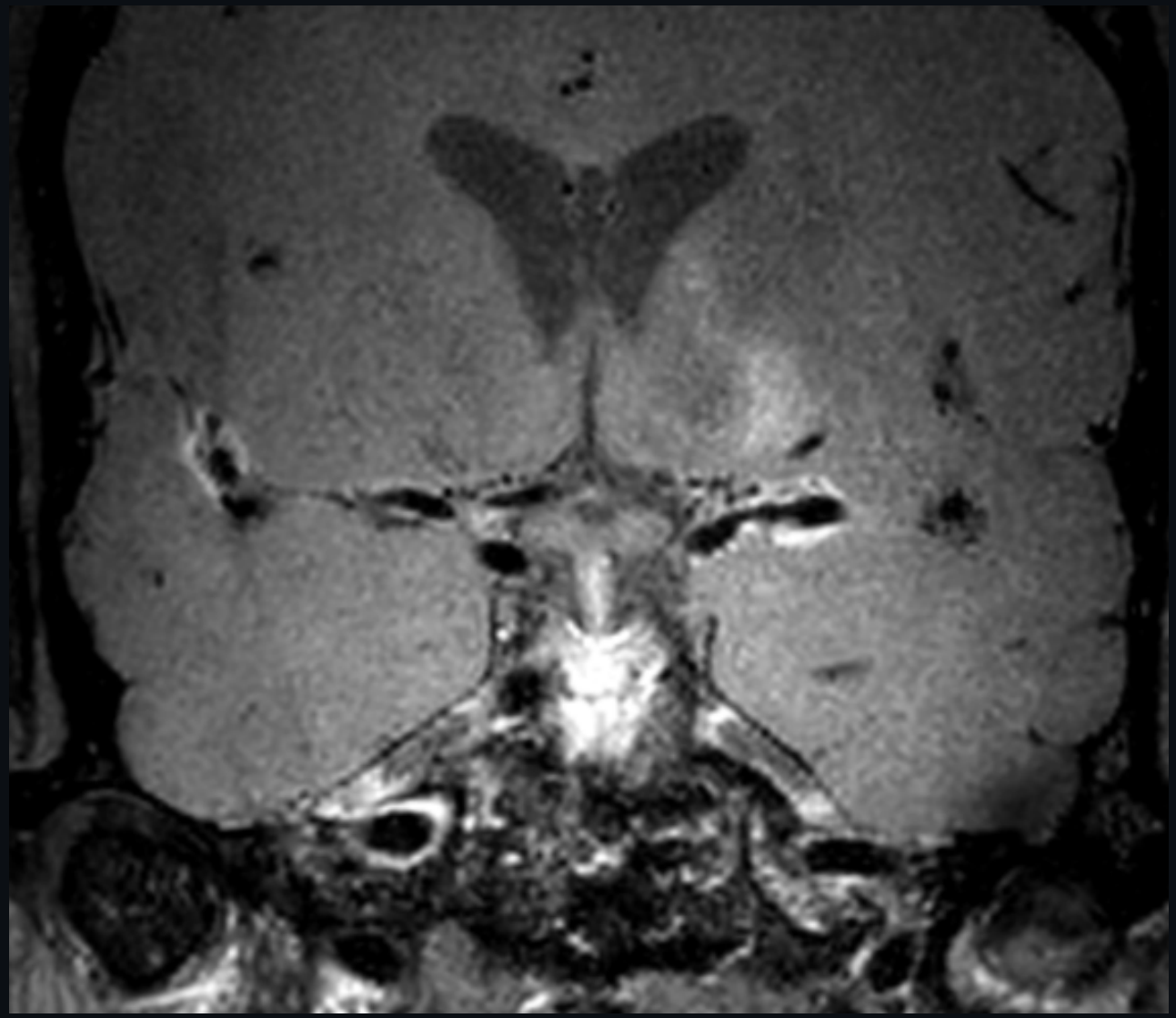
50TH ANNUAL SOUTHEASTERN NEURORADIOLOGICAL SOCIETY (SENRS) MEETING · INTERESTING CASE

Meningovascular Neurosyphilis with Vessel-Wall Imaging Correlate



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CLINICAL PRESENTATION

37-year old male presented to the emergency department with **fever** and **altered mental status.**

PAST HISTORY

Untreated HIV (CD4, viral load unknown).
Substance use disorder.

