Evaluation

For the **Provincial Stroke Rounds Planning Committee**:

- To plan future programs
- For quality assurance and improvement

- For **You**: Reflecting on what you've learned and how you plan to apply it can help you enact change as you return to your professional duties

For **Speakers**: The responses help understand participant learning needs, teaching outcomes and opportunities for improvement.

https://forms.office.com/r/71LXRtm1b5

Please take 2 minutes to fill the evaluation form out. Thank you!

Non-atherosclerotic Carotid Bifurcation Diseases: Phenotypes and their Management

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Provincial Stroke Rounds

May 7, 2025

Disclosure of Affiliations & Financial Support

Speaker Name: Dr. Ravinder-Jeet Singh

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- *This session/program has not received financial or in-kind support.*

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Nature of relationship(s)	Name of for-profit or not-for-profit organization(s)	Description of relationship(s)
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Patents on a drug, product or device		
All other investments or relationships that could be seen by a reasonable, well- informed participant as having the potential to influence the content of the educational activity		

Learning objectives

Upon completion participants will be able to:

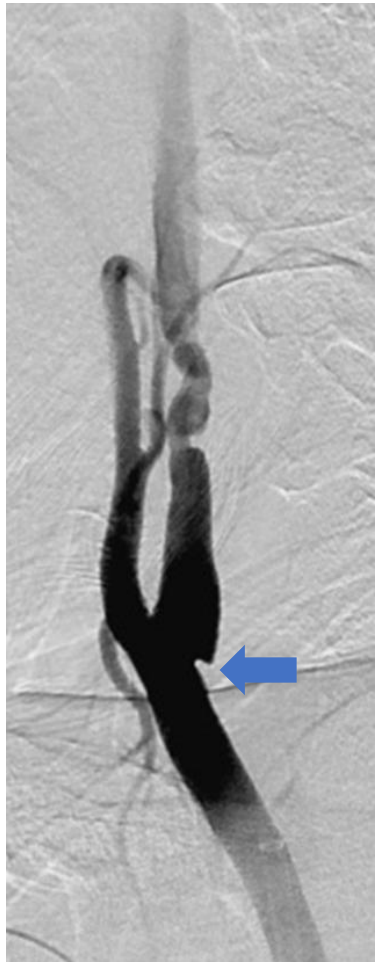
1. Explain non-atherosclerotic carotid bifurcation disease spectrum
2. Describe management of carotid webs, intraluminal thrombus, stylocarotid and TIPIC syndrome

