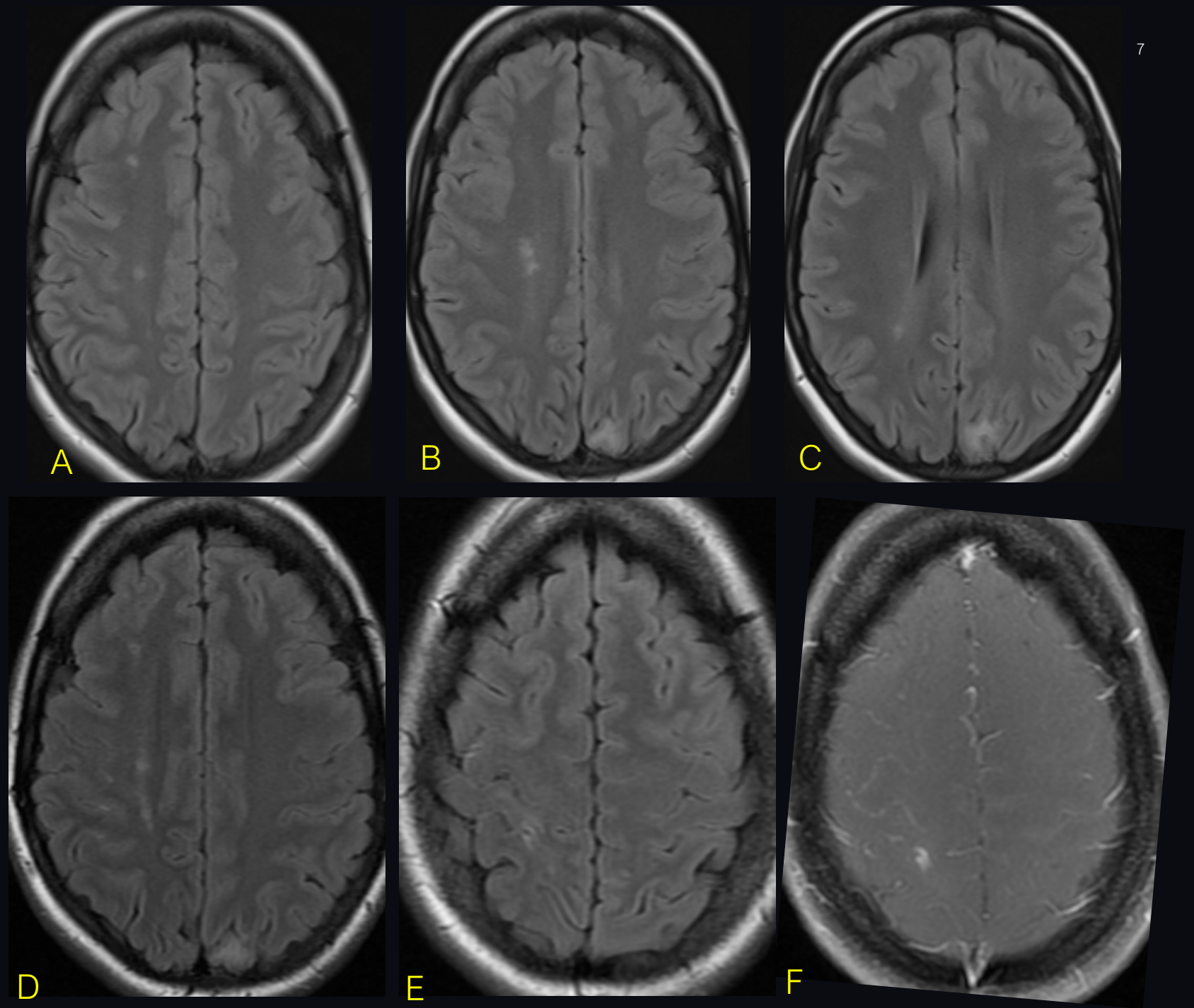
Difficulty in diagnosing young adult stroke

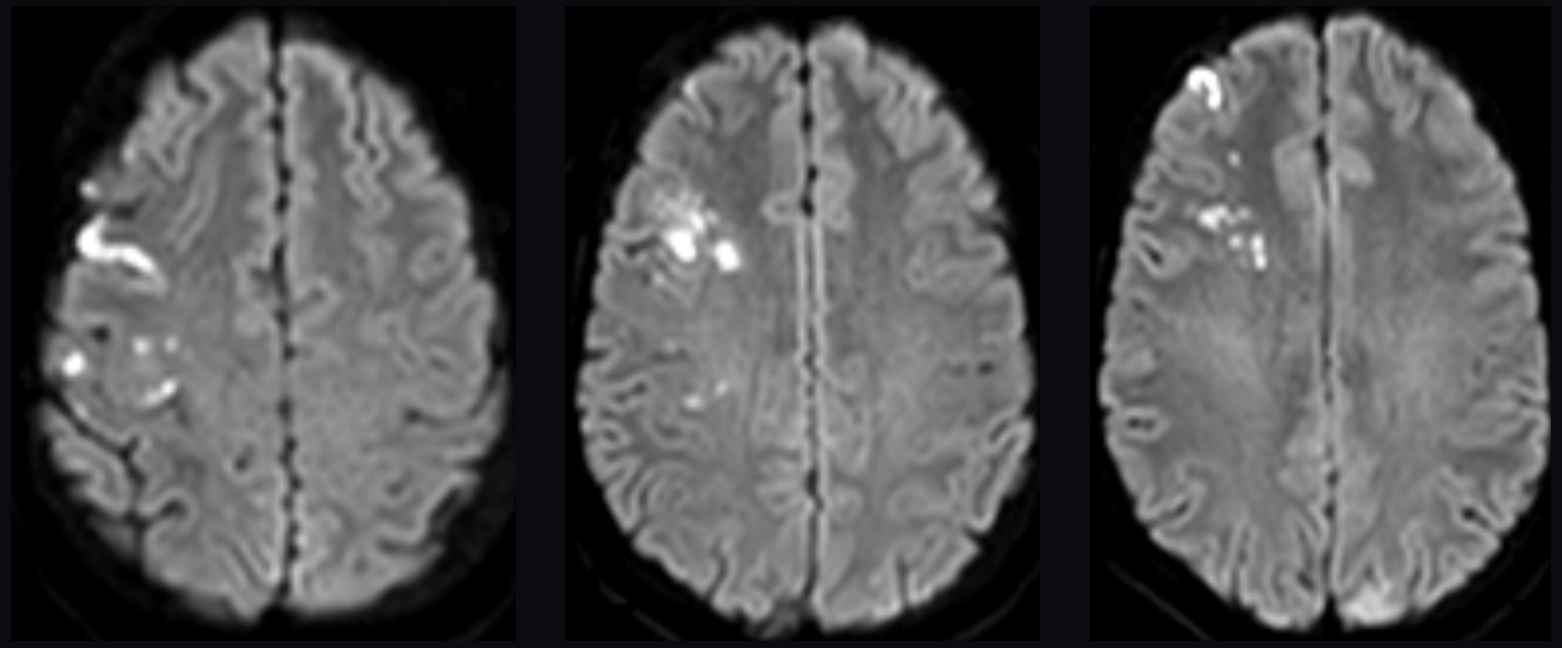
- Diffuse etiologies make diagnosis more difficult
- Often have atypical presentations such as vertigo and headache rather than dysarthria or lateralizing symptoms
- Age group is more likely to have other stroke mimics such as demyelinating disease
- Some papers site a clinician bias to fail to suspect ischemia or acute hemorrhage as a cause of neurologic symptoms in young patients which results in missed or delayed diagnoses in the young stroke patients
- More dire consequences for missing stroke in young patients as this can result in decades of disability when the patient would otherwise be at peak productivity