EXAM

No focal neurologic deficit, however **encephalopathic** with limited attention and “odd answers to questions.”

INITIAL HEAD CT



Left basal ganglia hypoattenuation concerning for subacute to chronic infarct.

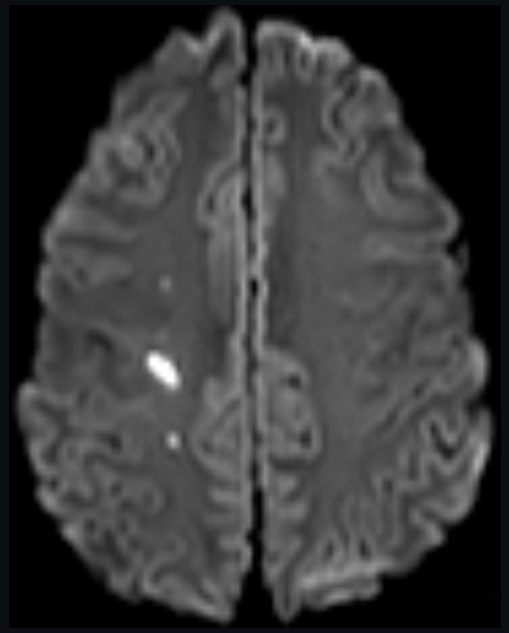
The neurology team felt that this finding did not explain the patient’s encephalopathy or current presentation, particularly given patient was high-risk for intracranial infection.

Further evaluation with contrasted MRI was recommended.

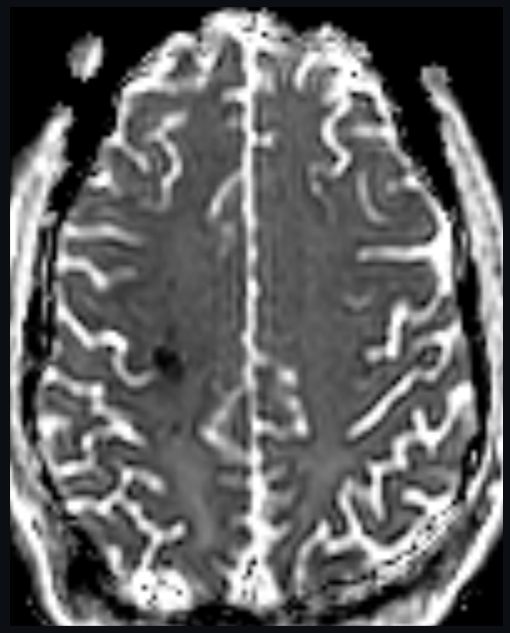
IMAGING DISCUSSION

INITIAL MRI WITH AND WITHOUT

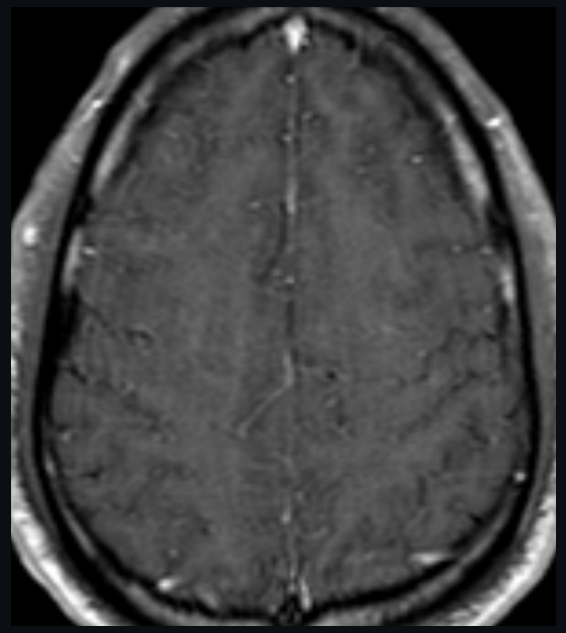
- Right vascular border (ACA-MCA) zone infarcts (top row).



