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# Caught in the (Carotid) Web

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## Rhode Island Stroke Symposium

# Financial Relationship Disclosure(s)

**Farhan Khan, MD, MS**

- Nothing to disclose





THE WARREN ALPERT  
**Medical School**  
BROWN UNIVERSITY

# Caught in the Carotid Web: Pathophysiology, Diagnostic Challenges, and Treatment Options

Farhan Khan  
09/05/2025



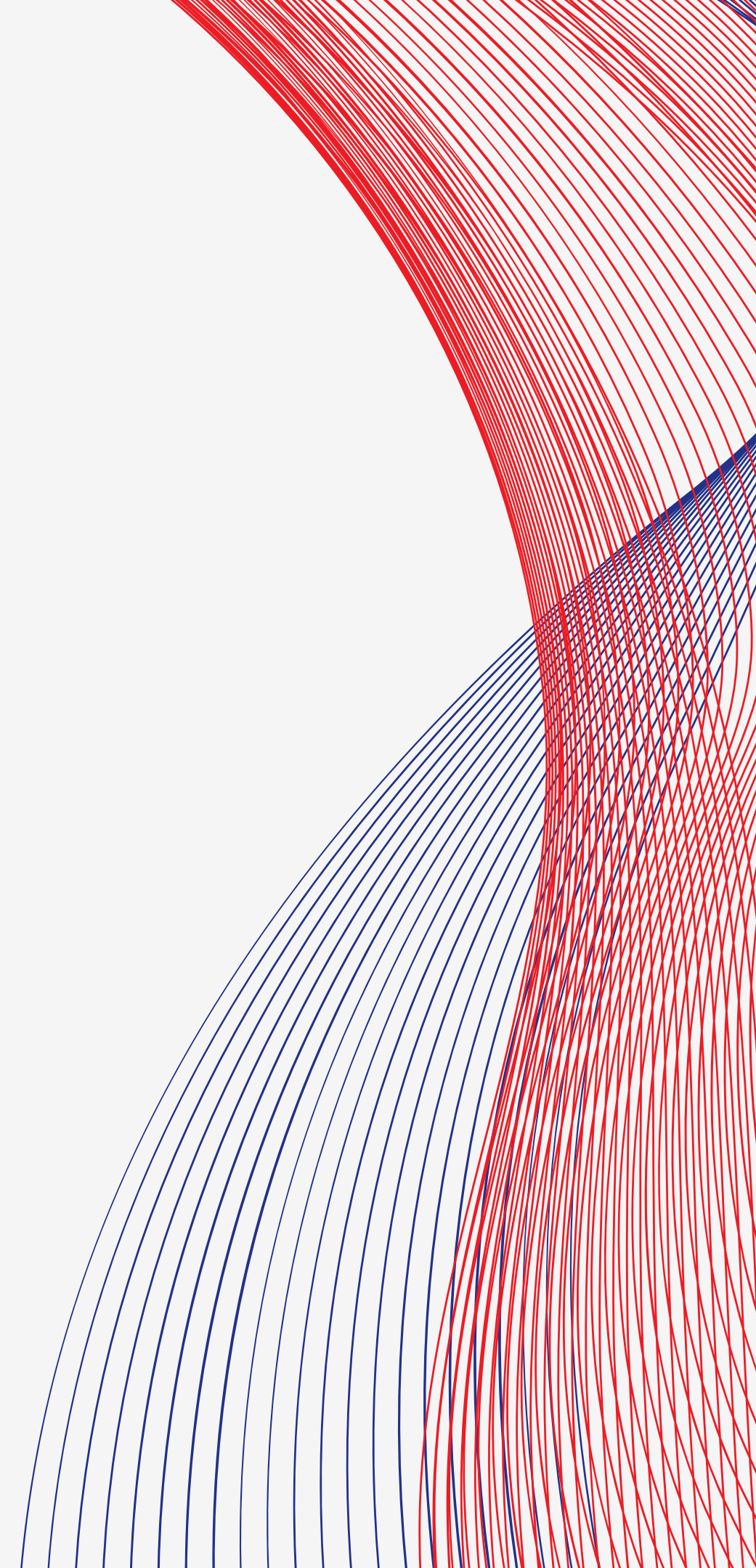
# DISCLOSURE

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- No financial relationships to disclose

# Outline

- Background
- Epidemiology
- Diagnosis & Imaging
- Pathophysiologic Mechanisms
- Morphologic & Hemodynamic Features
- Diagnostic Challenges and case presentations
- Management and Practice Patterns
- Future Directions





# Background

- Described by Rainer et al. in 1967
  - Patient with nocturnal episodes of R hemiparesis
  - Dynamic angiography
  - Treated with surgical resection
- Bulb, diaphragm, shelf-like structure
- Morgenlander and Goldstein described case of recurrent TIAs and ipsilateral CW

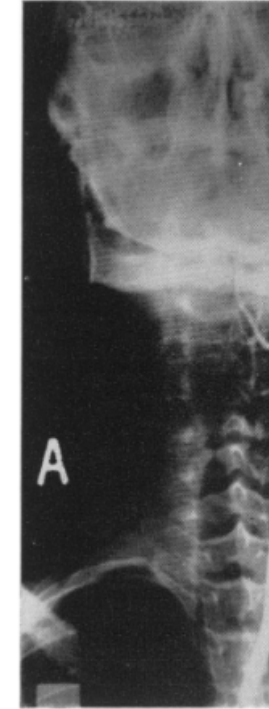
## Fibromuscular Hyperplasia of Causing Positional Cere

W. GERALD RAINER, M.D., GLEN  
JAMES P. NEWBY, M.D., J. PH

Volume 167  
Number 3

### FIBROMUSCULAR HYPERPLASIA

FIG. 1. Left carotid arteriograms taken with the patient's head in neutral (A) and right lateral (B) positions. The discrete defect at the internal carotid origin and post-stenotic dilatation in the positional view are clearly shown.



were no abnormal pulses or bruits and sequential carotid compression was tolerated without ill effect. There were no signs of residual neurologic deficit. Positional ophthalmodynamometry showed no significant changes in retinal artery pressures.

Submitted for publication September 5, 1967.  
Reprint requests to 701 E. Colfax Avenue, Denver, Colorado 80202 (Dr. Rainer).

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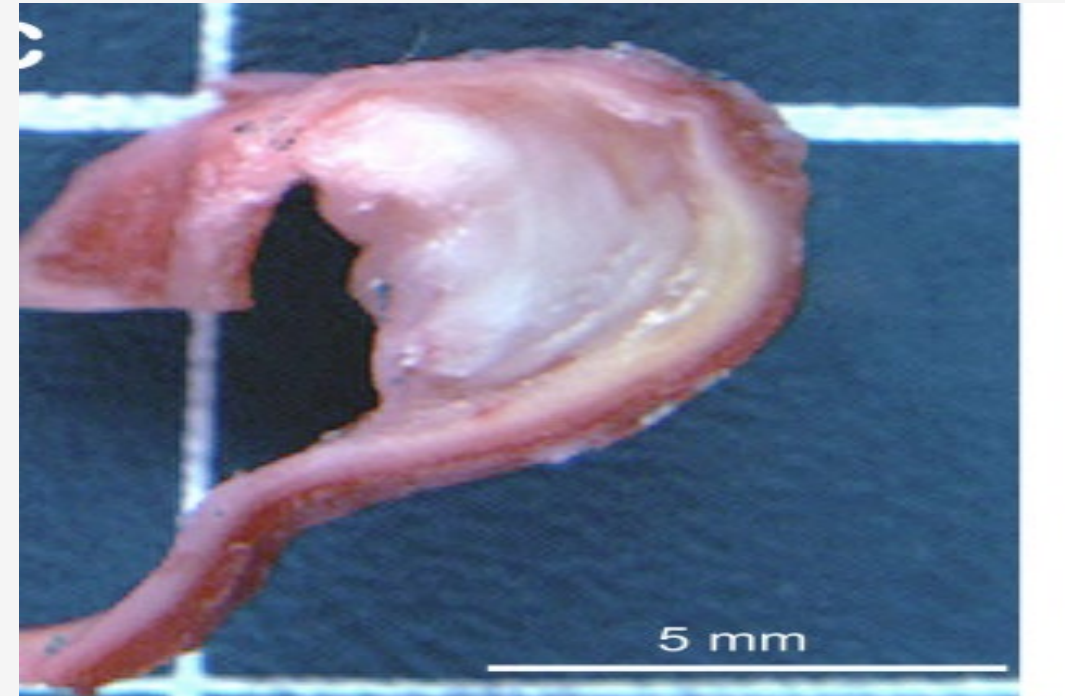


FIGURE 1. Left carotid angiogram, lateral projection, showing web of proximal internal carotid artery.



# Histopathology

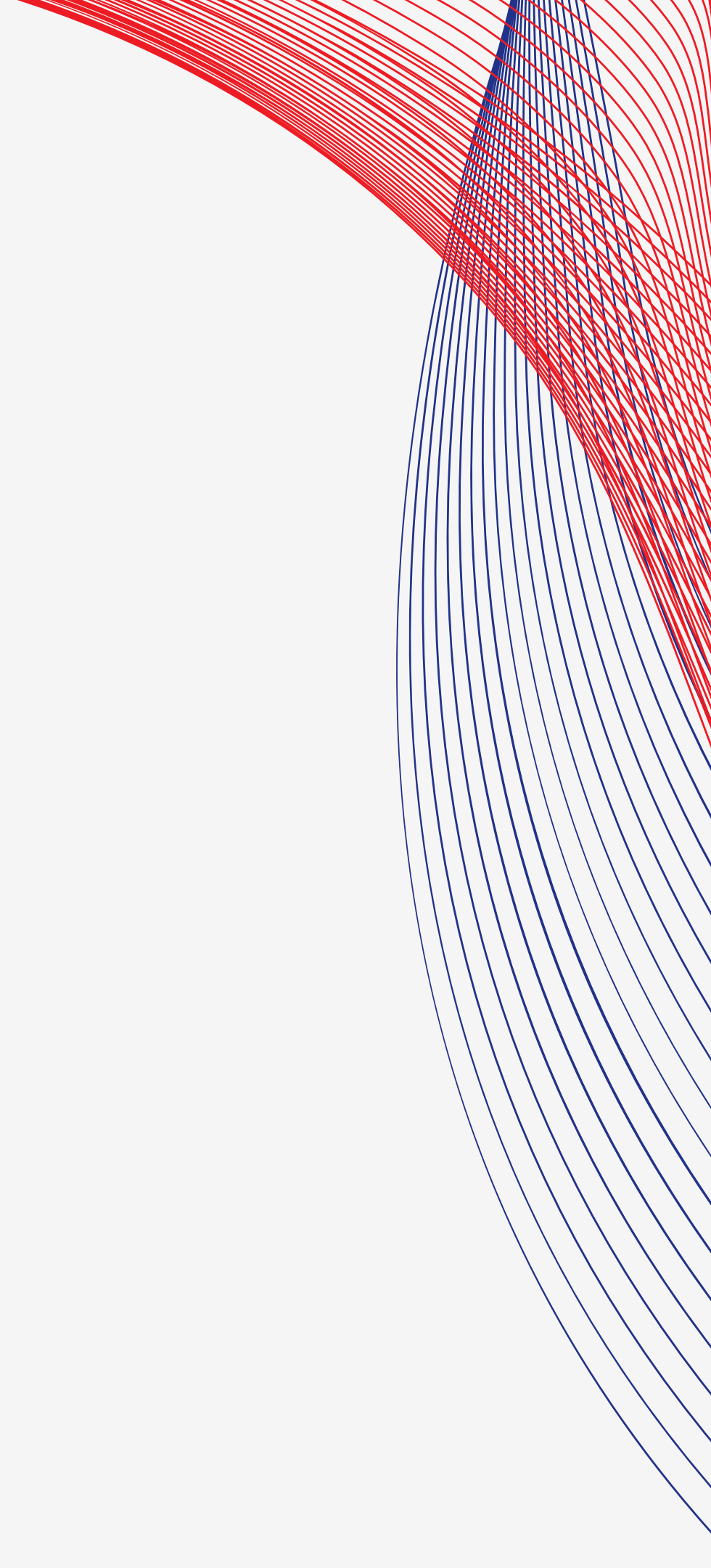
- Described as “Fibromuscular Hyperplasia” by Rainer et al.
- Intimal variant of fibromuscular dysplasia
- Gross: smooth contour with eccentric intimal thickening
- Microscopy
  - Absence of internal elastic lamina
  - Increase fibrosis
  - Myxoid degeneration