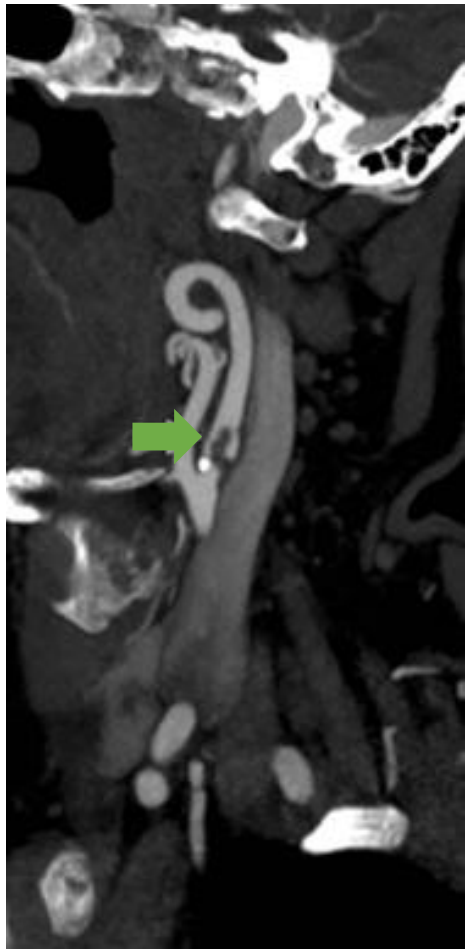
Carotid artery and stroke

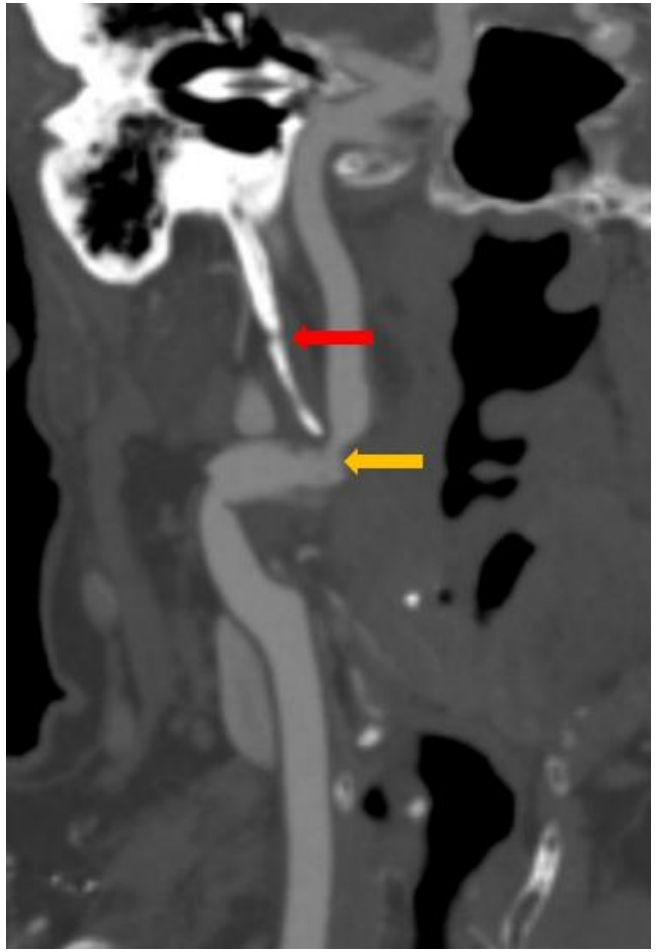
- The concept of stroke was first noted in 5th century by Hippocrates. At this time, the symptoms of convulsions and paralysis were referred to as apoplexy.
- In the 17th century, Johannes Wepfer, a Swiss physician, first demonstrated that apoplexy was caused by an intracranial hemorrhage; Thomas Willis, an English anatomist, explored the role of the cerebral arteries.
- Despite some early understanding on the role of carotid artery in stroke, it was the work by C. Miller Fisher, which led to widespread recognition of the importance and the therapeutic implication of the carotid artery in stroke.

Storey CE, Pols H. A history of cerebrovascular disease. Handb Clin Neurol. 2010;95:401-15.
Fisher CM. Occlusion of the internal carotid artery. Arch Neurol Psychiatry. 1951 65: 346–377.