DWI

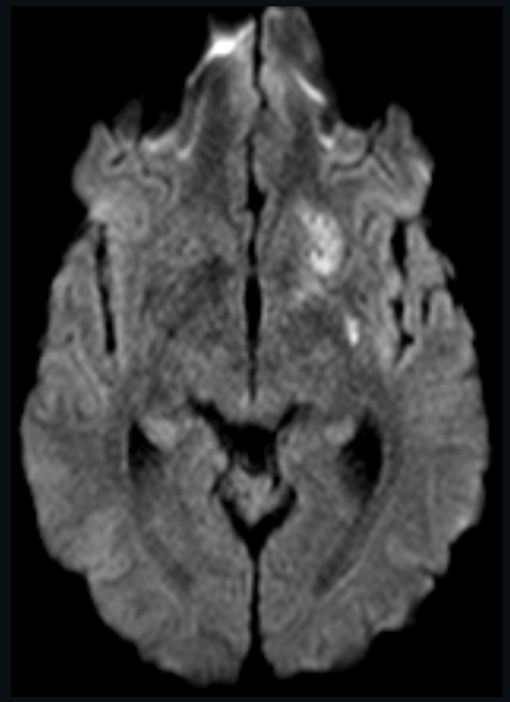


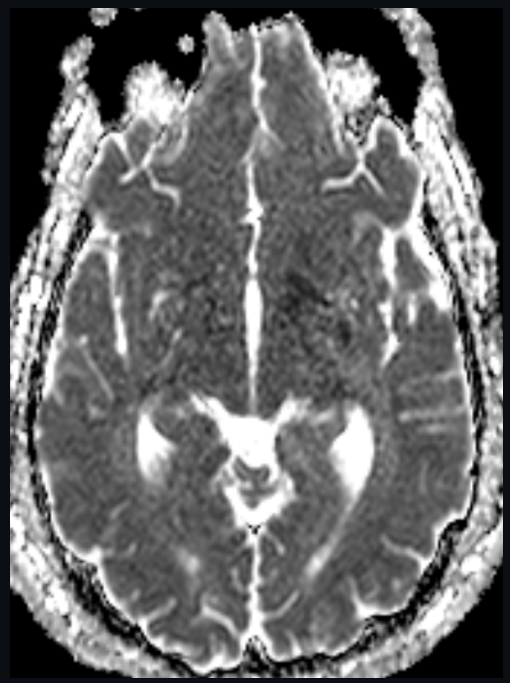
ADC

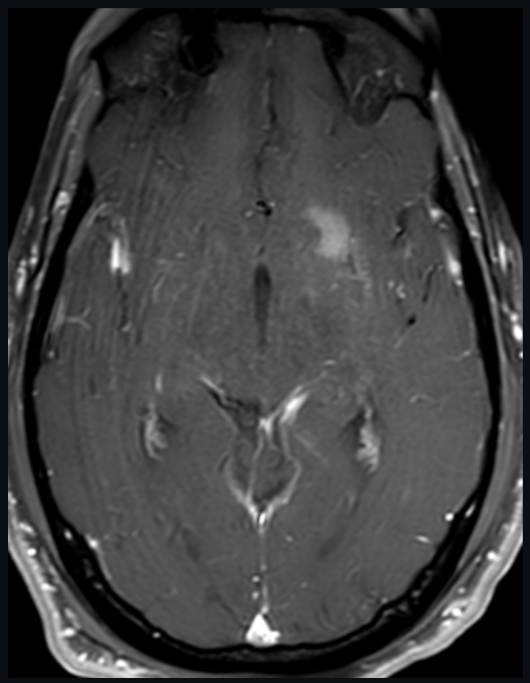


T1+C

- Left basal ganglia subacute infarct versus cerebritis given mild enhancement (bottom row).