# Epidemiology

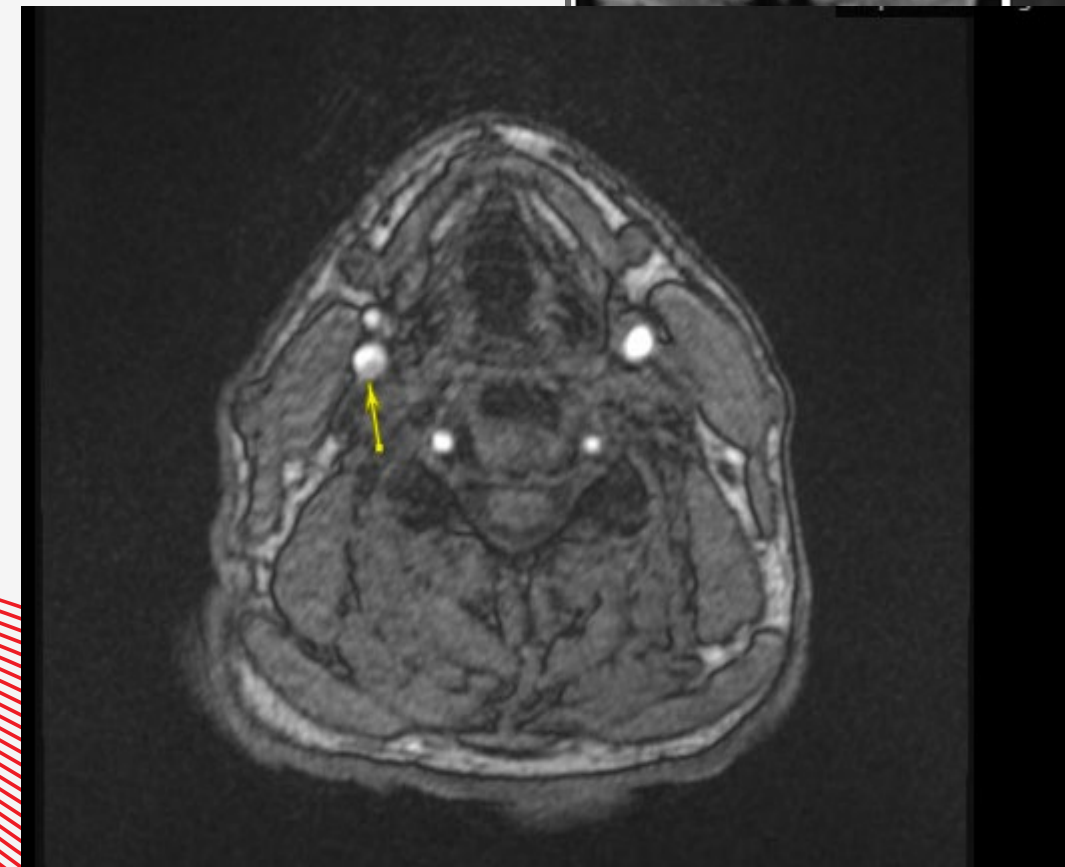
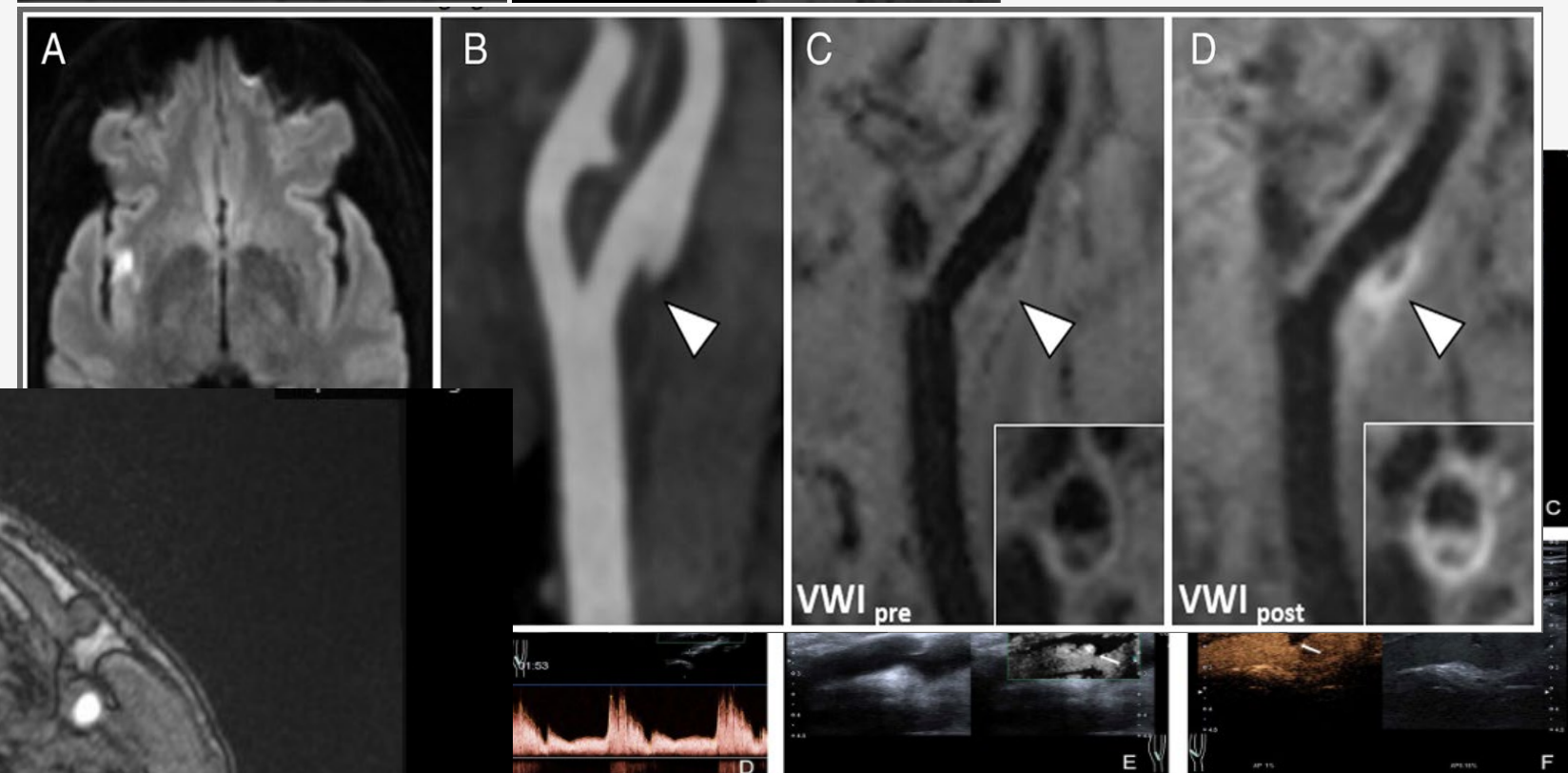
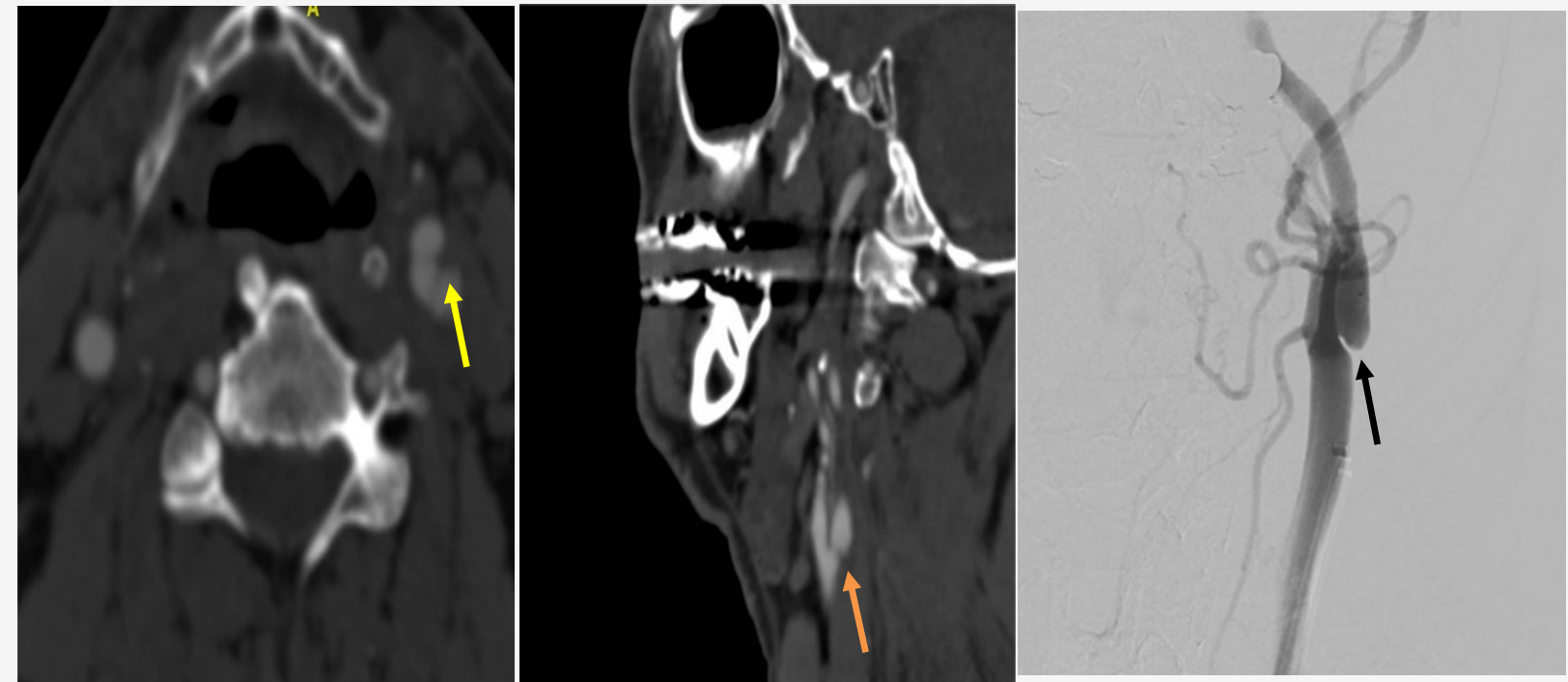
- Prevalence is unknown in general population
- Reported data is from stroke registries
- Period Prevalence in a hospital-based study
  - 1.2%(Choi et al.)
- Affects Women > Men
- Black > White
- Unilateral carotid webs are more common than bilateral carotid webs
- Association with FMD
  - Carotid FMD: ~9%
  - Other vascular beds; <1%





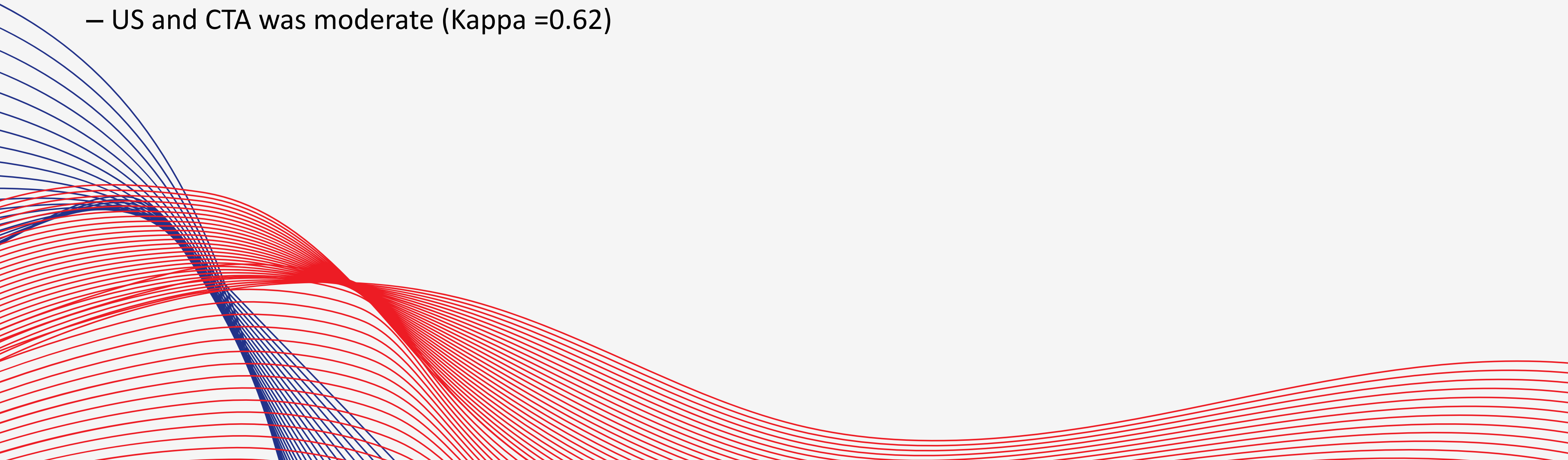
# Diagnosis of CaWs

- Shelf like projection
  - Originates from the proximal ICA
  - Posterolateral wall
  - Must be seen on axial and sagittal planes
- Found within 2cm of ICA
- Superimposed thrombus can be seen
- Diagnosed by both invasive and non-invasive carotid imaging
  - CUS, CTA, DSA and HRVWI



# Multimodality Imaging

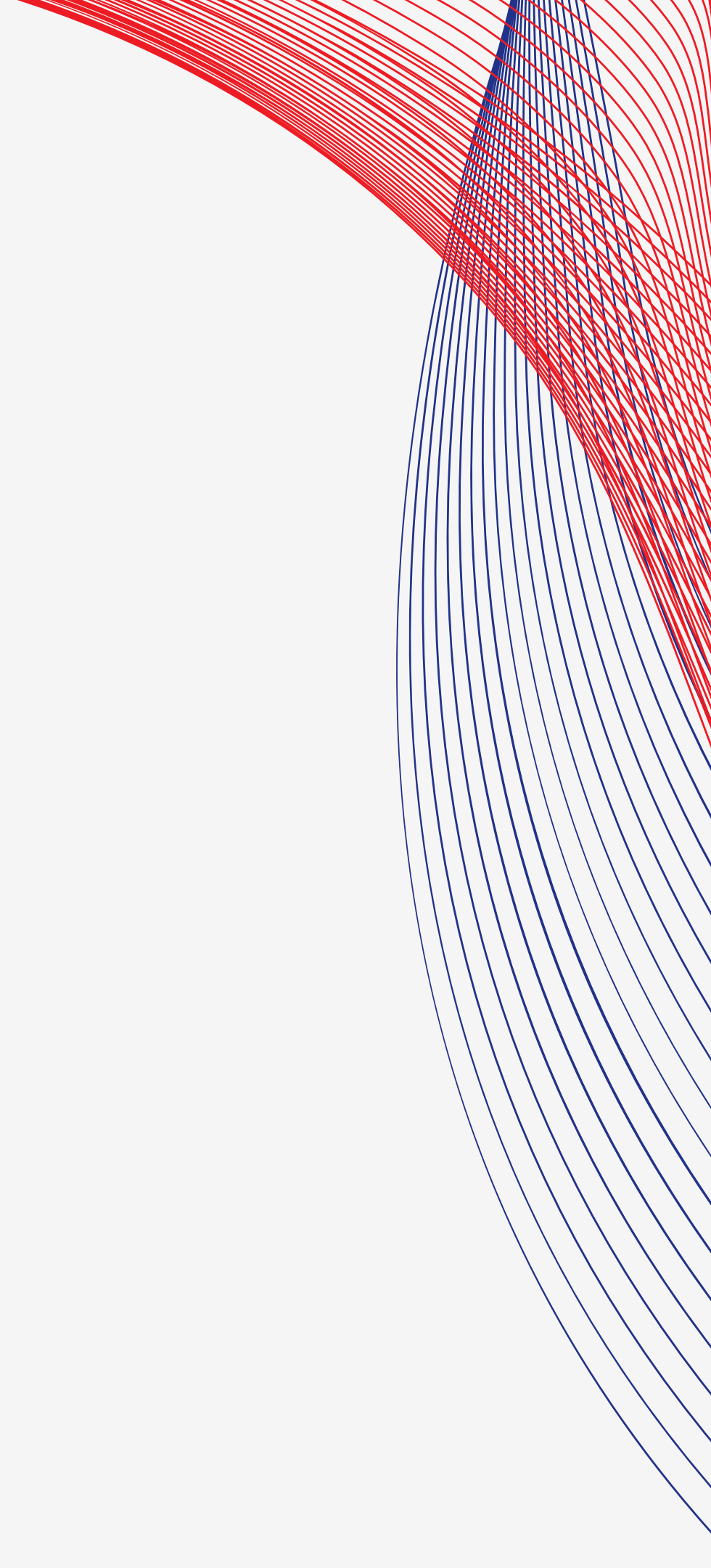
- CUS vs. CTA
  - Cohen's kappa=0.553,  $p<0.01$
- DSA vs. CTA
  - Cohen's kappa = 0.92,  $p<0.01$
- DSA vs US vs CTA
  - Correlation was perfect(Kappa =1)
  - US and CTA was moderate (Kappa =0.62)





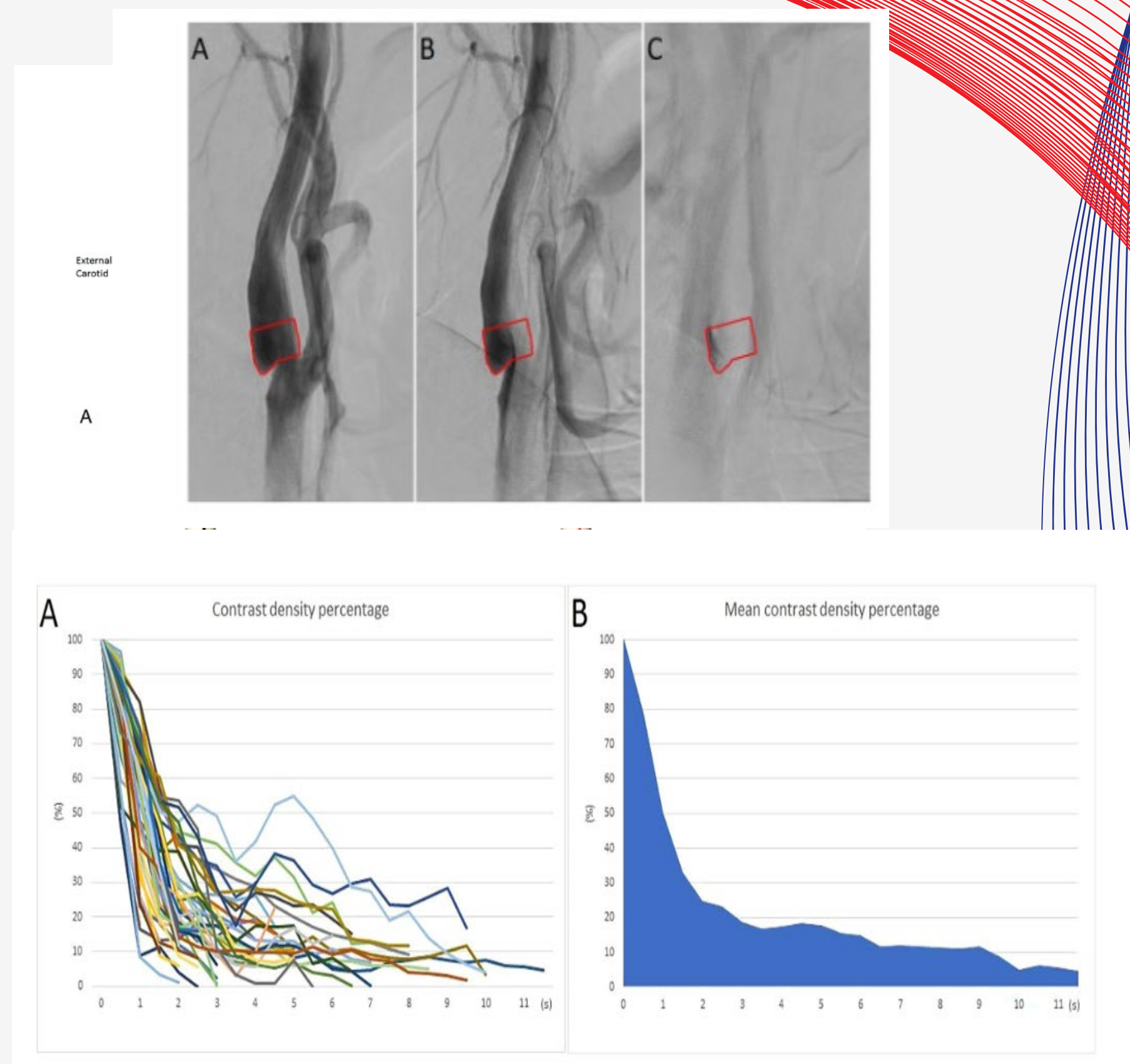
# CW & Ischemic Stroke

- Responsible for 1.6% of cryptogenic stroke
- MR CLEAN
  - Symptomatic CW(2.5%)
  - Asymptomatic CW(0.5%)
- RIH LVO Registry
  - 1.8% ipsilateral carotid webs
  - >70% were ESUS
  - Over 90% were not detected
- Patient Characteristics
  - Young black women
  - Less conventional risk factors(HTN, HLD, DM)
- Large proportion of early studies were performed in French Africa