Case 1

- 31 yo who initially presented with left sided numbness at outside hospital.
- Patient later had a bout of what was diagnosed as Bell's palsy.
- The patient had FLAIR hyperintense signal in the centrum semiovale on MRI and was diagnosed with and treated for multiple sclerosis without positive oligoclonal bands.
- Patient had a subsequent bout of left sided numbness and weakness

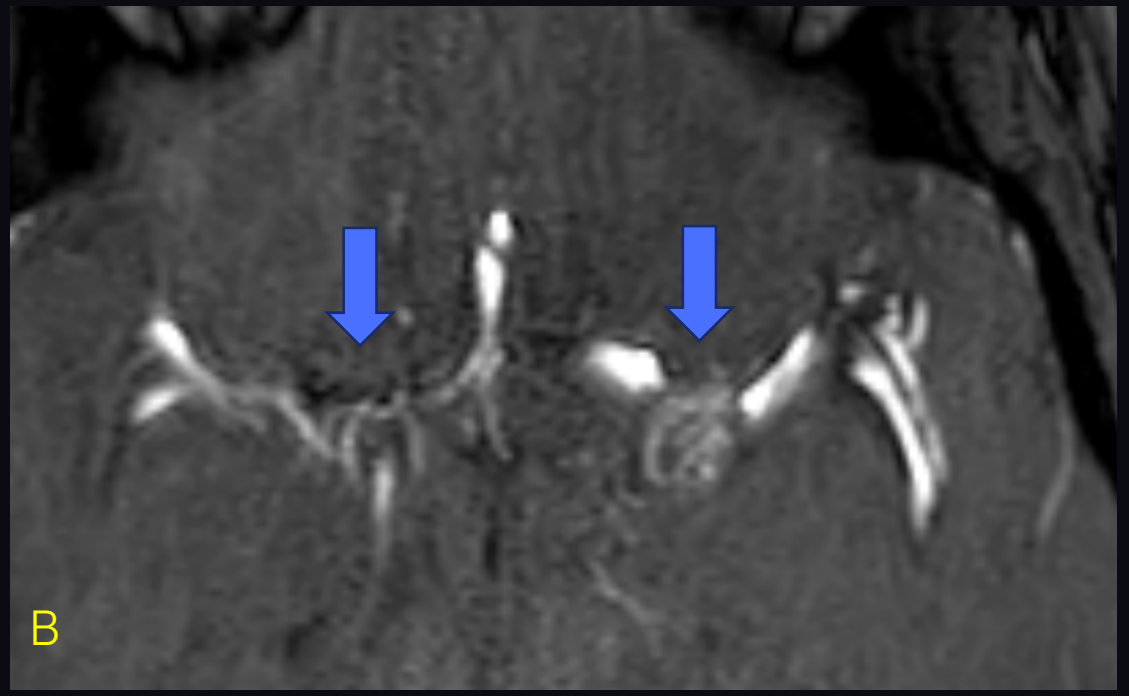
A,B,C: Initial outside images demonstrate FLAIR hyperintense foci in right centrum semiovale, described as representing demyelinating plaques, but nonspecific and more suspicious for FLAIR hyperintense signal from prior watershed infarcts.
D,E,F: Outside images 4 months later demonstrate more FLAIR hyperintense signal and subtle cortical FLAIR signal in the right convexity, likely in the precentral gyrus with linear enhancement suggestive of a subacute infarct.





Subsequent MRI 8 months from the initial images performed when the patient experienced a bout of left sided weakness and numbness demonstrates diffusion restriction scattered in the right MCA territory now diagnosed with an acute infarct.





3 month follow up MRA: A: 3D reformation demonstrates a likely severe stenosis in the right carotid terminus (orange arrow) and severe stenosis at the origin of the right M1 segment and moderate stenosis at the origin of the left M1 segment (Blue Arrows). B: MRA axial MIP demonstrates prominent lenticulostriate collaterals. Findings are consistent with a moyamoya pattern of stenosis and collateralization. The FLAIR hyperintense signal was likely related to remote watershed infarcts. No findings particularly concerning for multiple sclerosis.