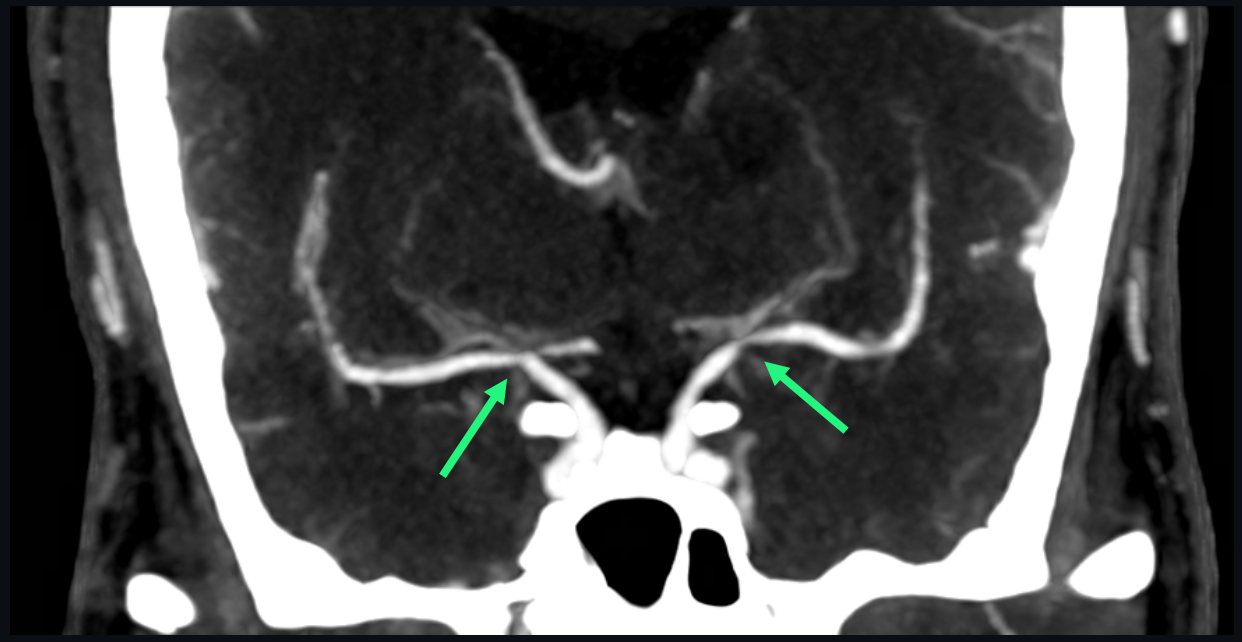


Infarcts in multiple, bilateral vascular territories suggests either **embolic phenomenon** or **diffuse/multifocal arteriopathy**.

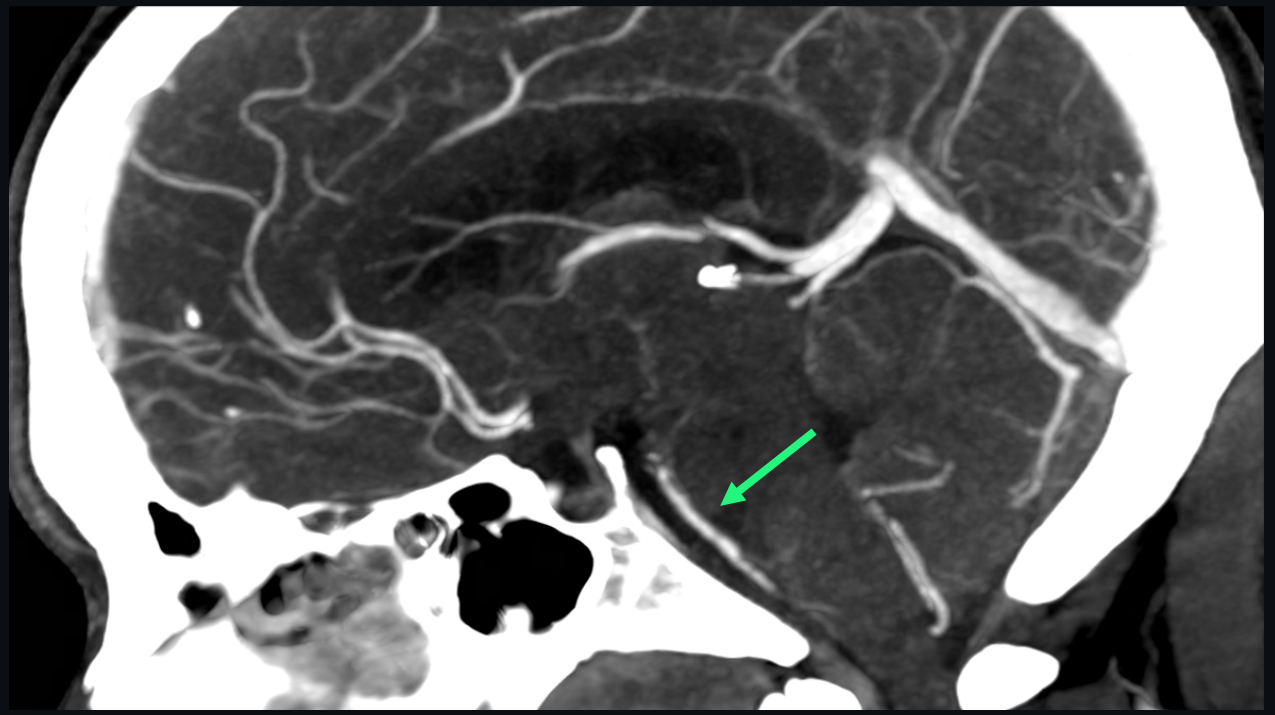
IMAGING DISCUSSION

CTA

- Multifocal intracranial arterial stenoses
  – left greater than right ICA terminus/proximal MCAs, basilar.