Carotid bifurcation

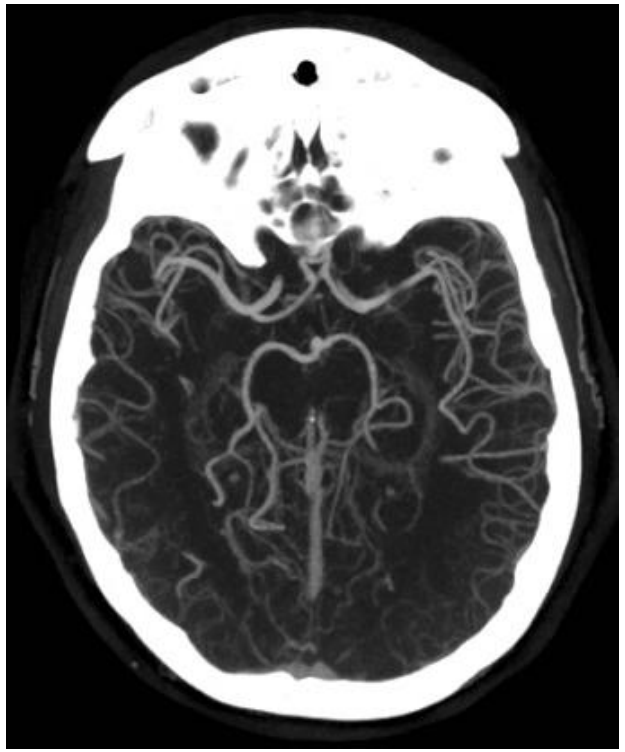
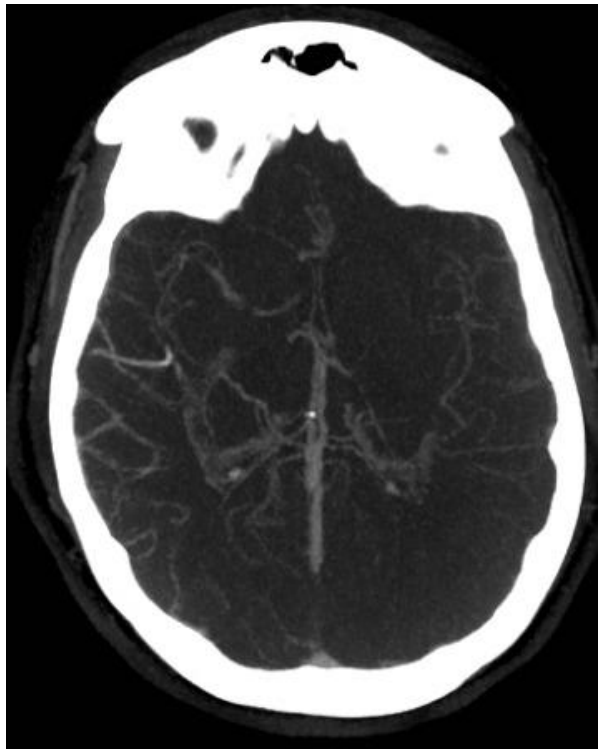
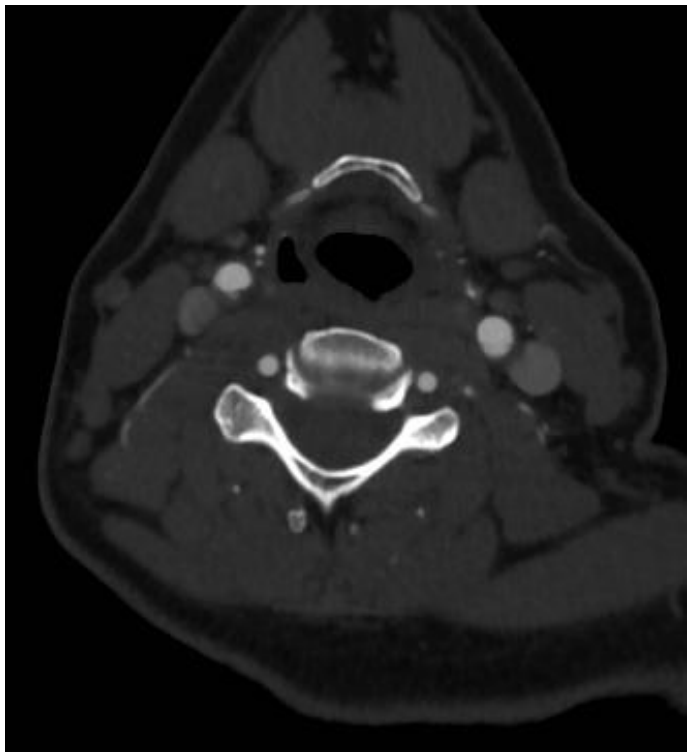