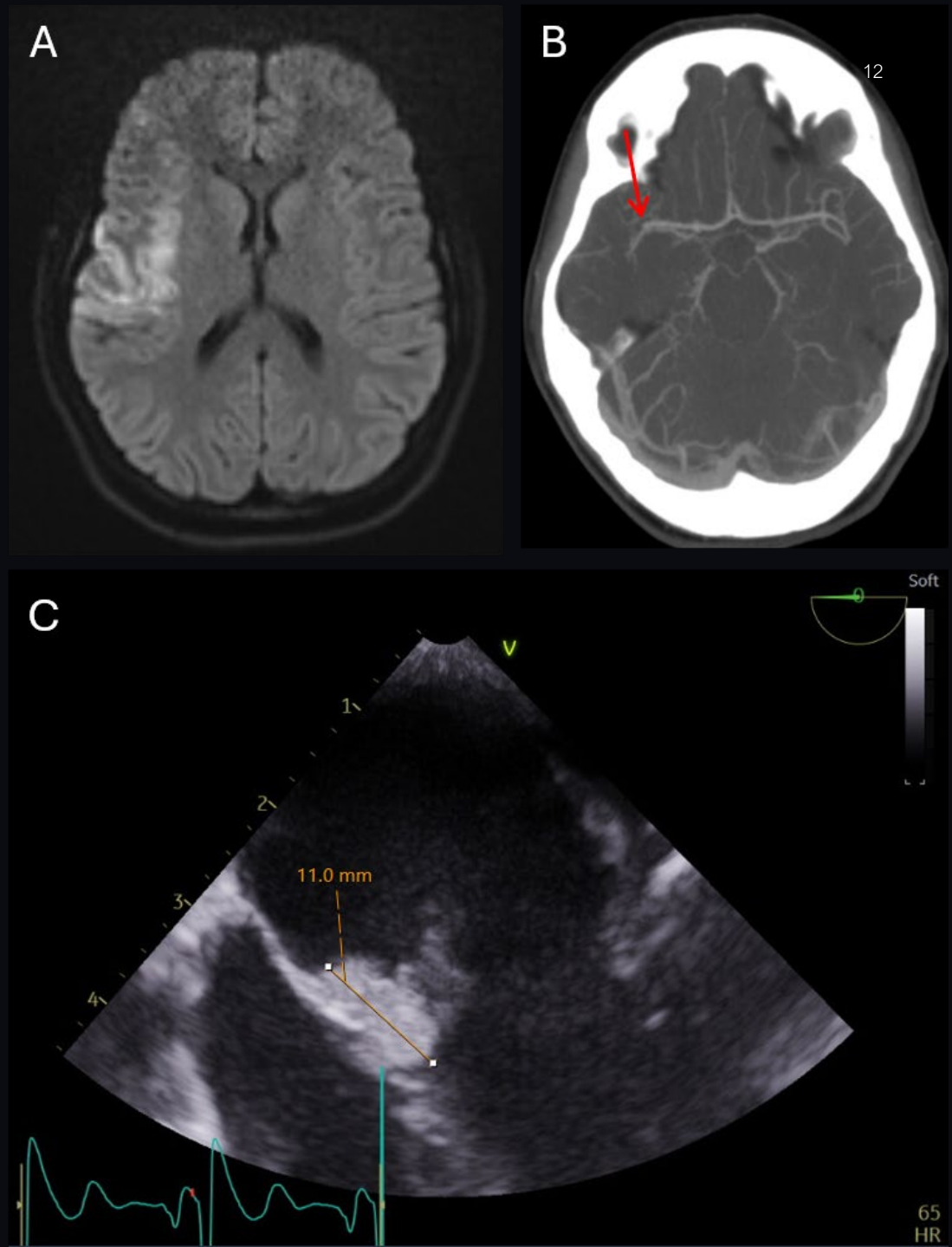
Key Takeaways: Moyamoya disease/syndrome(MMD)

- Moyamoya disease is characterized by severe stenosis of one or more of the vessels of the circle of Willis, most commonly the supraclinoid ICA with formation of lenticulostriate collaterals
- The disease is idiopathic and sometimes genetic
- Moyamoya syndrome has features of moyamoya disease but with an underlying associated disease process such as Neurofibromatosis Type 1, Sickle Cell Disease, and many other conditions
- MMD can cause recurrent ischemic strokes, especially watershed infarcts
- Remote deep watershed infarcts are FLAIR hyperintense located with a linear configuration in the deep white matter.
  - Sometimes these are confused with demyelinating plaques.
  - Demyelinating plaques will more typically be oriented perpendicularly to the ventricles and will have a more periventricular distribution
  - CSF studies can also confirm demyelinating disease.

Case 2

- 19 yo with slurring words, and mild L-sided weakness 1d prior to admission
- Parent initially presumed pt. to have allergic reaction, gave diphenhydramine
- Persistent symptoms the next morning, so patient presented to the ED
- PLT: 43 → 29 in the ED
- Initial head noncontrasted head CT negative

MRI (A) demonstrates diffusion restriction in the right MCA territory consistent with an MCA infarct. CTA (B) demonstrates occlusion at the origin of one of the right MCA M2 segments (arrow). TEE (C) demonstrates a mobile, 11-12mm mass on the atrial side of the anterior mitral leaflet. Infection labs were negative. Antiphospholipid antibodies were positive. Diagnosed with non-Bacterial Thrombotic Endocarditis.