Coronal (above) and Sagittal (below) MIP



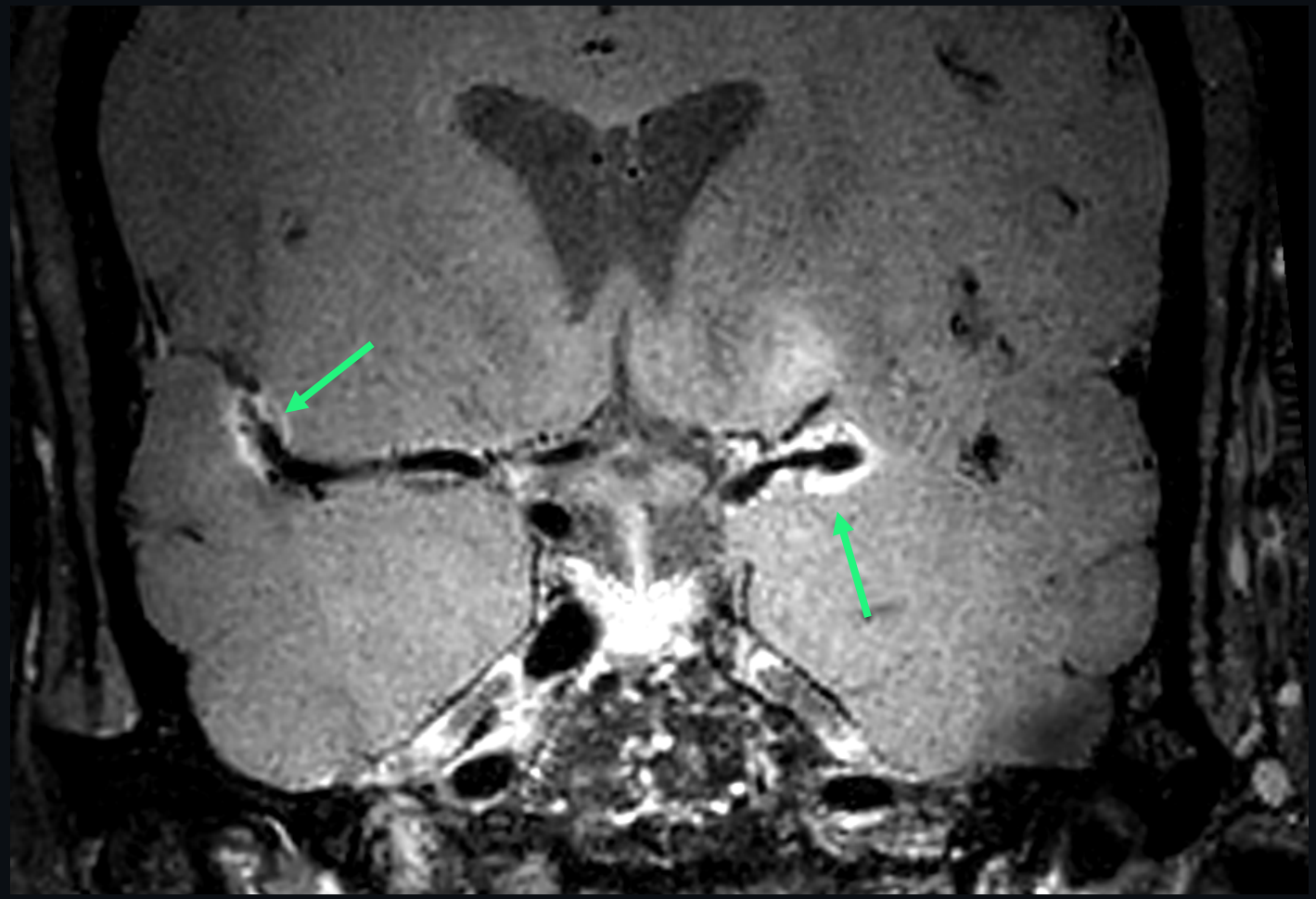
The demographic is less typical of atherosclerotic disease (adult <40). Furthermore, there was no evidence of atherosclerosis of the arch or vessels of the neck.