# Mechanism of Stroke

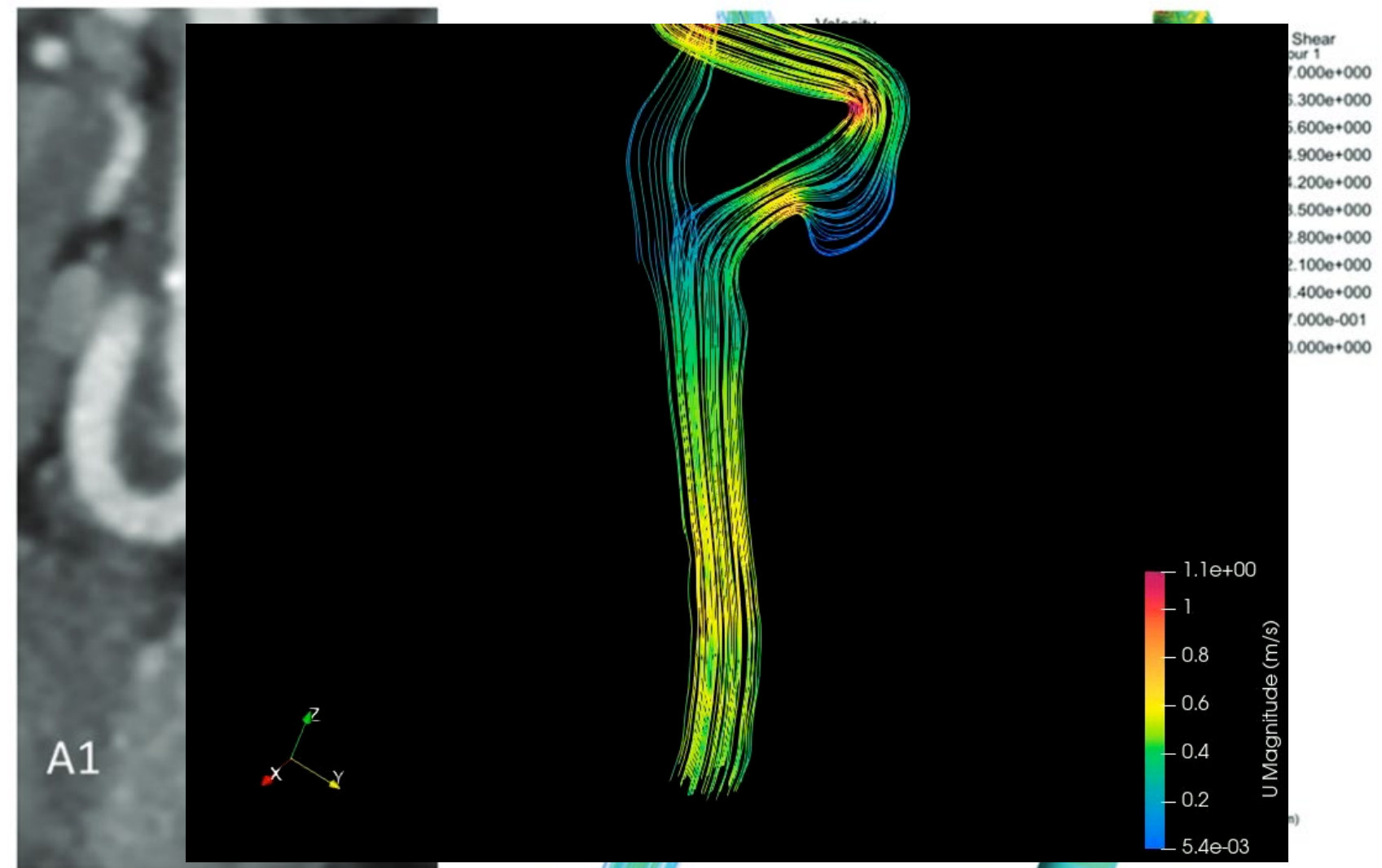
- Virchow's triad
  - Stagnant flow
  - Hypercoagulability
  - Endothelial injury
- Disruption of blood flow
  - Thrombus formation
  - Thromboembolism
- DSA time density curve
  - TDC showed an initial fast decay from the peak concentration followed by a plateau with a negative exponential pattern.
  - No association with morphology(size, shape, angle)



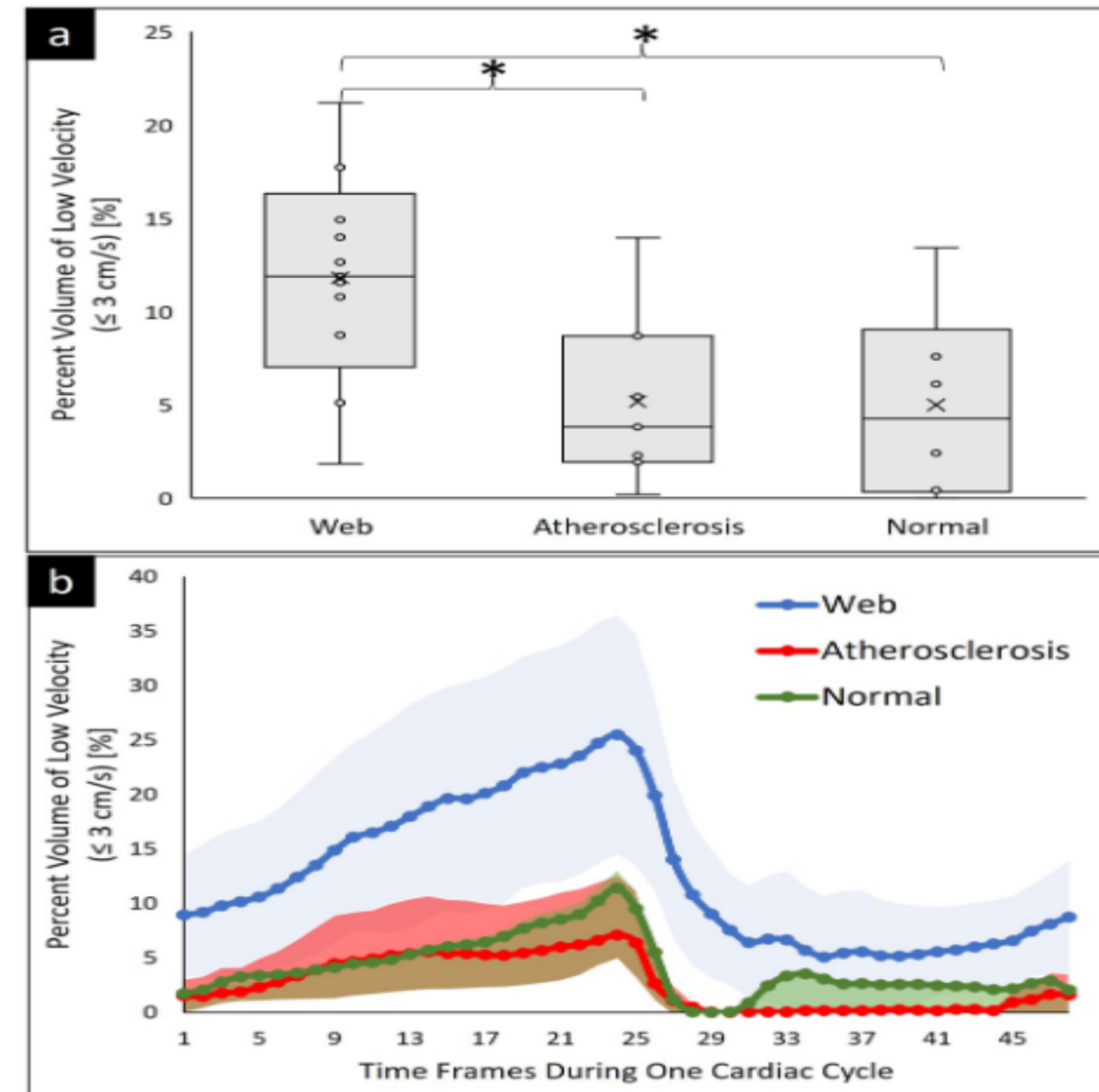


# Flow Patterns

- OSI & low TAWSS are markers of recirculation zones
- Average TAWSS( $\pm 0.4$ Pa)
- Increased wall shear stress  $\rightarrow$  endothelial damage
- Increased recirculation zones  $\rightarrow$  blood flow stasis
- Low shear stress
  - CW vs. mild atherosclerosis
    - 2.84% vs. 0.047%,  $p < 0.01$
  - CW vs. normal subjects
    - 2.84 vs. 0.47%  $p = 0.01$
- Low flow velocity regions



- Volume of low flow velocity regions(<3cm/sec)
  - CW 11.8% vs. athero 5.2%(p=0.03)
  - CW 11.8% vs. normal 5.0%(p=0.03)
- Stasis(% of cardiac cycle<3 cm/s)
  - CW 95% vs athero 58%, p<0.001
  - CW 95% vs. normal 84%, p=0.02
- No of pts exposed to low velocity across full cardiac cycle
  - 9/13 in CW
  - 0/7 in athero
  - 0/6 in normal





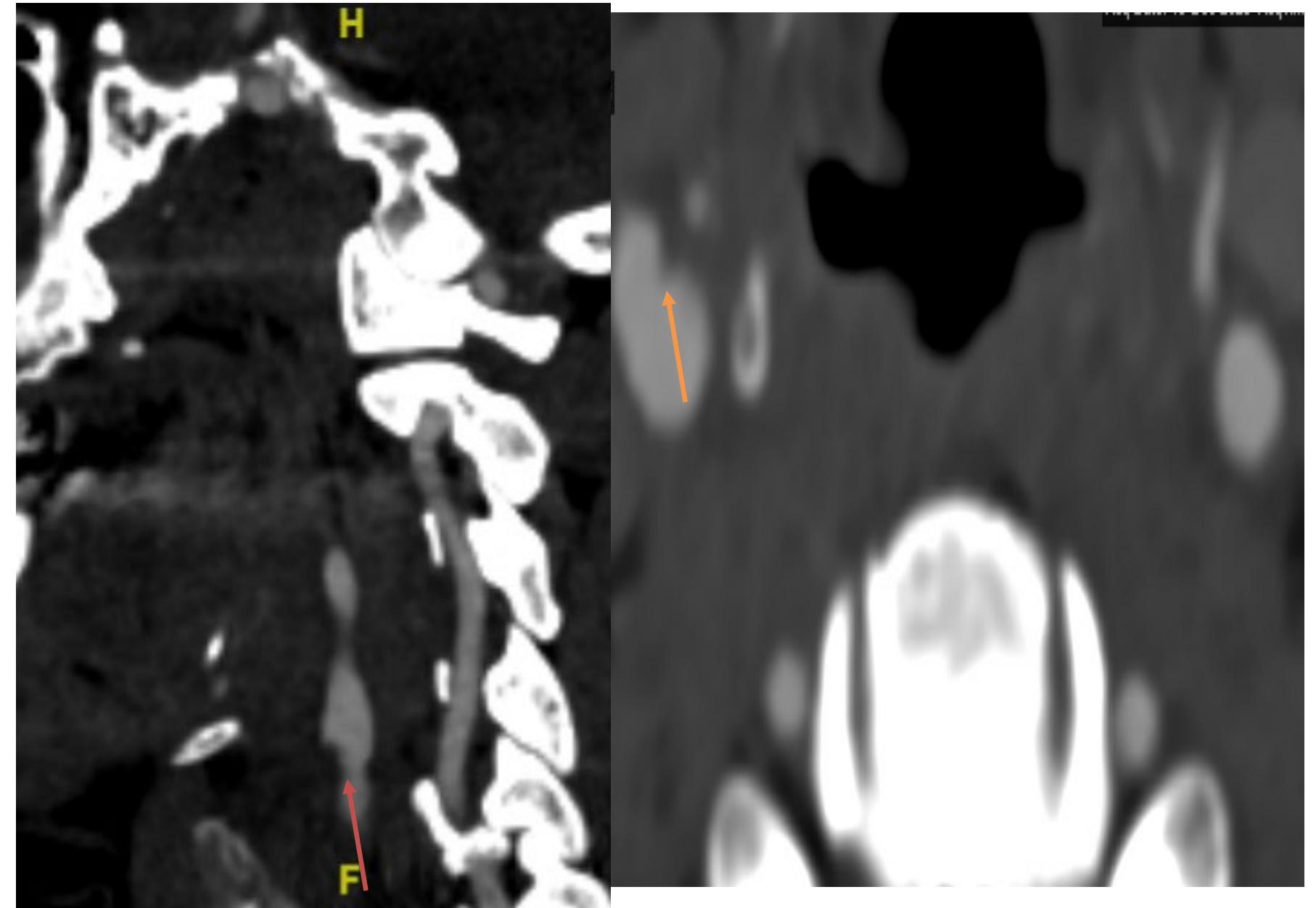
# Case 1

- 50-year-old with hx of HTN presented with mild right weakness and fluent aphasia
- NIHSS 3 on arrival
- Vessel imaging showed no LVO and intraluminal thrombus vs. ulcerated plaque in L ICA
- Heparin gtt was initiated
- Work up unremarkable(TTE, EKG, hypercoagulable)