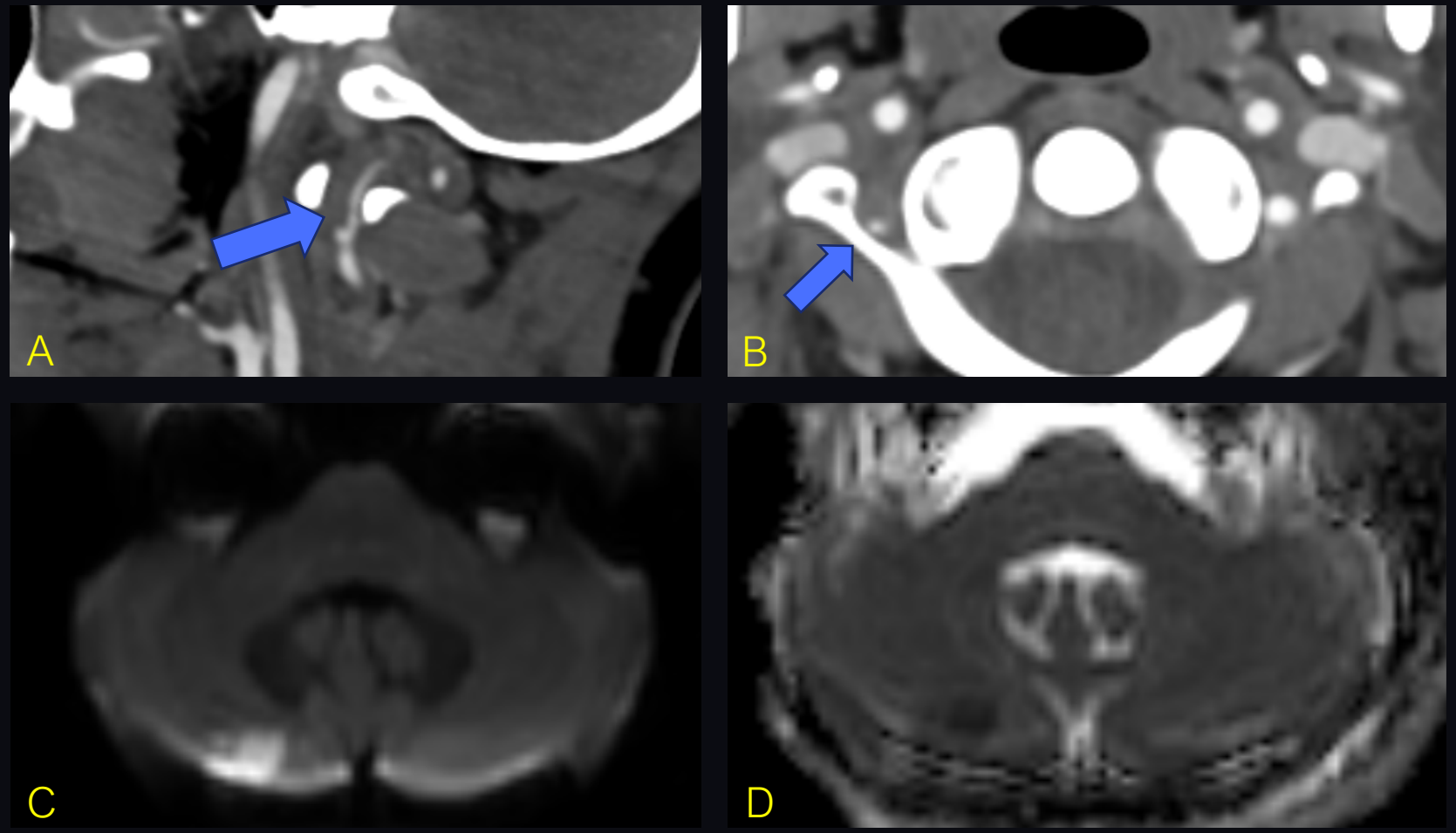


Key Takeaways: Antiphospholipid Syndrome/Non-Bacterial Thrombotic Endocarditis

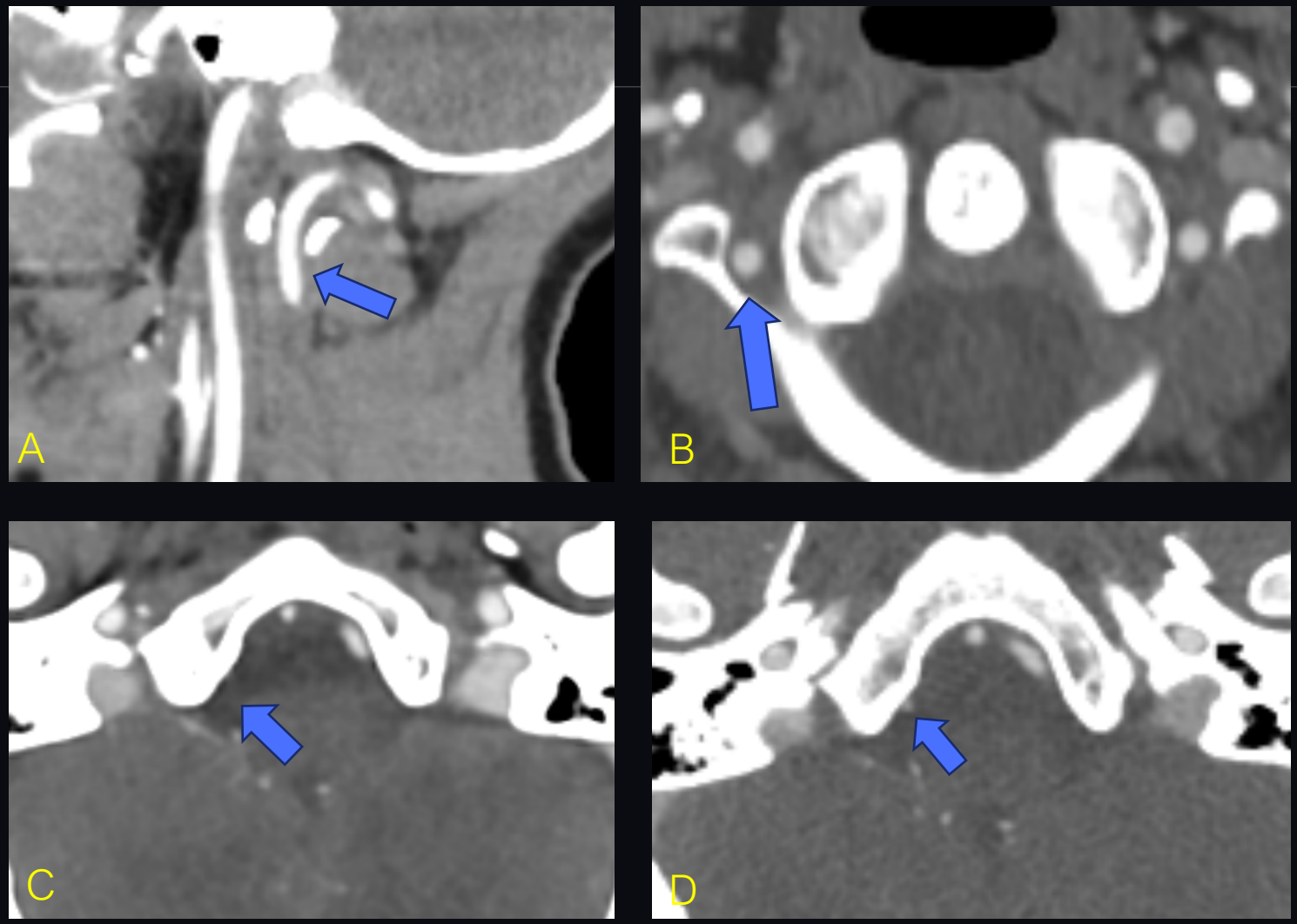
- Non-bacterial thrombotic endocarditis (NBTE):
  - *aka* Libman-Sacks, marantic, verrucous endocarditis
  - Systemic hypercoagulable state forms thrombus on heart valves
  - Main causes: cancer, autoimmune conditions
  - Also: DIC, sepsis, and burns also cause NBTE
  - Can give off emboli that cause strokes and infarcts in other organs
- Antiphospholipid syndrome (APS) characteristics:
  - Can cause thrombosis, vasculopathy, pregnancy complications
  - Can be associated with concurrent autoimmune process (Secondary APS) or independent (Primary APS)

Case 3

- 32 yo with intermittent dizziness,neck pain, and headache for 3 weeks which started after turning head during a carnival ride
- Subsequently developed acute worsening of neck pain and new gait imbalance after swinging a golf club and presented to the ED



Sagittal (A) and axial (B) CTA images demonstrate severe stenosis of the right cervical vertebral artery consistent with dissection. MRI demonstrates DWI hyperintesity (B) with ADC hypointensity (C) consistent with diffusion restriction from an acute infarct in the right cerebellum