Hence, **vasculopathy/vasculitis** became the most likely consideration.

IMAGING DISCUSSION

MRI WITH VWI (HOSPITAL DAY 4)

- Concentric vessel wall enhancement in areas of stenoses confirming vasculitis.



Coronal Post-Contrast Vessel Wall Imaging (VWI)

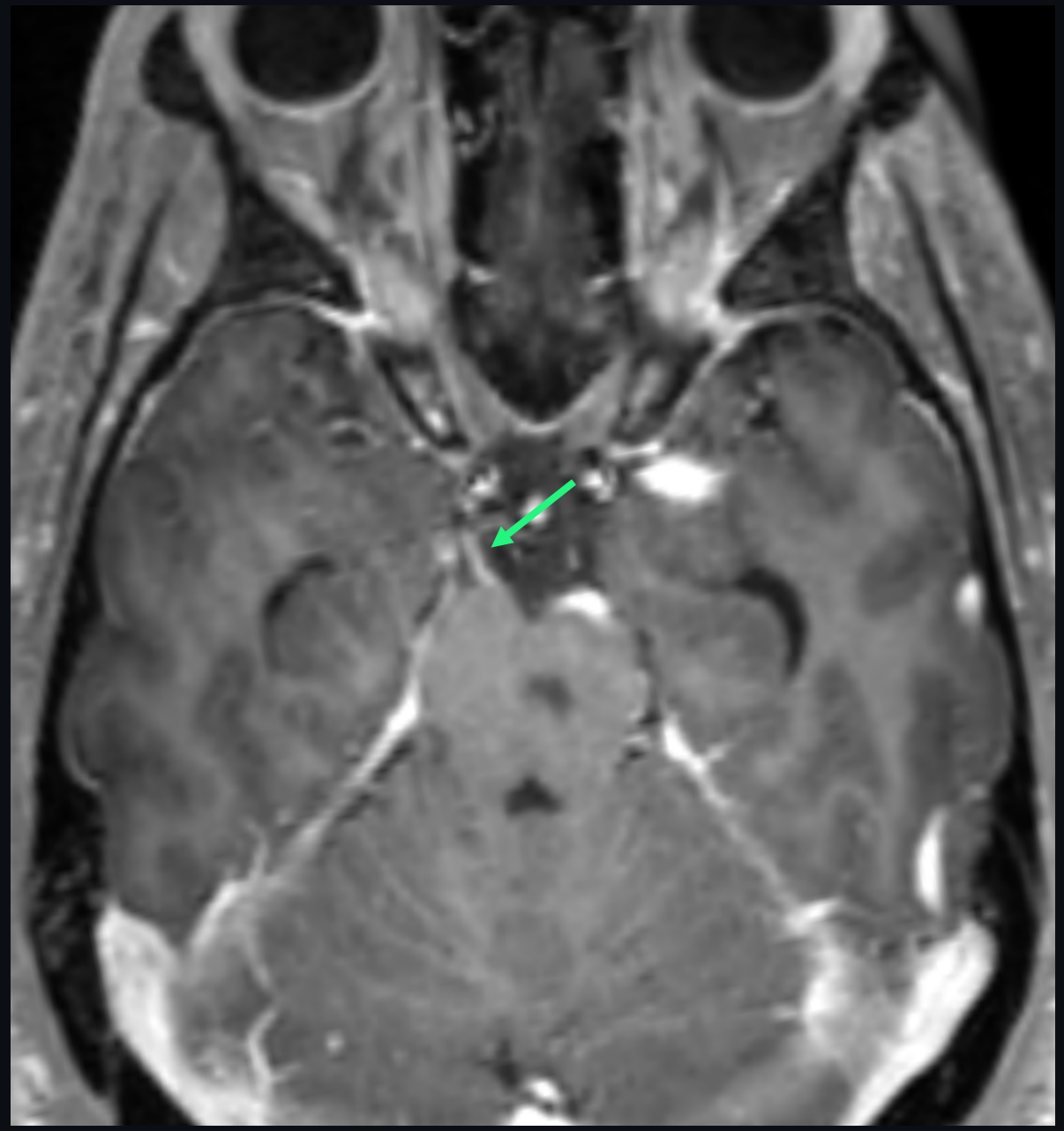
Vessel wall imaging involves suppression of signal in blood and CSF, with post-contrast acquisition allowing evaluation for vessel wall enhancement.