## Case 2

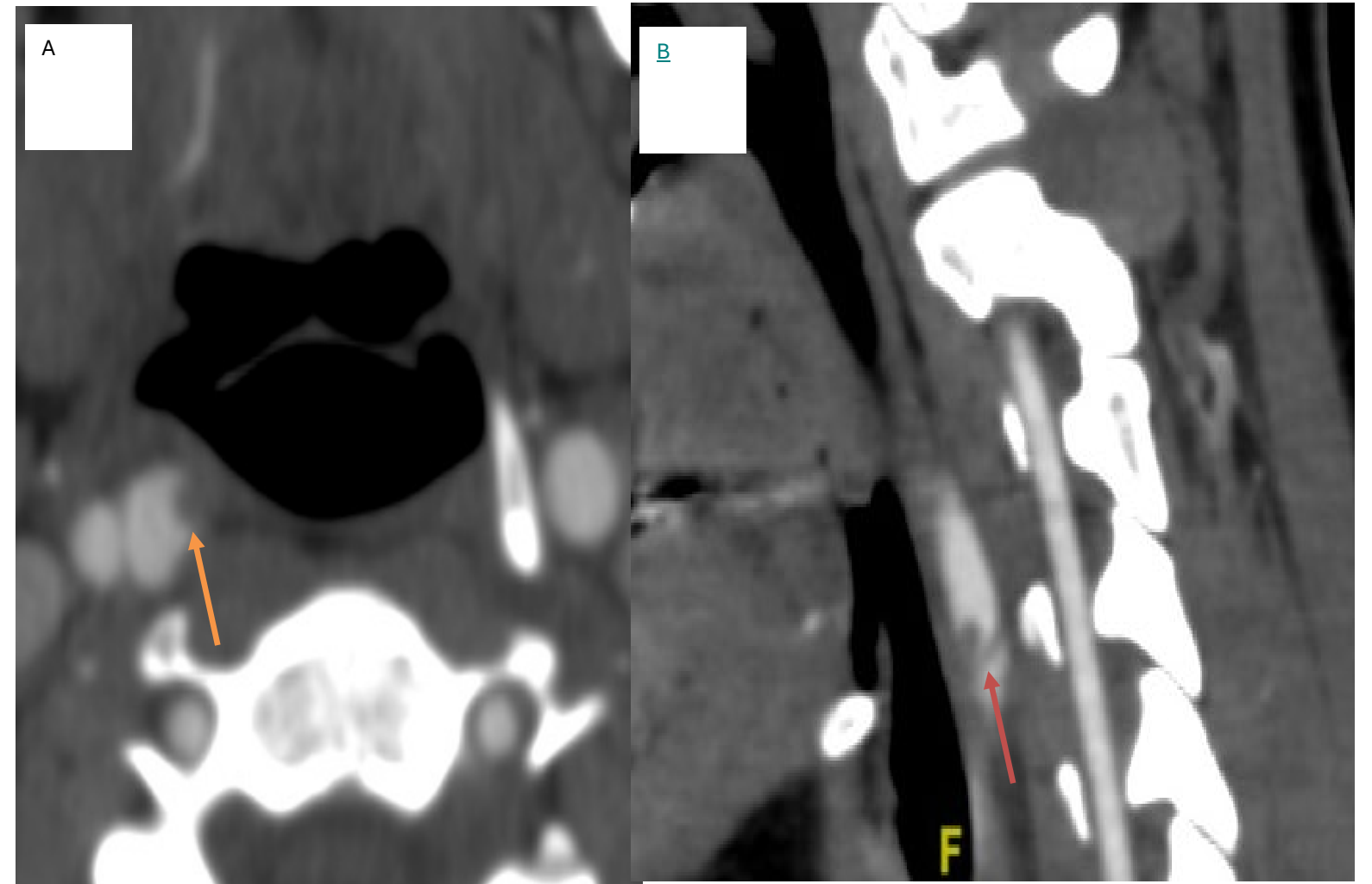
- 50 YO M presented with right MCA syndrome after a TIA three weeks ago
- Vessel imaging showed soft non stenosing plaque in the proximal ICA
- Work up unremarkable(TTE, EKG, hypercoag.)





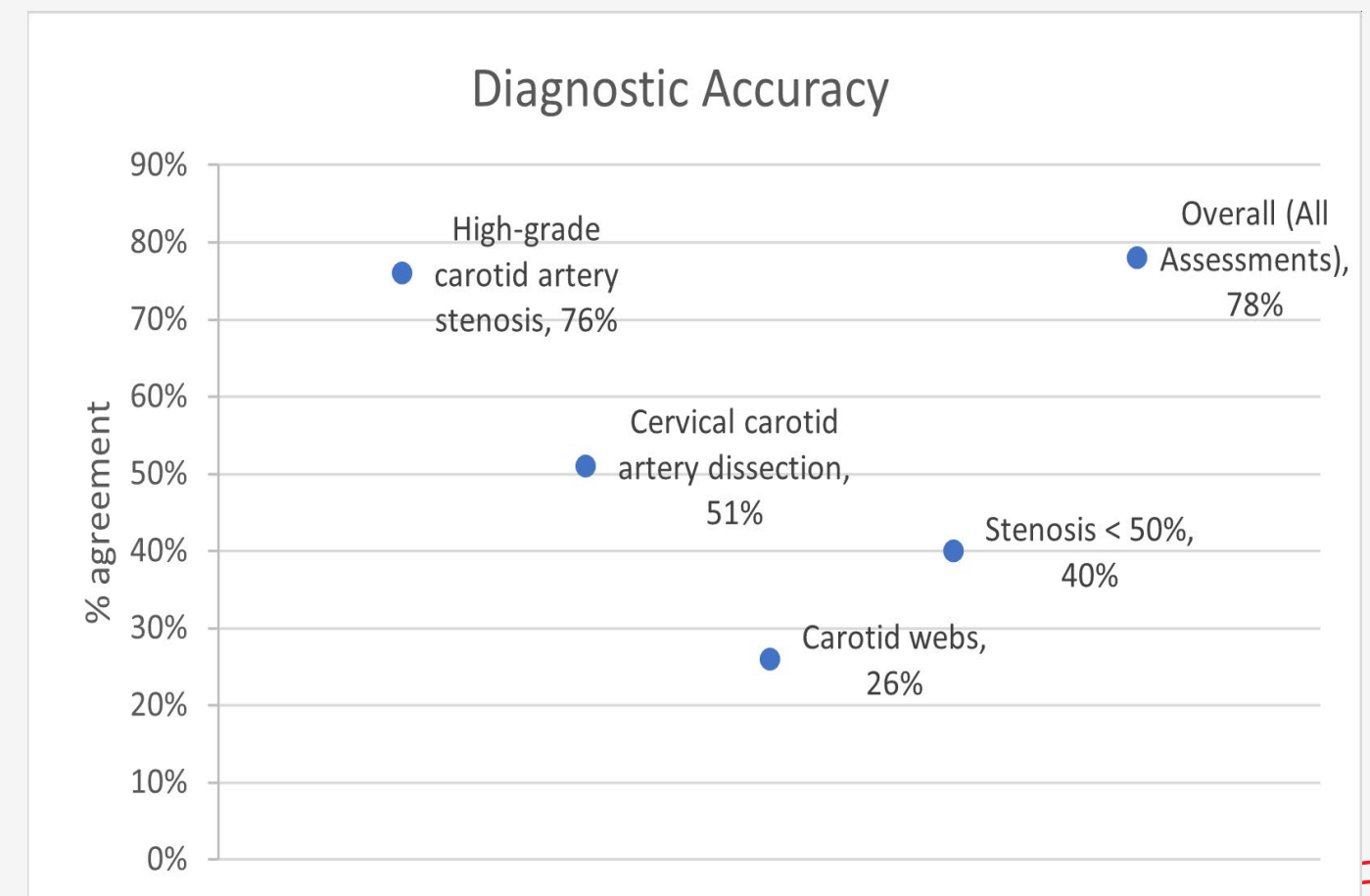
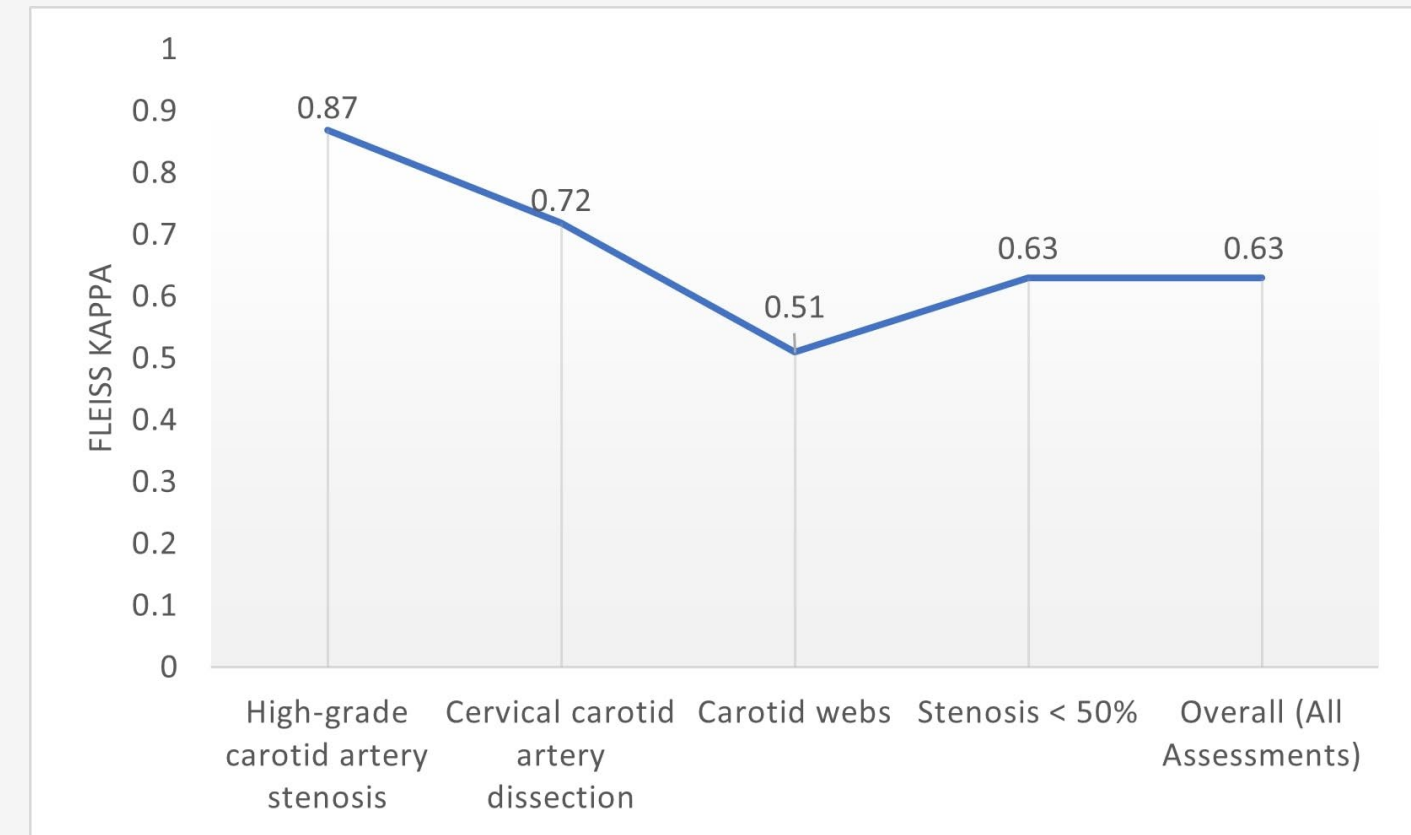
## Case 3

- 21 YO F presented with dysarthria and left-hand numbness
- OSH concern for right ICA dissection → transferred for further evaluation
- MRI brain with embolic-appearing right frontal lobe infarct
- Work-up
  - TTE, cardiac monitoring, hypercoagulable panel (incl. APS) -  
ive



# Diagnostic Challenges

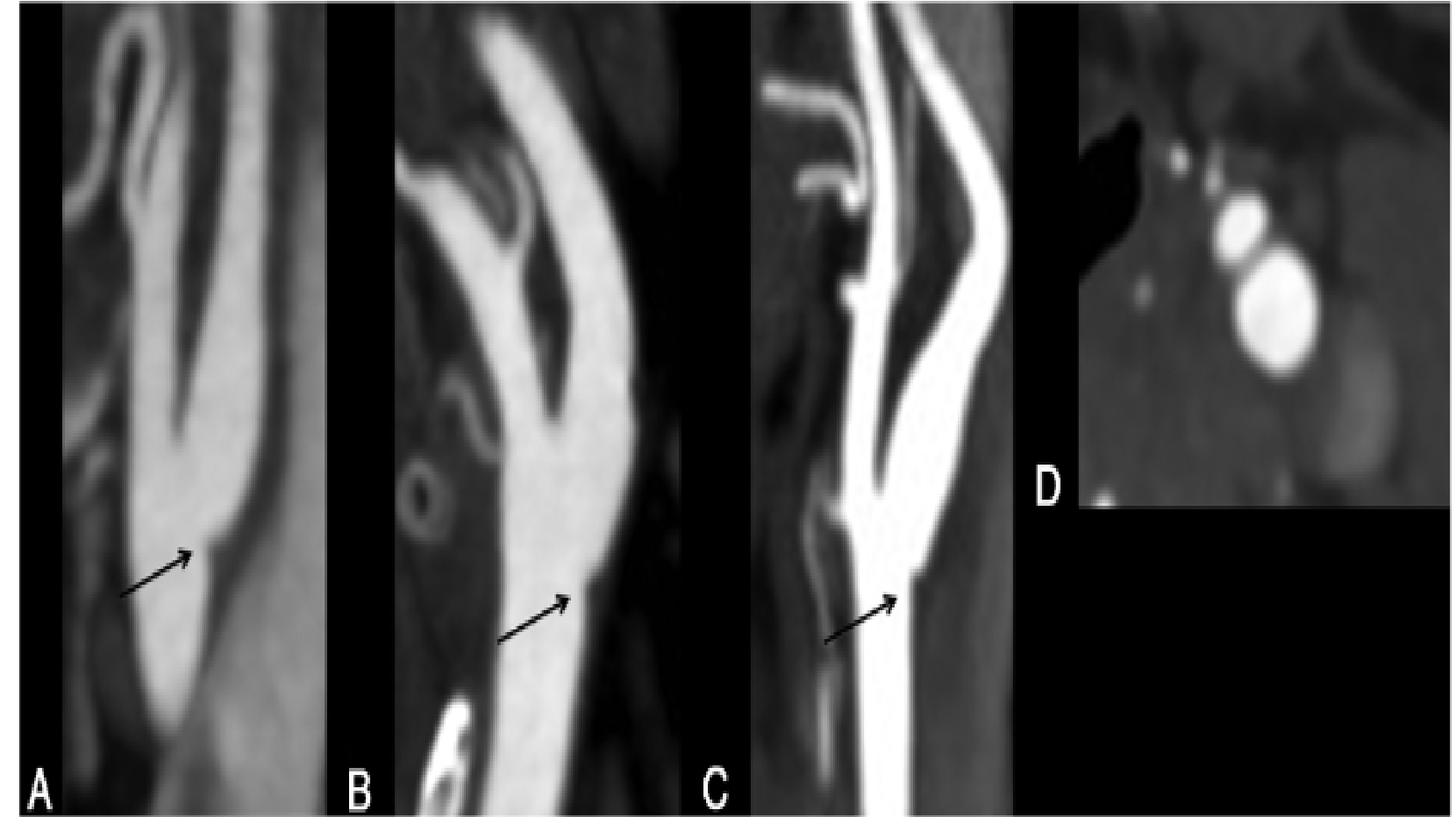
- Agreement amongst subspecialists(*in Press*)
  - CW
    - Fleiss kappa = 0.51,  $p < 0.001$
  - Large artery atherosclerosis >70% stenosis
    - Fleiss kappa = 0.87,  $p < 0.001$
- Accuracy
  - Poor diagnostic accuracy(26%)
- Consensus decision involving three or more raters
  - Poor agreement for CW( kappa=0.21,  $p < 0.01$ )
  - Perfect agreement for LAA(kappa=1,  $p < 0.01$ )