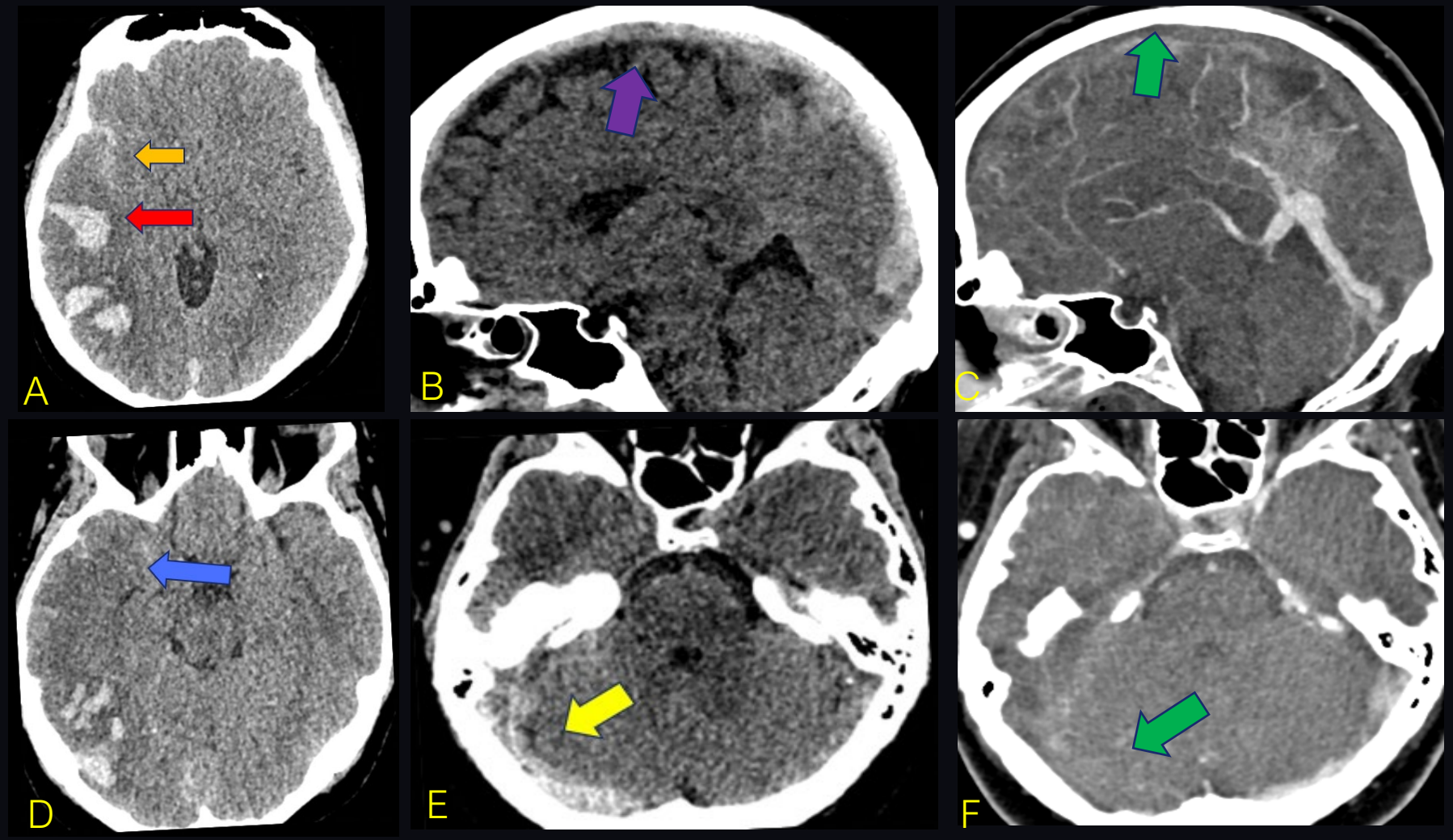
3 month follow up after patient was discharged on aspirin and clopidogrel. Sagittal (A) and axial (B) images demonstrate resolution of the previously demonstrated stenosis in the right vertebral artery. Axial image (C) from the initial CTA demonstrates near complete occlusion of the right posterior inferior cerebellar artery which is now apparent as the follow up axial CTA image (D) demonstrates resolution of the right PICA stenosis.

Key Takeaways: Vertebral Artery Dissection

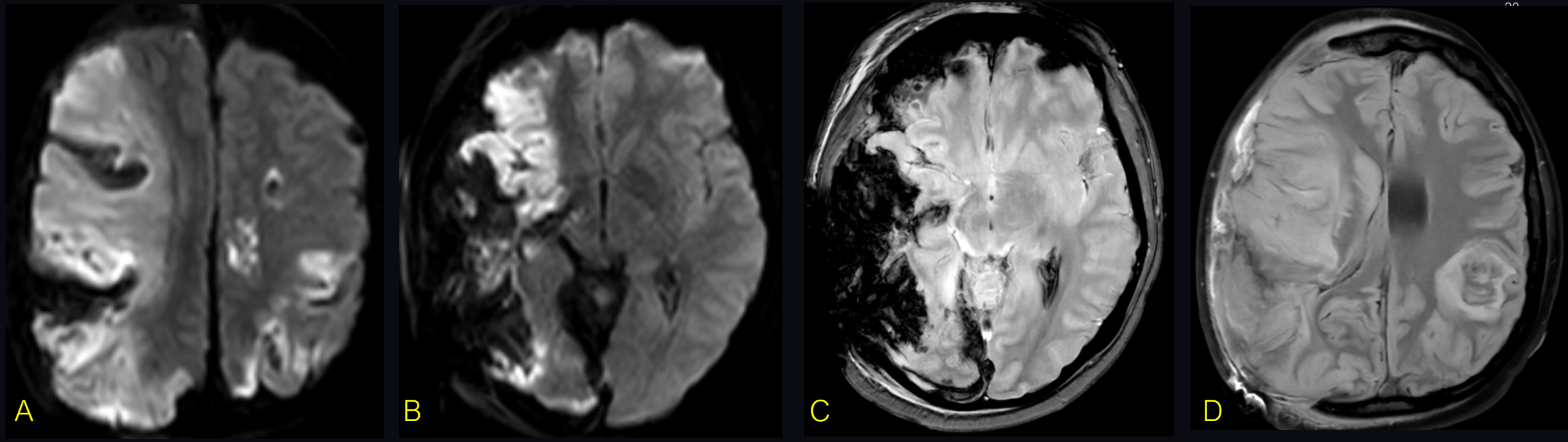
- Posterior circulation strokes are difficult to diagnose due to nonspecific, common symptoms such as vertigo and imbalance
- Dissections much more common cause of stroke in young adults than older adults
- New neck pain/headache + clinical suspicion/context → consider dissection
- Dissection can also happen in the internal carotid arteries which would case an anterior circulation stroke, especially after trauma.
- Often appear as abrupt caliber change in vessel on CTA, can also do MRA with fat saturated T1 sequence to look for hemorrhage in the false lumen.
- Dissections typically cause stroke by embolism, not by reduced flow
    - Check downstream branches and regions – PICA, cerebellum
- Dissections often recanalize within 3-6 months
    - Pseudoaneurysms can persist

Case 4

- 25 yo female with new, severe headache
- Presented to urgent care 3 days prior to admission, diagnosed with migraines
- Headache did not improve
- Developed seizure + altered mental status
- Presented to the ED



A,D: Noncontrasted head CT demonstrates intraparenchymal hemorrhage in the right temporal lobe (red arrow), subarachnoid hemorrhage (orange arrow) near sylvian fissure and generalized hypoattenuation in the right cerebral hemisphere (blue arrow) suspicious for edema. Sagittal (B) and Axial (E) noncontrasted head CT images demonstrate hyperattenuation in the superior sagittal sinus (purple arrow) and right transverse sinus (yellow arrow) suspicious for dural venous sinus thrombosis. Sagittal (C) and Axial (F) CTV images demonstrate lack of enhancement in these dural venous sinuses confirming thrombosis.