Concentric vessel wall enhancement supports a diagnosis of vasculitis versus non-inflammatory arteriopathies.

IMAGING DISCUSSION

MRI WITH VWI (HOSPITAL DAY 4)

- Enhancement of the right oculomotor nerve (CN III).

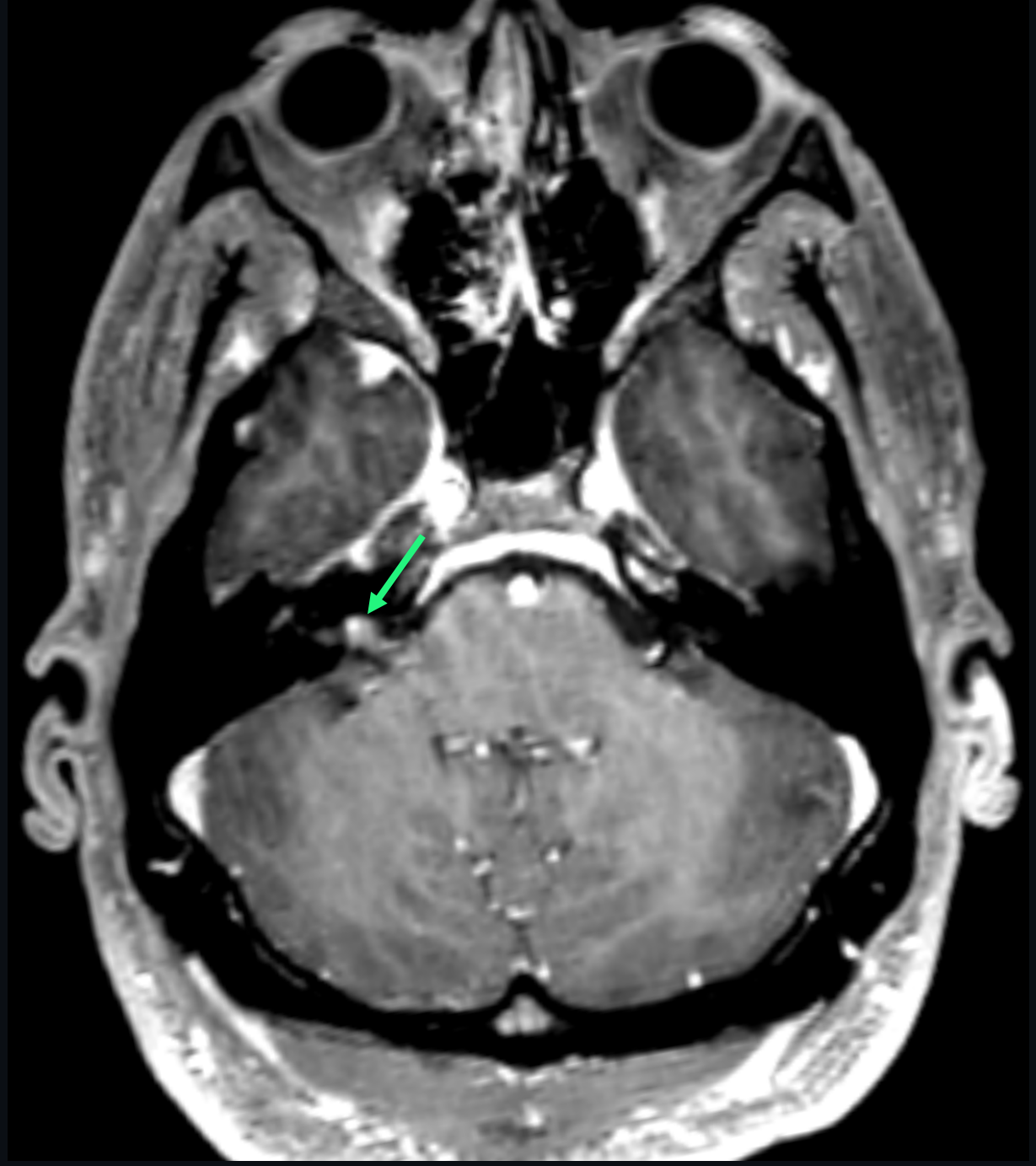


Cranial nerve enhancement is suggestive of basilar meningitis versus granulomatous/infiltrative disease. This raises concern for atypical infections such as neurosyphilis, tuberculosis, or cryptococcus.