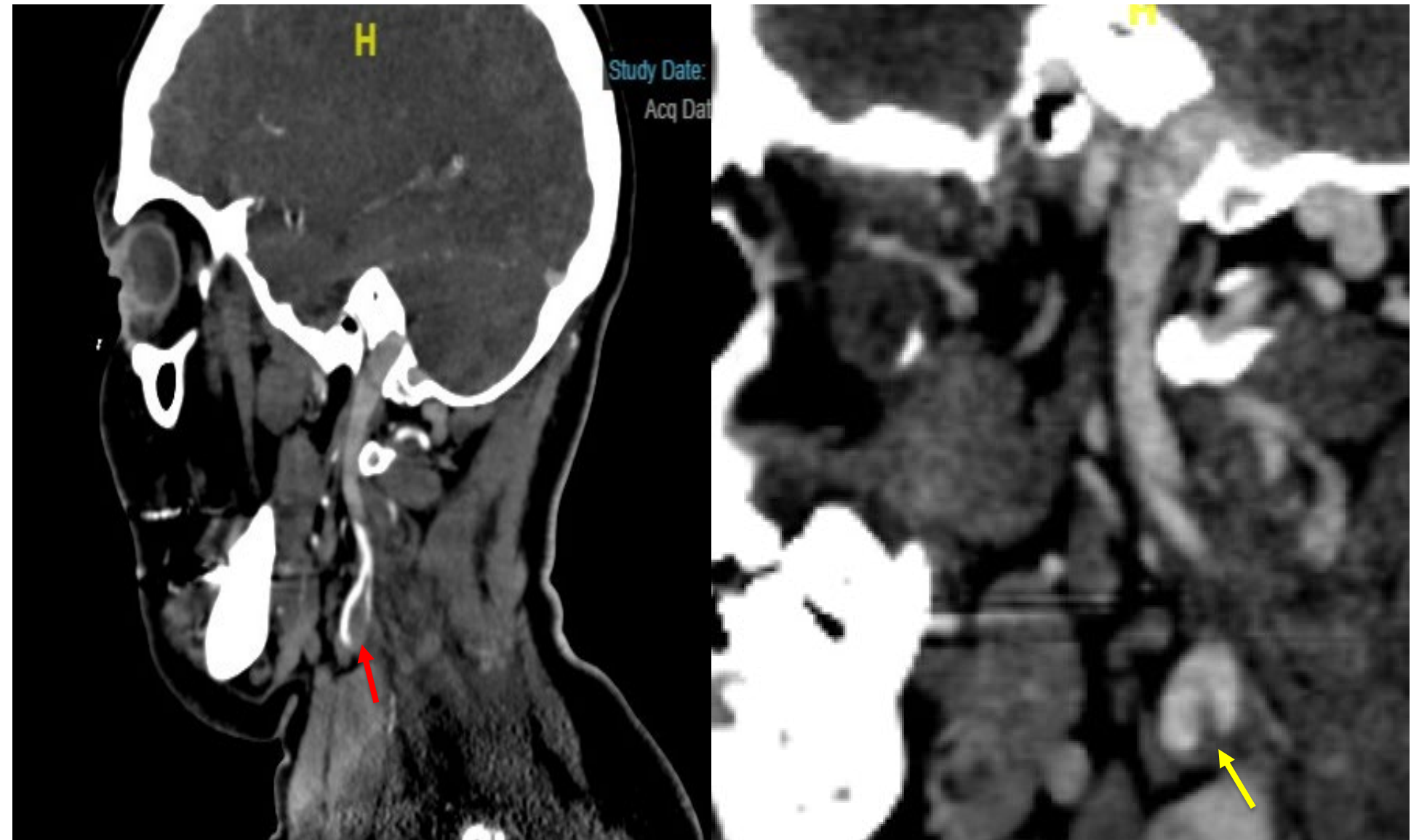
# Causes of Diagnostic Challenges

- Similar appearing lesions
  - Small protruding lesion
  - Small cervical dissections
  - Superimposed thrombus appearing as soft plaque
- Under-recognized entity
  - Subtle nature of these lesions
  - Lack of flow limiting stenosis
  - Lack of education



# Case 1

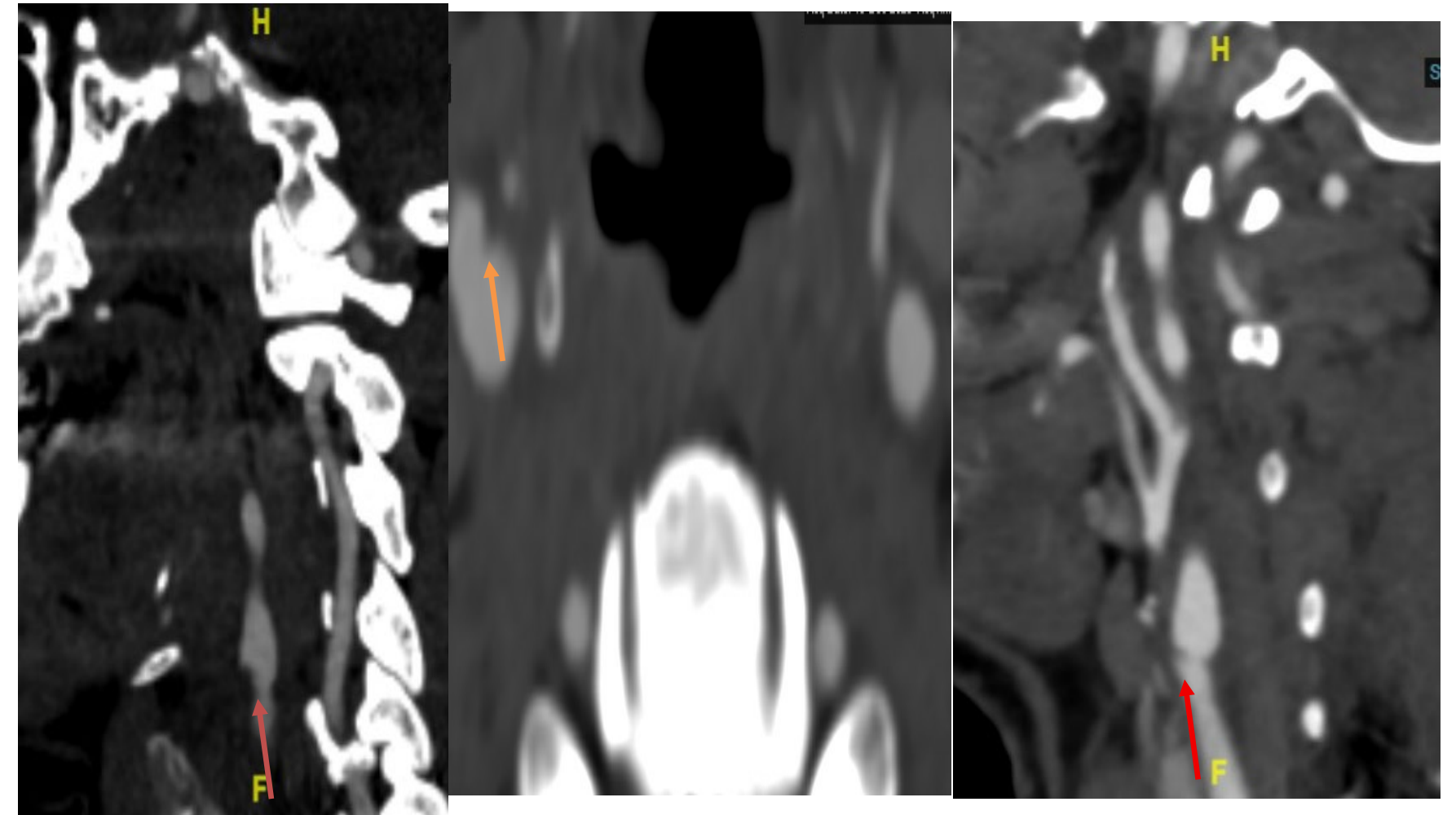
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- NIHSS 3 on arrival
- Vessel imaging showed no LVO and intraluminal thrombus vs. ulcerated plaque in L ICA
- Heparin gtt was initiated
- At 72 hours
  - Resolution of thrombus
  - Carotid web was noted





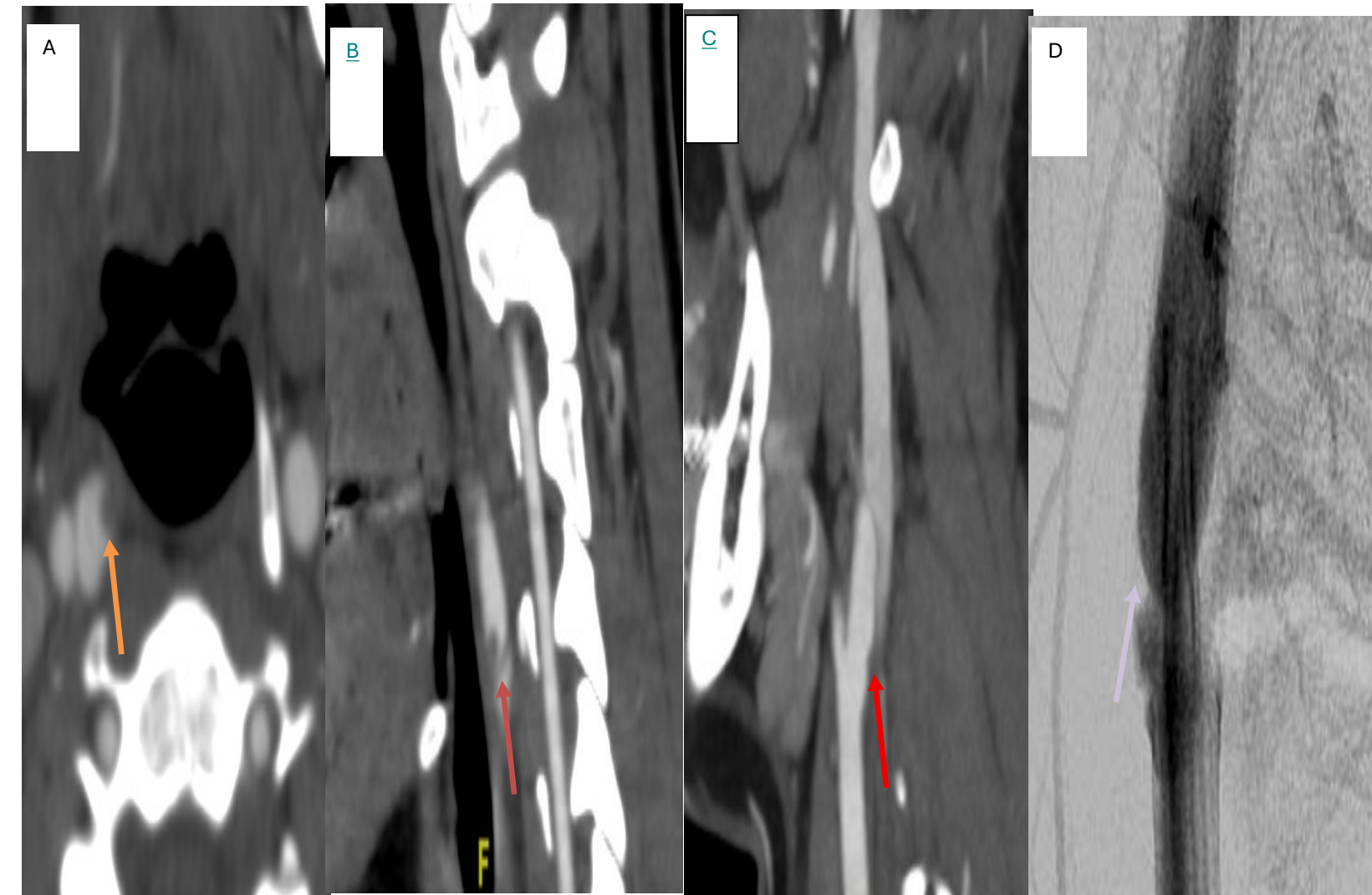
## Case 2

- 50 YO M presented with right MCA syndrome after a TIA three weeks ago
- Vessel imaging showed ?small CW with superimposed thrombus
- No thrombus seen on prior imaging three weeks ago (red arrow)



## Case 3

- 21 YO F presented with dysarthria and left-hand numbness
- OSH concern for right ICA dissection → transferred for further evaluation
- MRI brain with embolic-appearing right frontal lobe infarct
- Work-up
  - TTE, cardiac monitoring, hypercoagulable panel (incl. APS) –  
ive
- At 3 months, repeat CTA showed resolution of clot with underlying possible CW
- 6 months later
  - Diagnostic angiogram confirmed proximal right ICA carotid web





# How to Overcome?

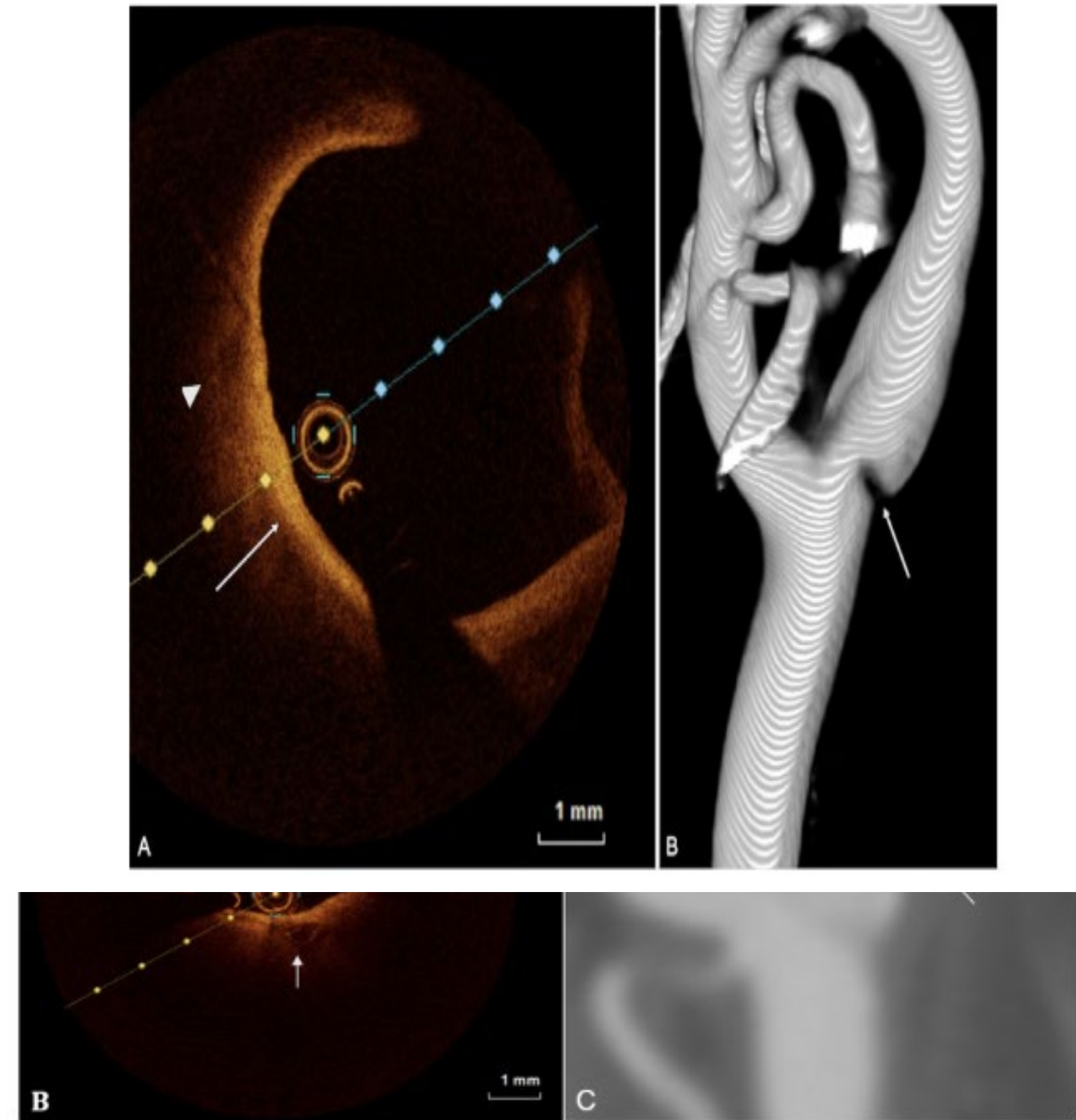
- Contrast enhanced US(non-invasive)
  - Differentiate CW with and without plaque
- Carotid artery web with plaque
  - Longer web length
  - Increased web thickness
  - More likely to have >50% stenosis

Comparison of patients with ultrasonic features

Parameters	Total (n = 299)	CW (n = 68)	CW with plaque (n = 231)	t/ $\chi^2$	p value
Web number, n (%)				8.465	0.003
Single	246 (82.3)	64 (94.1)	182 (78.8)		
Multiple	53 (17.7)	4 (5.9)	49 (21.2)		
Web length (mm)	6.51 ± 2.82	4.99 ± 2.22	7.00 ± 2.82	-5.307	< 0.001
Web thickness (mm)	1.55 ± 0.73	0.95 ± 0.43	1.23 ± 0.79	-2.702	0.007
Web location, n (%)				1.346	0.271
Right artery	146 (48.8)	29 (42.6)	117 (50.6)		
Left artery	153 (51.2)	39 (54.4)	115 (49.6)		
Arterial wall, n (%)				28.344	< 0.001
Anterior	71 (23.7)	4 (5.9)	67 (29.0)		
Posterior	186 (62.2)	61 (89.7)	125 (54.1)		
Sides	42 (14.0)	3 (4.4)	39 (16.9)		
Web angle (°)	52.42 ± 34.83	37.33 ± 23.14	58.72 ± 36.95	-4.255	< 0.001
Plaque length (mm)	/	/	18.41 ± 7.39 <sup>a</sup>		
Plaque thickness (mm)	/	/	3.91 ± 1.33		
≥ 50% stenosis, n (%)	141 (47.2)	7 (10.3%)	134 (58.0%)	47.997	< 0.001
CEUS enhancement, n (%)				129.48	< 0.001
Grade 0	121 (40.5)	68 (100)	53 (22.9)		
Grade 1	87 (29.1)	0	87 (33.7)		
Grade 2	91 (30.4)	0	91 (39.4)		