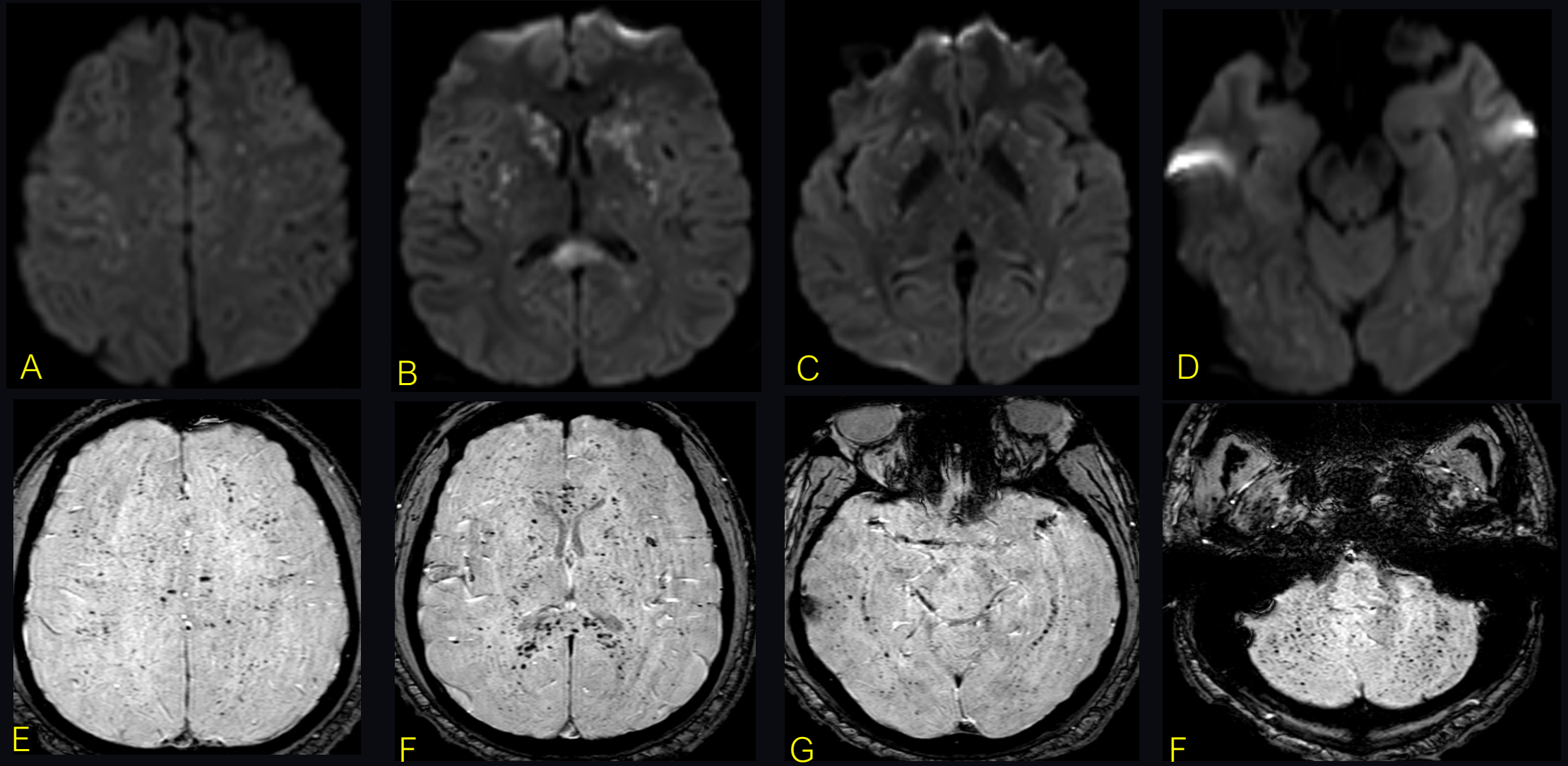
A,B: Diffusion weighted imaging from an MRI performed 2 days later demonstrates DWI hyperintensity in most of the right cerebral hemisphere confirming a large infarct which is increased in size from the prior exam CT. There are also smaller infarcts in the left cerebral hemisphere. C: Susceptibility weighted image demonstrates drop out of signal consistent with hemorrhage which is considerably increased, involving much of the right temporal lobe. This hemorrhage causes areas of decreased DWI signal due to susceptibility artifact. D: FLAIR sequence demonstrates the cytotoxic edema in the areas of infarct. There is also absence of a large portion of the right calvarium, indicating changes of a craniectomy which was performed to alleviate some of the intracranial mass effect caused by the infarcts and hemorrhage.

Key Takeaways: Dural Venous Sinus Thrombosis

- Dural venous thrombus is in the differential for first-ever severe headaches
- Hyperattuneating dural venous sinus or cortical vein should prompt CT or MR venography to exclude thrombus
- Venous infarcts do not follow arterial territories and are often associated with hemorrhage
    - Transverse sinus thrombosis classically associated with temporal lobe hemorrhage
    - Early edema/infarct related to a smaller dural venous thrombus may not have diffusion restriction
    - Venography pitfalls: arachnoid granulations, hypoplastic sinuses, and flow artifacts on MRV can mimic/hide thrombosis
    - Contrast-enhanced venography and an MR brain with volumetric postcontrast T1 imaging such as MPRAGE can provide better assessment for thrombus

Case 5

- 18 yo with SCD S-Beta thalassemia transferred initially from OSH for acute chest syndrome and acute hypoxic respiratory failure
- Had been complaining of worsening lower extremity pain prior to admission
- Initial CTA-head negative
- Developed altered mental status, declining neurological exam