IMAGING DISCUSSION

MRI WITH VWI (HOSPITAL DAY 4)

- Enhancement of the right facial/vestibulocochlear nerves.



Syphilis most commonly involves the facial/vestibulocochlear nerves first.

IMAGING DISCUSSION

Imaging timeline/summary

Day 0

CT head (ED)

Left basal ganglia infarct

Day 1

MRI brain

Bilateral infarcts in different vascular territories

Day 1

CTA head and neck

Multifocal intracranial stenoses

Day 4

MRI brain + VWI

Circumferential vessel wall enhancement with stenosis; cranial nerve enhancement

Day 18

Follow-up MRI

Improved enhancement and edema; expected infarct evolution

IMAGING DISCUSSION

Differential diagnosis

Based on these imaging findings, the differential diagnosis included meningovascular processes producing vasculitis, infarcts, and with cranial nerve involvement.

Cryptococcal meningoencephalitis with vasculitis	Favored on early imaging in an immunocompromised host (cystic basal ganglia lesions, meningeal and cranial nerve enhancement). Serum and CSF cryptococcal antigen negative.
Meningovascular neurosyphilis	Multifocal infarcts, vessel wall enhancement with stenosis, and cranial nerve enhancement. Reactive treponemal IgG (RPR 1:512) and reactive CSF VDRL.
Other opportunistic infection	Considered given HIV status. Toxoplasma IgG/IgM, QuantiFERON, and hepatitis B/C testing negative.
CNS lymphoma or toxoplasmosis	Less likely given the relative paucity of enhancement.
Atherosclerotic or embolic infarcts	Atypical for age and distribution; no arch or cervical atherosclerosis and no PFO on TTE.

MANAGEMENT

1:512	1:4	101,000	245
Serum RPR	CSF VDRL (reactive)	HIV viral load	CD4 count

DIAGNOSTIC WORKUP

- Given HIV status, infectious processes were considered most likely, specifically syphilis, cryptococcosis, and other opportunistic infection
- Serum treponemal IgG reactive
- CSF: reactive VDRL, 21 nucleated cells, protein 96 mg/dL, glucose 36 mg/dL
- Negative: serum and CSF cryptococcal antigen, Toxoplasma IgG/IgM, hepatitis B/C, QuantiFERON

TREATMENT

- Empiric meningitis coverage on presentation: vancomycin, ceftriaxone, ampicillin, acyclovir
- IV penicillin G, 24 million units/day for 14 days, followed by IM penicillin 1-time dose 2.5 million units
- Outpatient infectious disease follow-up to start antiretroviral therapy

OUTCOME

Imaging response

Follow-up MRI (Day 18) showed interval improvement of enhancement with decreasing edema and expected evolution of the multifocal infarcts.

Clinical status

Mild expressive aphasia and cognitive-communication deficits (MoCA 18/30), attributed to the multifocal strokes and meningitis.

Follow-up

Outpatient infectious disease follow-up to start antiretroviral therapy – delayed to lower risk of Immune Reconstitution Inflammatory Syndrome (IRIS)

Take home points

1. CNS invasion occurs in about 40% of syphilis cases and can happen at any stage; HIV co-infection tends to accelerate progression.[1]

2. Meningovascular syphilis is an arteritis of small and medium arteries that leads to ischemic strokes; about 10% of patients with syphilitic CNS involvement present with stroke.[1]

3. MRI and VWI show vessel wall thickening and enhancement with infarcts in any vascular territory, with or without meningeal and cranial nerve enhancement.[2]

4. Imaging overlaps with opportunistic infections such as cryptococcosis: in an immunocompromised patient with unexplained stroke or vasculitis, send treponemal serology and CSF VDRL.

1. Kano S, Inoue A, Masuoka S, et al. Clinical and Imaging Features of Syphilis from Head to Toe. RadioGraphics. 2025;45(6):e240170.
2. Neurosyphilis. Radiopaedia.org. radiopaedia.org/articles/neurosyphilis