# How to Overcome?

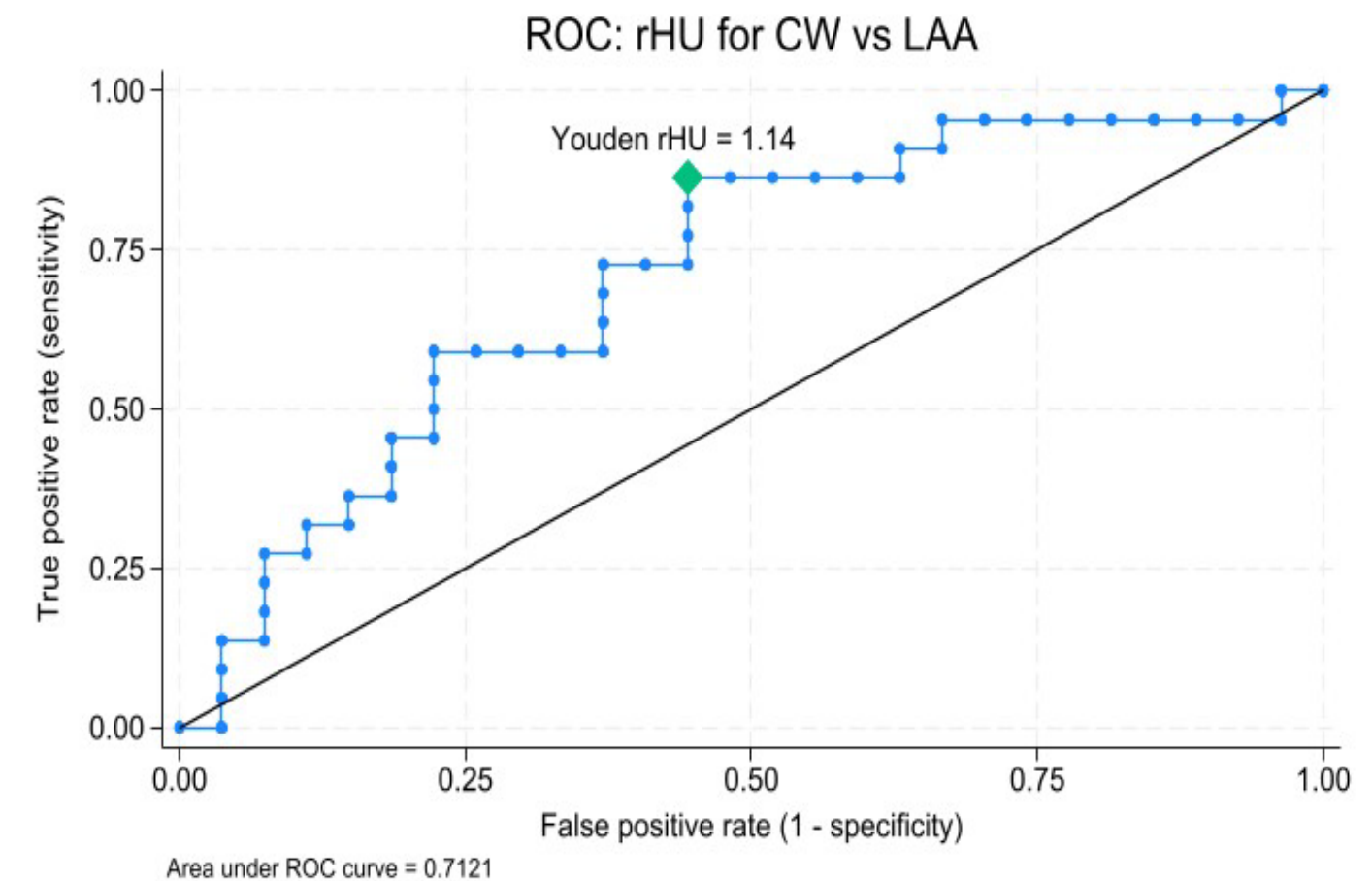
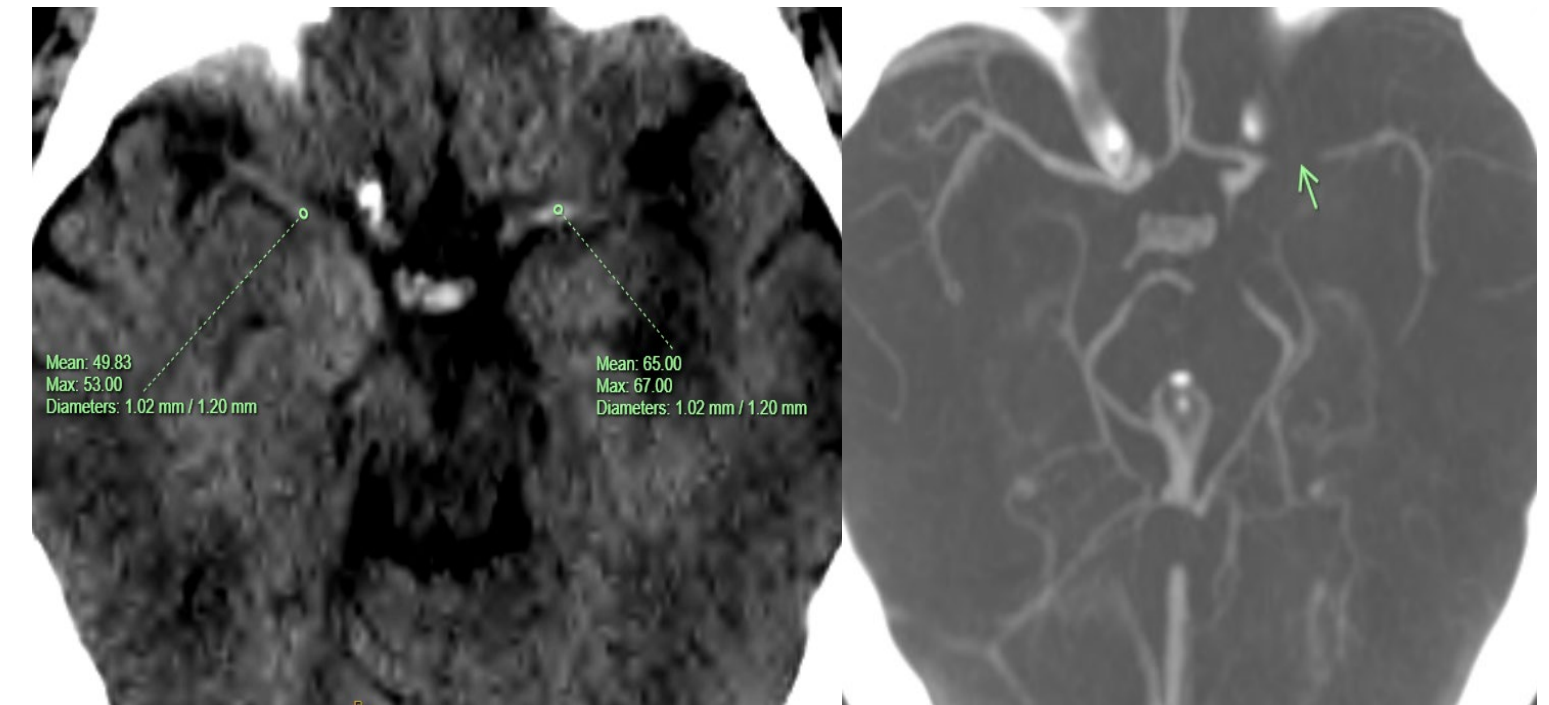
- Use of optical coherence tomography(OCT)
- Used NIR(backscattered light from particles)
- Ruled in 75% of cases
- 25% had presence of microthrombi





# Imaging Biomarker

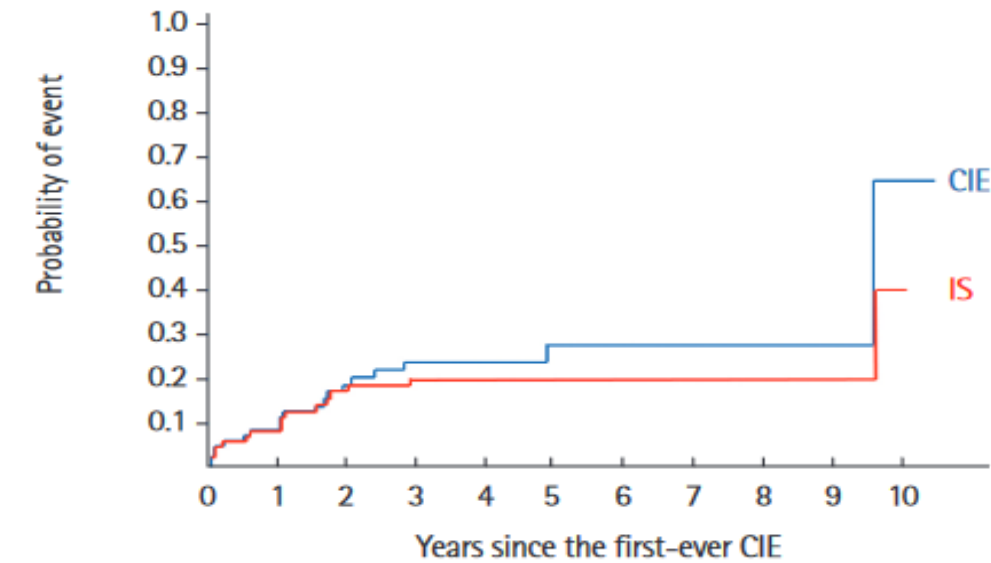
- Hyperdense MCA sign (HMCAS): observed in 60.5%(~80% in CW)
- Association with HMCAS
  - Higher CW vs LAA → aRR 1.89 (95% CI, 1.18–2.99; p = 0.007)
  - Higher CW vs CE → aRR 1.64 (95% CI, 1.07–2.51; p = 0.022)
- Optimal rHU threshold (CW vs LAA)
  - 1.14(Sensitivity 0.86, Specificity 0.56, AUC 0.71)
- Recanalization outcomes( $\geq$ TICI2c)
  - Higher rates in CW vs LAA → aRR 1.53 (95% CI, 1.15–2.02; p = 0.003)
  - No difference compared with CE



# Recurrent Ischemic Stroke

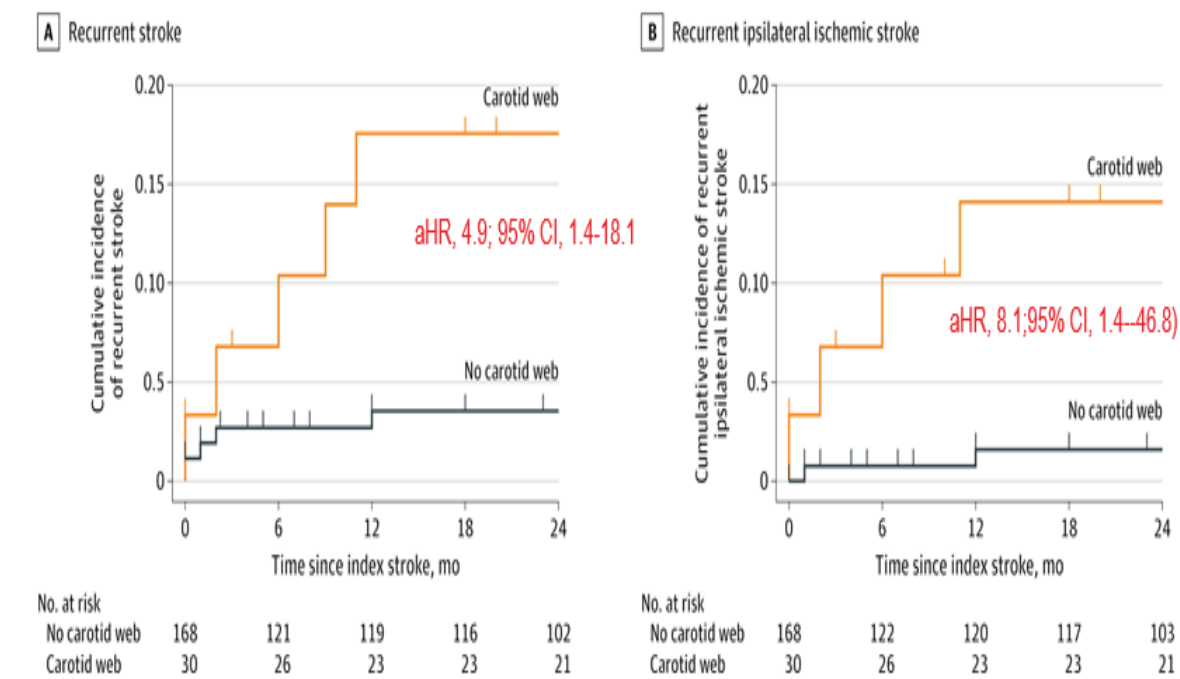
- High rates of early and late recurrence
  - 10%-40%
  - Early Recurrence
    - Increased in-hospital recurrence (RR 4.38, 95% CI: 1.38–13.85)
  - Silent cortical infarction
    - Detected in 7.6%
    - Predictor of recurrence (HR 8.4; 95% CI, 2.9 to 24)
  - Late Recurrence
    - Annual recurrent CIE rate was 5.4%/year
  - MR CLEAN registry
    - 17% with CW vs 3% without CW

Kaplan-Meier curves for time to recurrent CIE and IS under medical treatment



**Figure 1.** Kaplan-Meier curves of cumulative rates of cerebral ischemic event and ischemic stroke recurrences in symptomatic carotid web patients medically treated. CIE, cerebral ischemic event; IS, ischemic stroke.