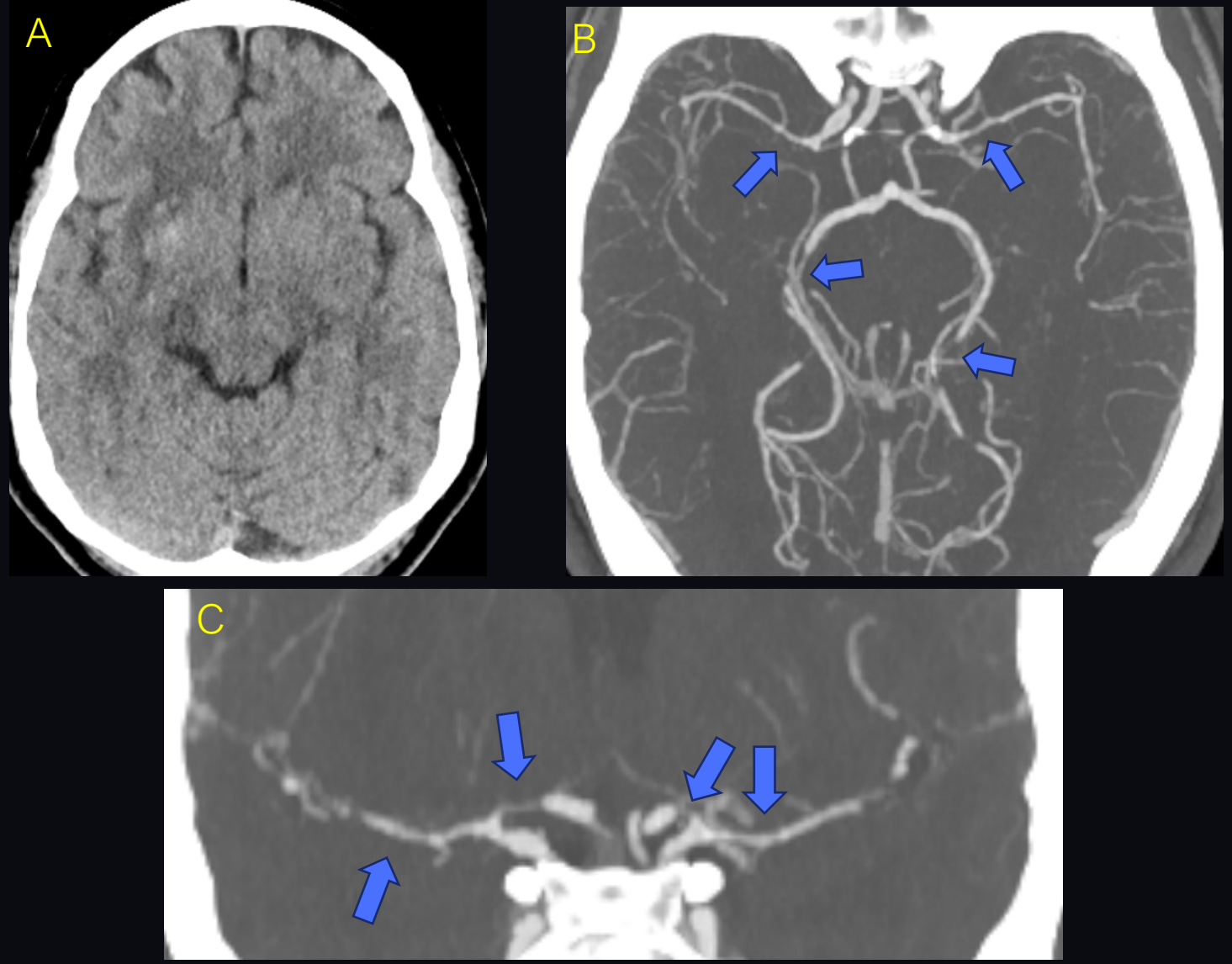
A-D: Diffusion weighted imaging in a subsequent MRI demonstrates innumerable tiny foci of diffusion restriction scattered in the brain. E-F: Heme sensitive sequence demonstrates innumerable tiny foci of drop out of signal scattered in the brain. Findings are consistent with fat emboli syndrome.

Key Takeaways: Sickle Cell Disease/Fat Emboli Syndrome

- Fat emboli syndrome caused by embolization of fat typically from bone marrow that lodges in small vessels of the brain.
  - More typically a complication of long bone fracture
  - Sometimes seen with acute sickle cell crisis presumed secondary to numerous bone infarcts
- Infarcts in children and young adults with sickle cell disease not related to fat emboli syndrome are common
- Moya-moya type stenosis is also associated with sickle-cell disease

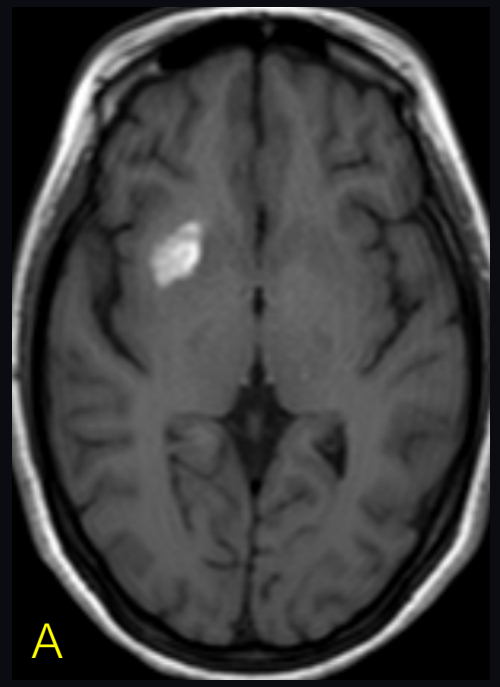
Case 6

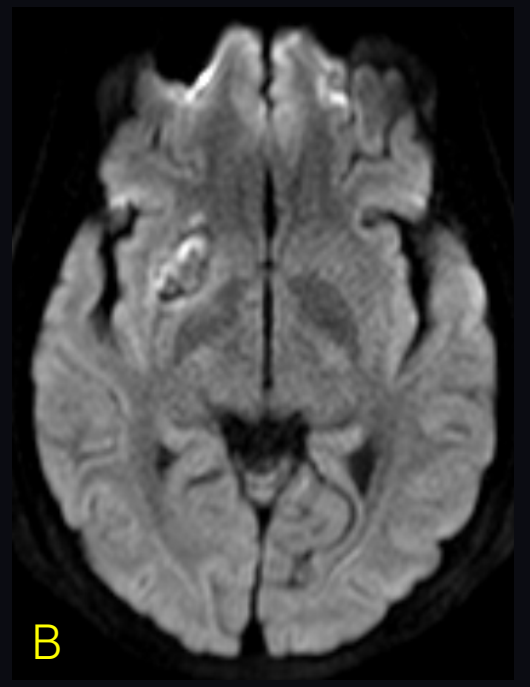
- 27yo 2 weeks postpartum with severe headache
- Abrupt onset, max intensity in ~2min
- Gradual, though minimal, improvement

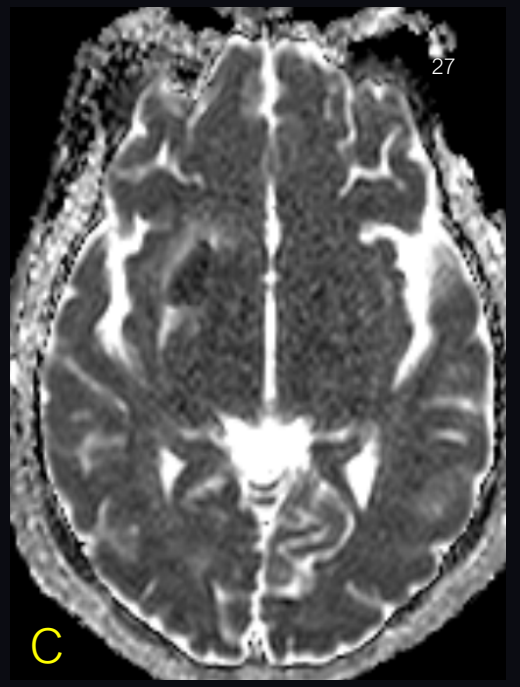


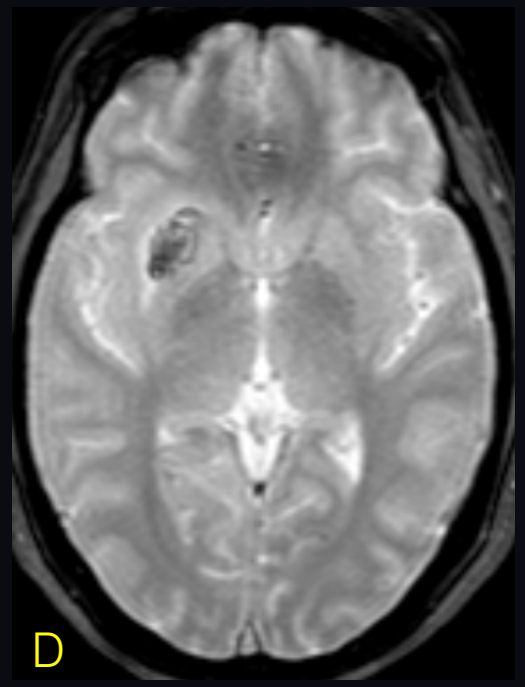
A: Noncontrast CT head demonstrates subtle increased attenuation in the right basal ganglia somewhat suspicious for a hemorrhage (likely subacute). B, C: axial and Coronal CTA MIP images demonstrate numerous severe stenoses in the intracranial arteries involving the bilateral anterior cerebral arteries, middle cerebral arteries and posterior cerebral arteries (arrows).

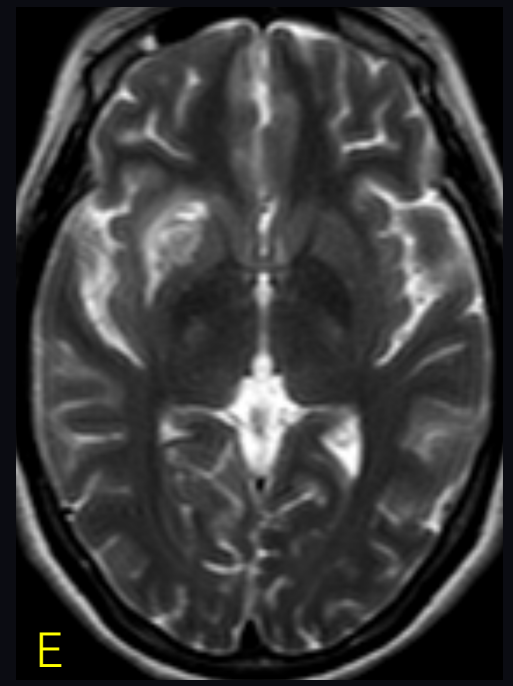
Subsequent MRI better demonstrates the intraparenchymal hemorrhage in the right basal ganglia. A: Axial T1 sequence demonstrates intrinsic T1 signal in the hemorrhage, suggesting that the hemorrhage is subacute. B,C: diffusion restriction in the hemorrhage, but this is most likely simply related to blood products as this corresponds to the T1 hyperintense signal. D: heme sensitive sequence demonstrates expected drop out of signal corresponding to the hemorrhage. E: T2 sequence demonstrates mild vasogenic edema surrounding the hemorrhage.



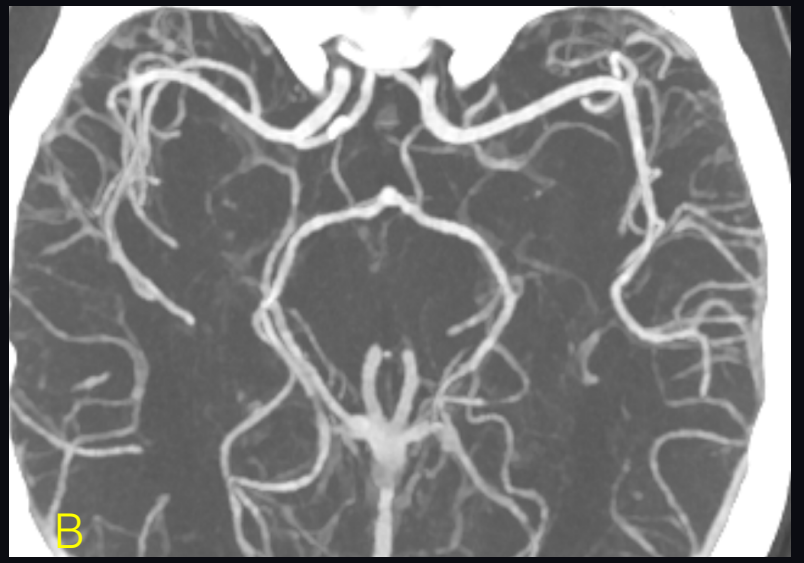














Patient was treated with verapamil. A: 6 week follow up head CT demonstrates developing encephalomalacia in the prior location of the intraparenchymal hematoma. B,C axial and coronal CTA MIP images demonstrate near complete resolution of the previously demonstrated stenoses. Patient ultimately diagnosed with post partum reversible cerebral vasoconstriction syndrome.

Key Takeaways: Reversible cerebral vasoconstriction syndrome (RCVS)

- RCVS often presents with sudden, severe “thunderclap” headache
- Associated with numerous triggers, peripartum state, eclampsia, sympathomimetic or vasoactive agents/medications, strenuous activity, and binge drinking.
- Classically presents with convexal subarachnoid hemorrhage with multifocal intracranial arterial stenoses which subsequently resolve
- Can also be associated with infarcts, intraparenchymal hemorrhage, or posterior reversible encephalopathy syndrome.
- Can have normal imaging. Initial CTA in particular can be normal. Digital subtraction angiogram is more sensitive.
- RCVS stenoses typically resolve within 3mo
- Peripartum patients are at particularly high risk for strokes and stroke-like pathology including RCVS, dural venous sinus thrombosis, ischemic infarcts and posterior reversible encephalopathy syndrome due to vascular changes in the peripartum period, increased thrombotic state, and also conditions such as ecplampsia.

Conclusion and Final Remarks

- YA stroke is sometimes misdiagnosed or has delayed diagnosis due to the myriad clinical challenges noted previously.
- Radiologists can help prevent this by having a high index of suspicion for stroke in young patients who have a suspicious history for stroke or suspicious imaging findings.
- Radiologists should also be aware of the conditions which are rarer for older patients but more frequently cause strokes in younger adults.
- Follow-up imaging is often critical to diagnosis
- Imaging findings often dictate treatment

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