**Fig.2** Kaplan-Meier Estimates of Recurrent Stroke and Recurrent Ipsilateral Ischemic Stroke During 2-Year Follow-up After the Index Stroke





# Impact of Diagnostic Delay

- RIH LVO registry
  - >90% were not detected
  - 20% had recurrence
- CAROWEB multicenter registry
  - 25% were undetected
- Predictors of diagnostic delay
  - Low NIHSS
  - Absence of CTA
  - Stroke recurrence before 2019
- Impact of delay
  - Increased recurrence(Adjusted hazard ratio, 5.02; p=0.014)
  - Delayed revascularization(median 248.5 versus 53.5; p<0.001)

# Management

- No clear guidelines from AHA/ASA
- Practice patterns are unclear
- Medical management(antiplatelet & anticoagulation)
  - High rates of recurrence(9%-40%)
  - Meta-analysis showed 32%(90/281)recurrence in medical arm

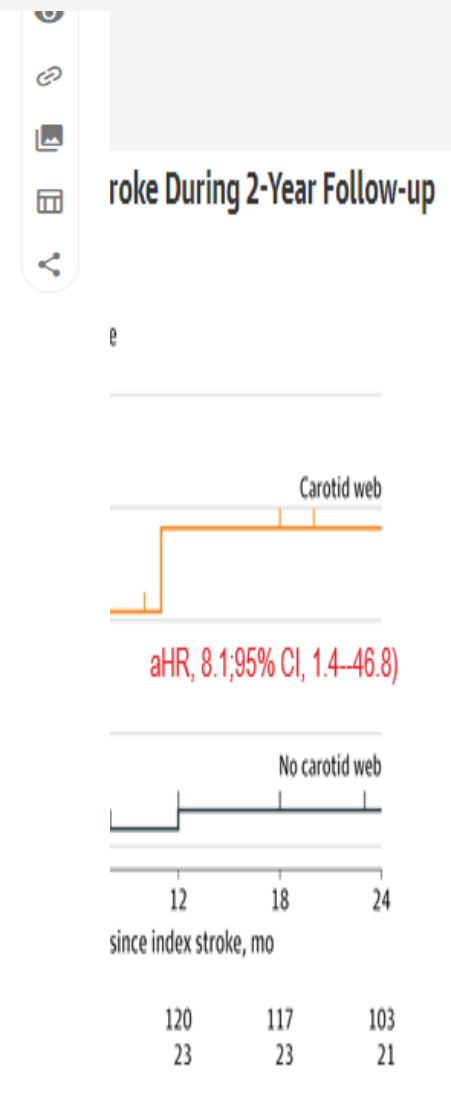
Carotid web is a thin, circumferential shelf-like filling defect that arises from the posterior wall of the ICA bulb visualized on CTA or carotid angiography. Pathologically, carotid web is a variant of fibromuscular dysplasia and can be classified as focal or multifocal. Platelet deposition can occur in the corrugations of carotid web, forming a nidus for potential blood flow stagnation and thromboembolism. Carotid web is a known cause of ischemic stroke in young patients <65 years of age; it is detected in up to 9.5% of patients <65 years of age with anterior circulation stroke of unknown cause.<sup>751</sup>

## Recommendation-Specific Supportive Text

1. The optimal management of symptomatic carotid web is unknown. Medical management with antithrombotic therapy is first-line treatment; however, it is not known whether SAPT, short-term DAPT, or anticoagulant therapy is superior. In the absence of such data, it is recommended to treat patients with antiplatelet therapy first line or to follow antithrombotic recommendations in this guideline.
2. There is a high risk of recurrent stroke or TIA in patients with symptomatic carotid web on medical management, estimated in 29% to 56% of patients.<sup>749,750</sup> Carotid stenting or CEA is a good alternative treatment for patients with symptomatic carotid web, with published series revealing no recurrent stroke risk.<sup>748,749</sup>

## Knowledge Gaps and Future Research

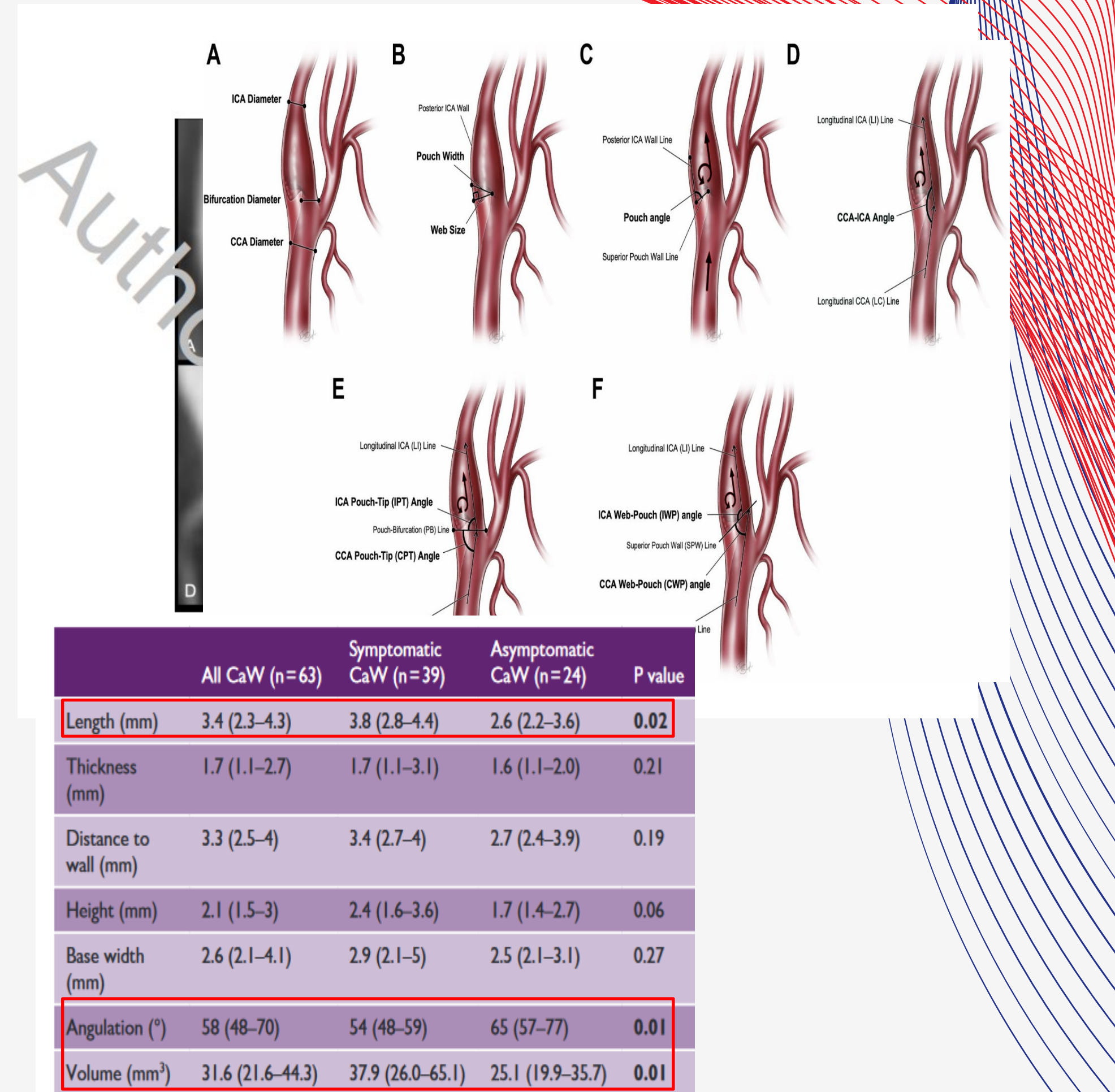
The optimal medical or interventional management of symptomatic carotid web is unknown. Future prospective research evaluating the natural history of symptomatic carotid web on medical management compared with interventional management would be of interest and evolve to multicenter randomized trials comparing medical management (ie, antiplatelet versus anticoagulant, then best medical therapy versus carotid stenting or CEA) if enough patients can be identified.



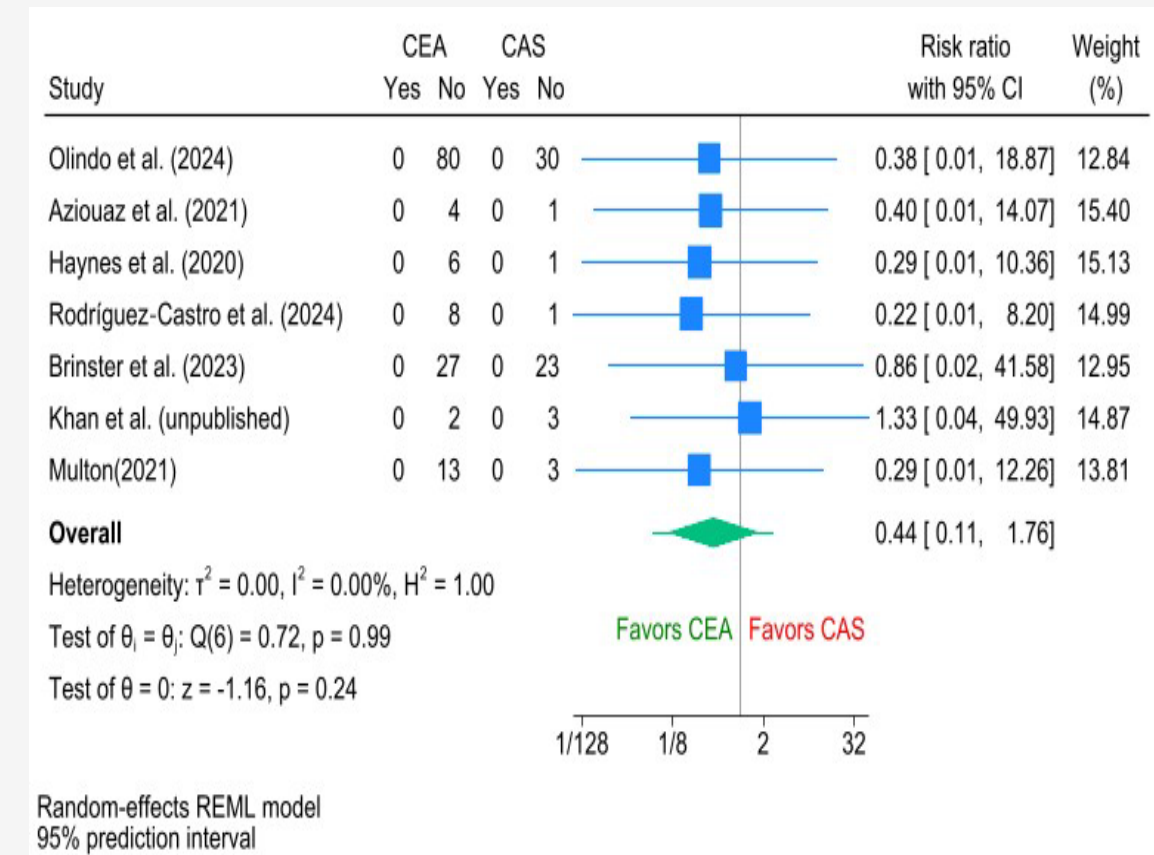
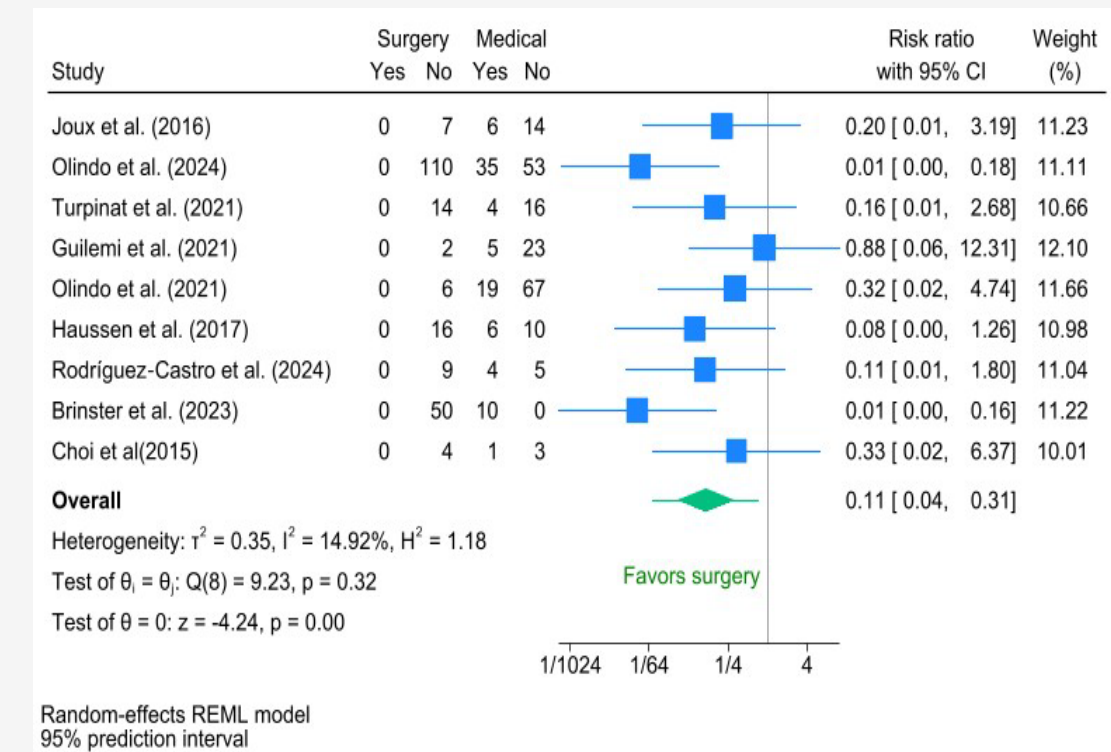


# Morphologic Features of CW

- Pooled analysis of 6 trials and registries
  - Symptomatic(n=39) vs. Asymptomatic webs(n=24)
- Angioarchitecture
  - Length size(3.4 mm vs 2.6 mm, p=0.02)
  - More acute angle(54° vs 68°, p=0.01)
  - Volume(37.9 mm<sup>3</sup> vs 25.1 mm<sup>3</sup>, p=0.01)
- Strong predictors of ischemic stroke
  - ICA-pouch tip angle
  - CCA-pouch tip angle
  - ICA web-pouch angle
  - CCA web-pouch angle



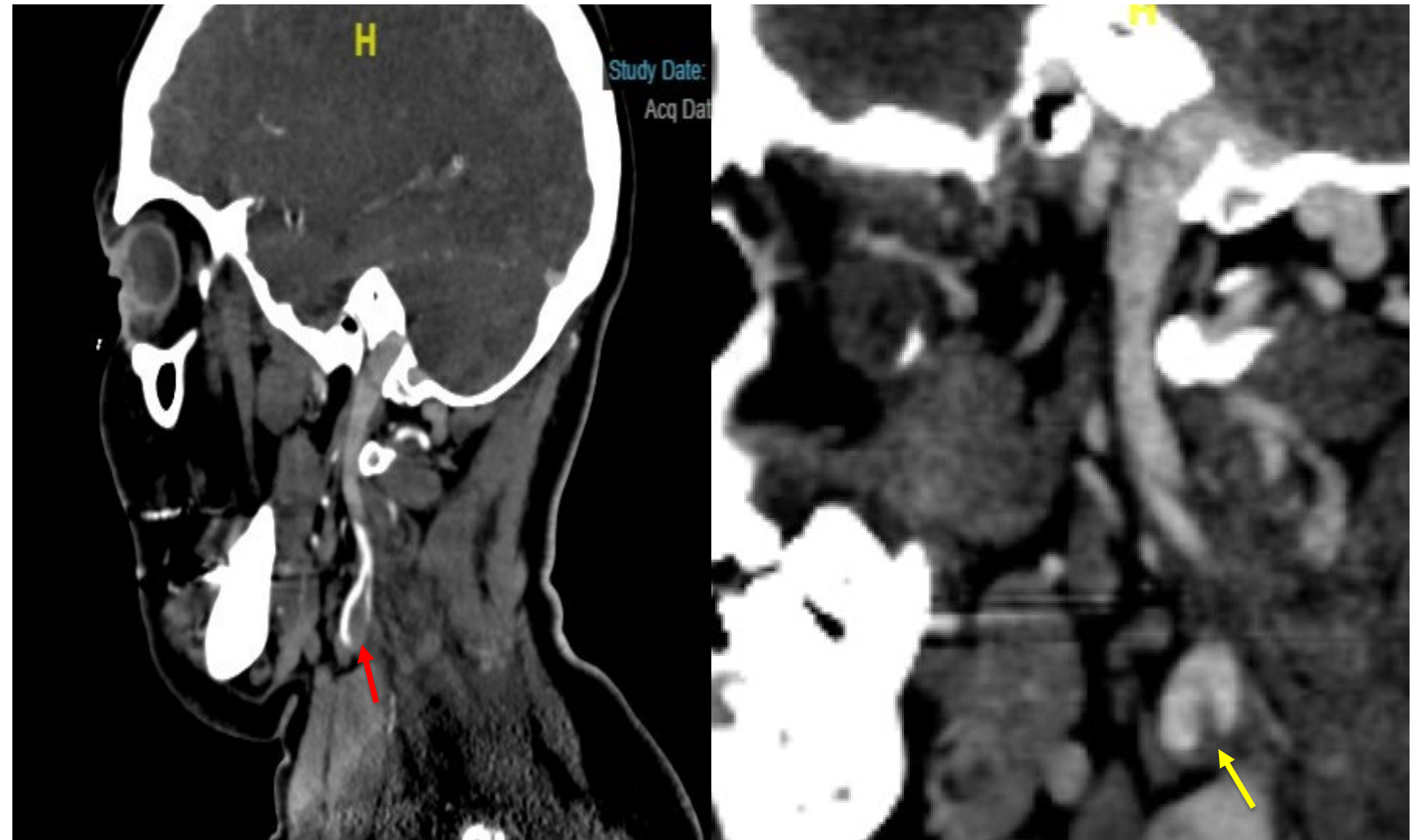
- Meta-analysis(medical vs. surgical)
- Reduced recurrence, RR 0.11; 95% CI, 0.04-0.31
- Carotid revascularization
  - CEA vs CAS vs TCAR
- CEA vs CAS
  - 287 underwent revascularization
  - 146 TFAS, 136 CEA, and 5 TCAR
  - No difference in outcomes, RR 0.44, 95% CI 0.11-1.76
  - No major complications were noted





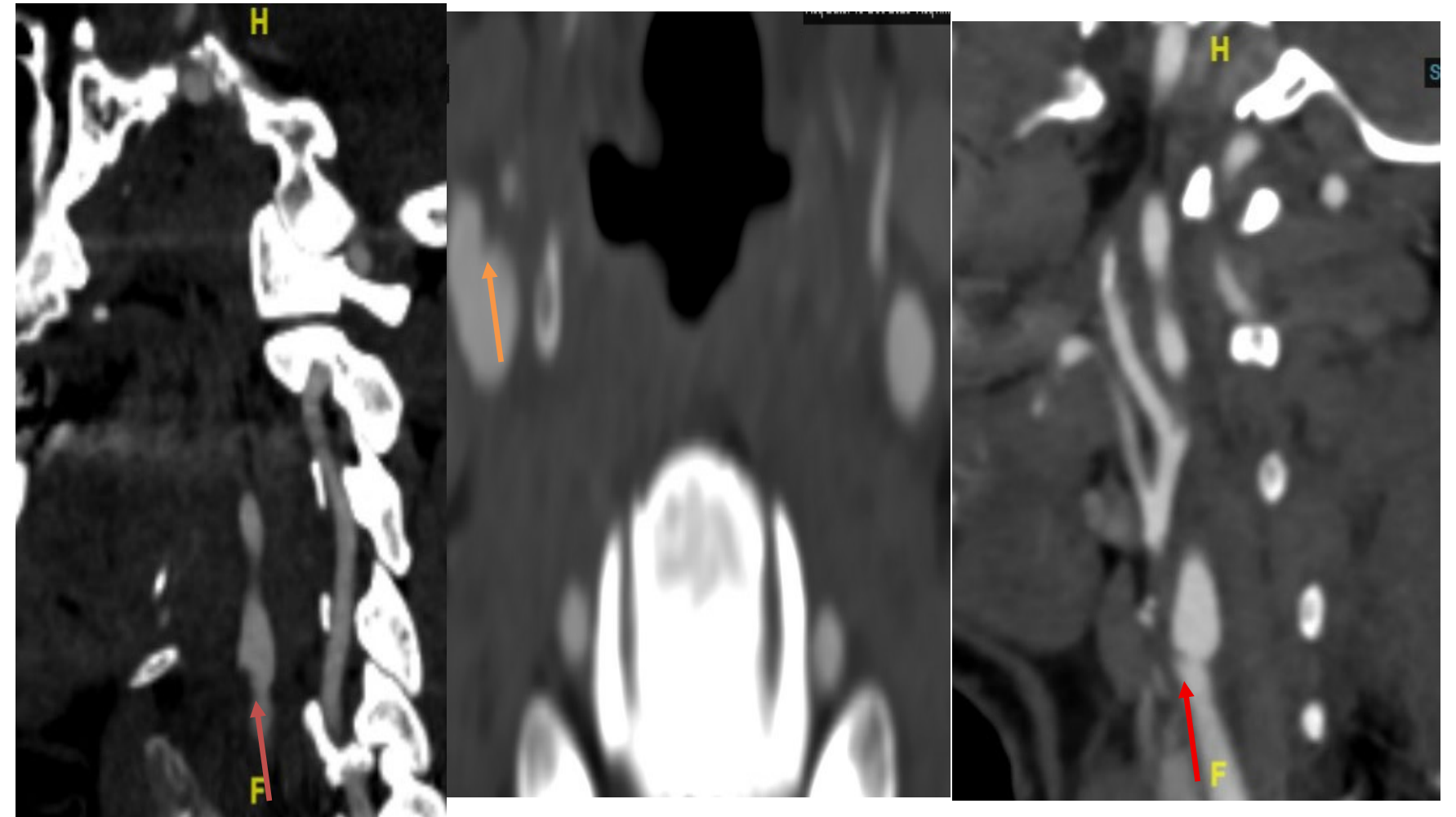
# Case 1

- 50-year-old with hx of HTN presented with mild right weakness and fluent aphasia
- NIHSS 3 on arrival
- Vessel imaging showed no LVO and intraluminal thrombus vs. ulcerated plaque in L ICA
- Heparin gtt was initiated
- At 72 hours
  - Resolution of thrombus
  - Carotid web was noted
- Management
  - Carotid revascularization(TCAR)
  - Follow up: 10 months stroke free



## Case 2

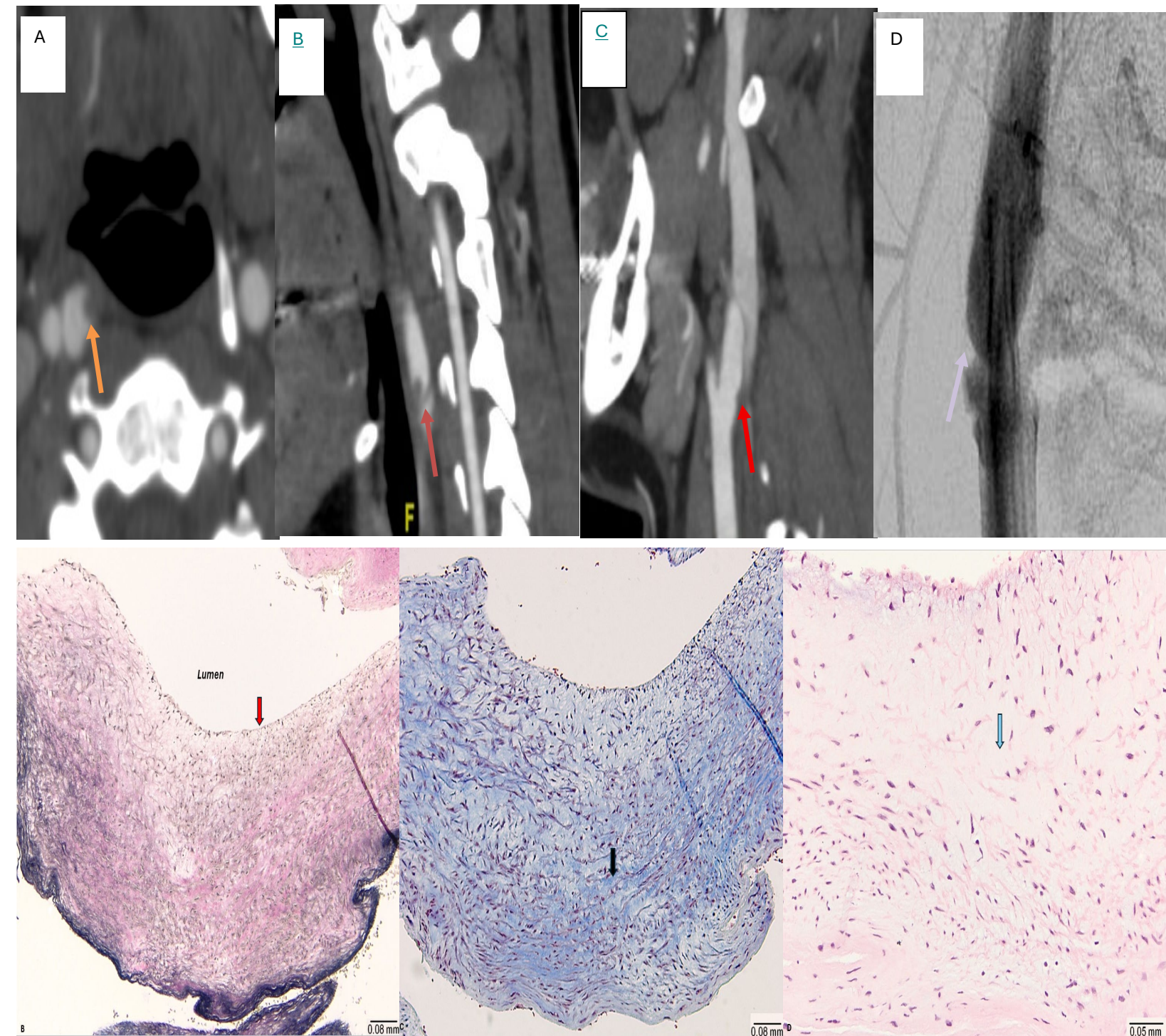
- 50 YO M presented with right MCA syndrome after a TIA three weeks ago
- Vessel imaging showed ?small CW with superimposed thrombus
- No thrombus seen on prior imaging three weeks ago (red arrow)
- Management
  - CEA
- Follow-up
  - 11 months stroke-free





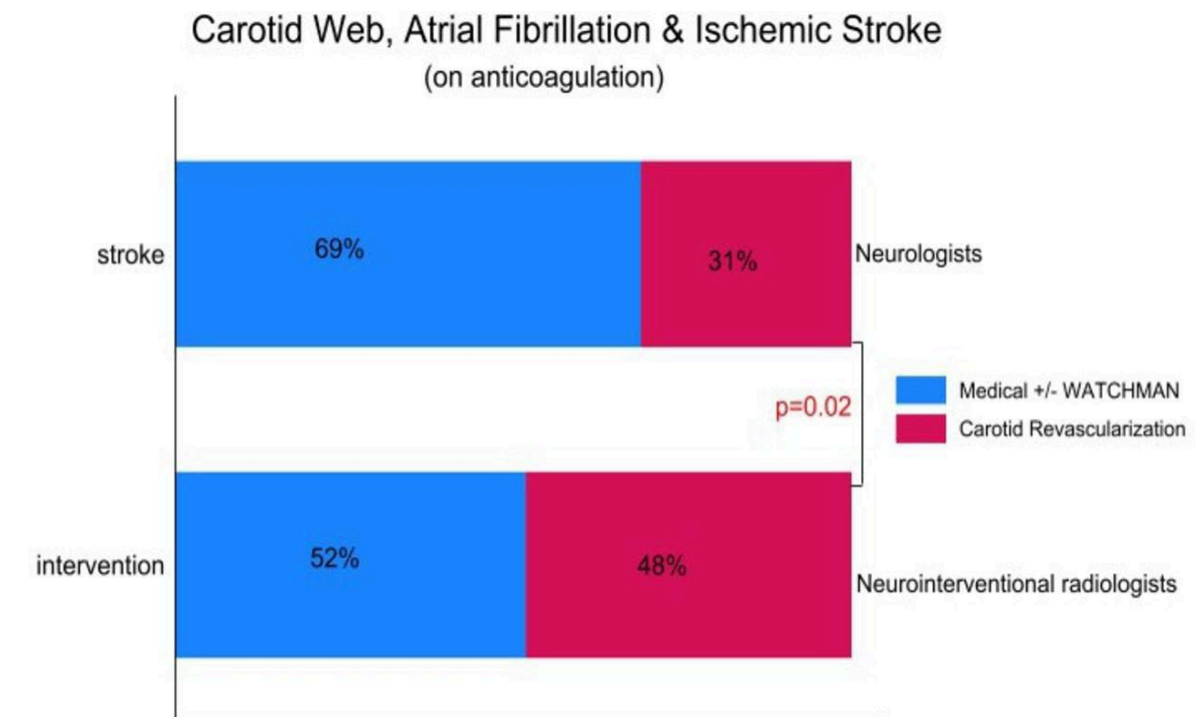
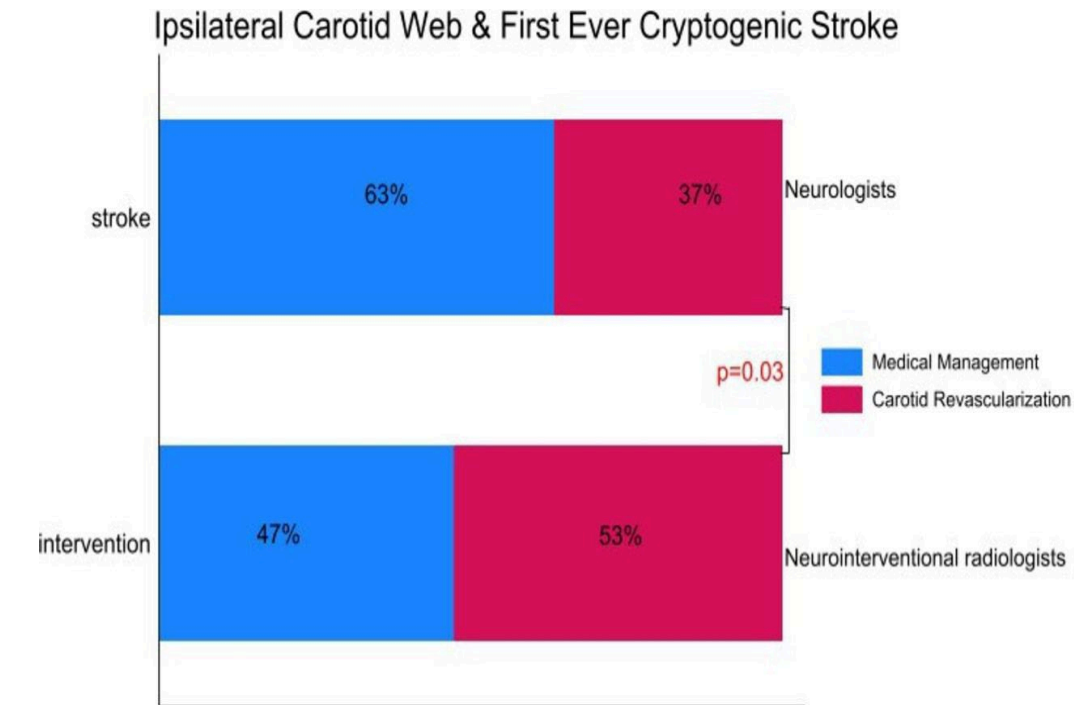
# Case 3

- 21 YO F presented with dysarthria and left-hand numbness
- OSH concern for right ICA dissection → transferred for further evaluation
- MRI brain with embolic-appearing right frontal lobe infarct
- Work-up
  - TTE, cardiac monitoring, hypercoagulable panel (incl. APS) -  
ive
- 6 months later
  - Diagnostic angiogram confirmed proximal right ICA carotid web
- Treatment
  - CEA
  - Follow up: 36 months stroke free



# Practice Patterns of VN and NIR

- Survey
  - 1660 to VN
  - NIR/NSG via SVIN and SNIS
- Cryptogenic Stroke with Ipsilateral CW
  - 53% NIR vs 37% VN,  $p=0.03$ (favored revasc.)
- Elderly Patients with AF & Ischemic Stroke
  - 48% NIR vs 31% VN,  $p=0.03$ (favored revasc.)
- TFAS as preferred technique
  - 86% NIR vs 35% VN,  $p=0.002$
- Stenosis threshold  $>70\%$  not required
  - 75% NIR vs 57% VN,  $p=0.013$



# Take-Home Message

- **CWs** are an underrecognized cause of ischemic stroke, often missed on initial imaging.
- **Diagnosis remains challenging** → modest inter-rater agreement and frequent misclassification as plaque or dissection.
- **Pathophysiology:** CW alters flow dynamics → stasis, thrombus formation, and embolization despite non-flow-limiting stenosis.
- **Neuroimaging features:** CW thrombi are more often **hyperdense, RBC-rich**, and more responsive to thrombectomy than LAA.
- **Quantitative tools:** Relative HU threshold (**rHU  $\approx$  1.14**) may improve diagnostic accuracy.
- **Management:** High recurrence risk on medical therapy alone; revascularization should be strongly considered.
- **Future directions:** Need for multicenter studies, standardized reporting, and risk stratification tools.



# Future Directions

- Large epidemiologic studies
  - SPIDER WEB
    - Retrospective multicenter study
    - Aim to have sample size ~2000 pts
    - Over 40 sites across the world
- Incorporation of CW in radiology reports
- Integration of morphologic and hemodynamic patterns
- Risk stratification scoring system



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Thank You