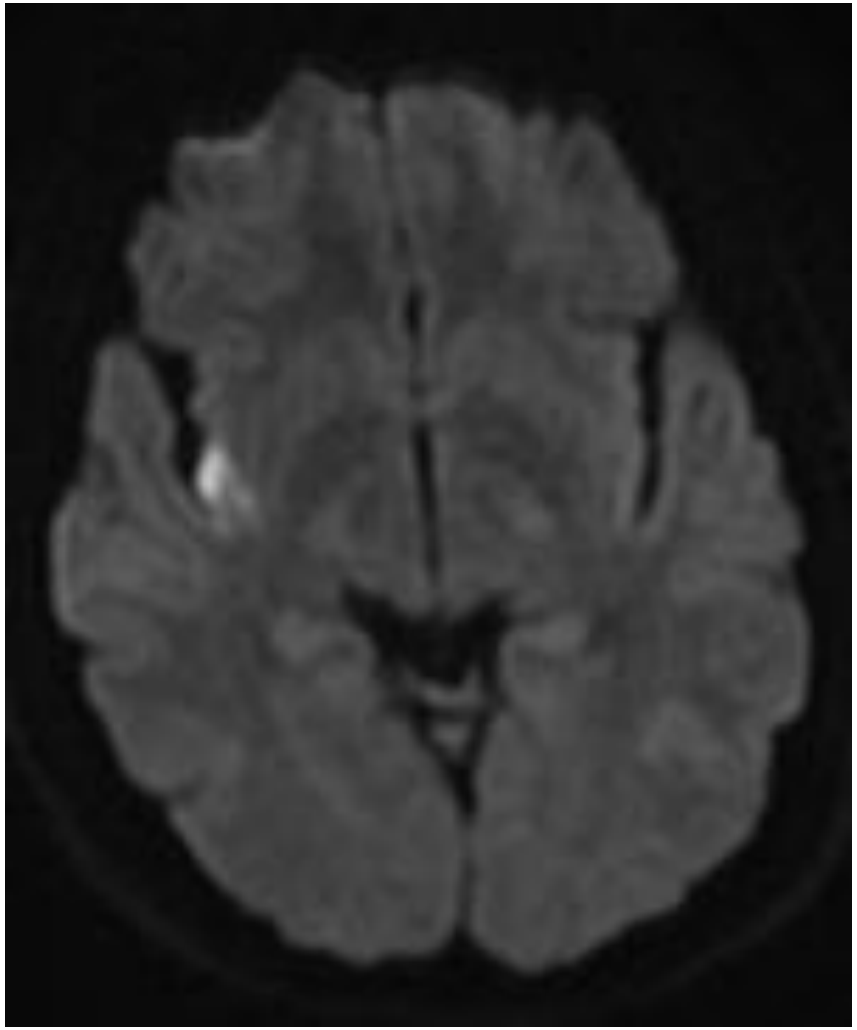


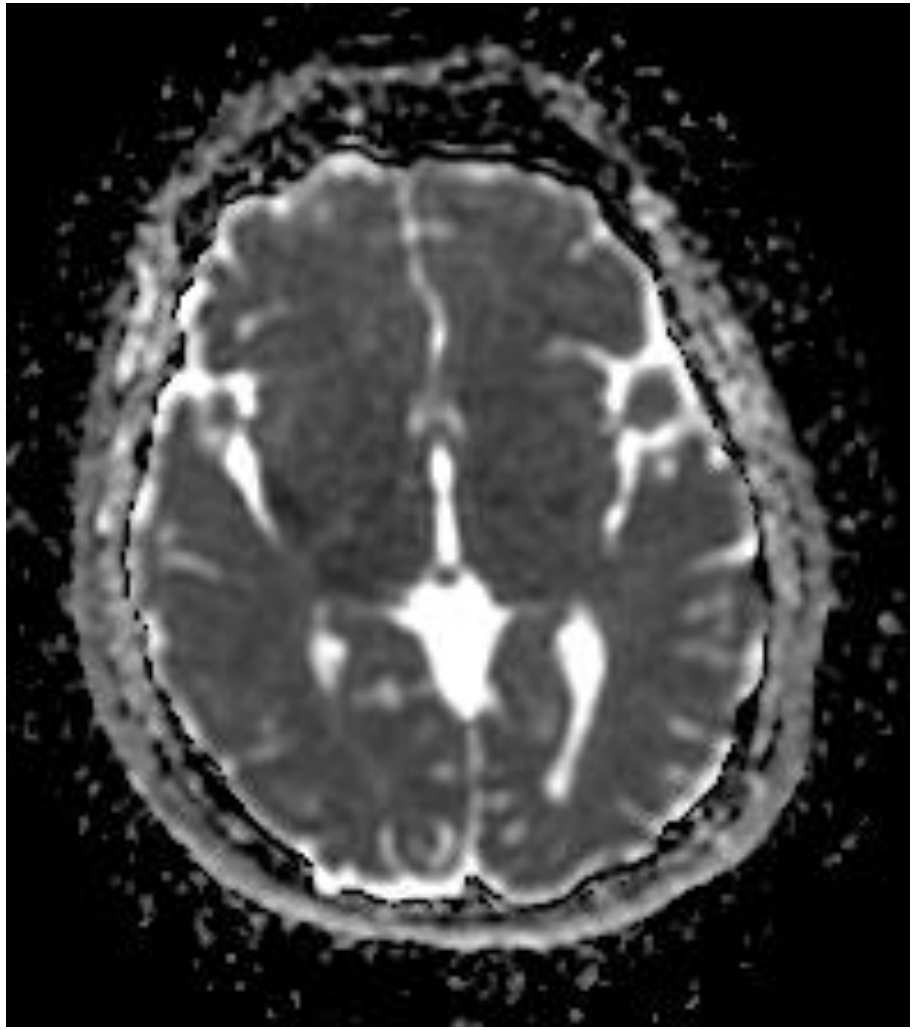
Case

- 49y RHD previously healthy female of middle eastern descent, no cardiovascular risk factors
  - Sudden onset headache, dysarthria, left sided numbness and weakness
  - Presenting to emergency department 2 hours after last seen well

Fu MRI



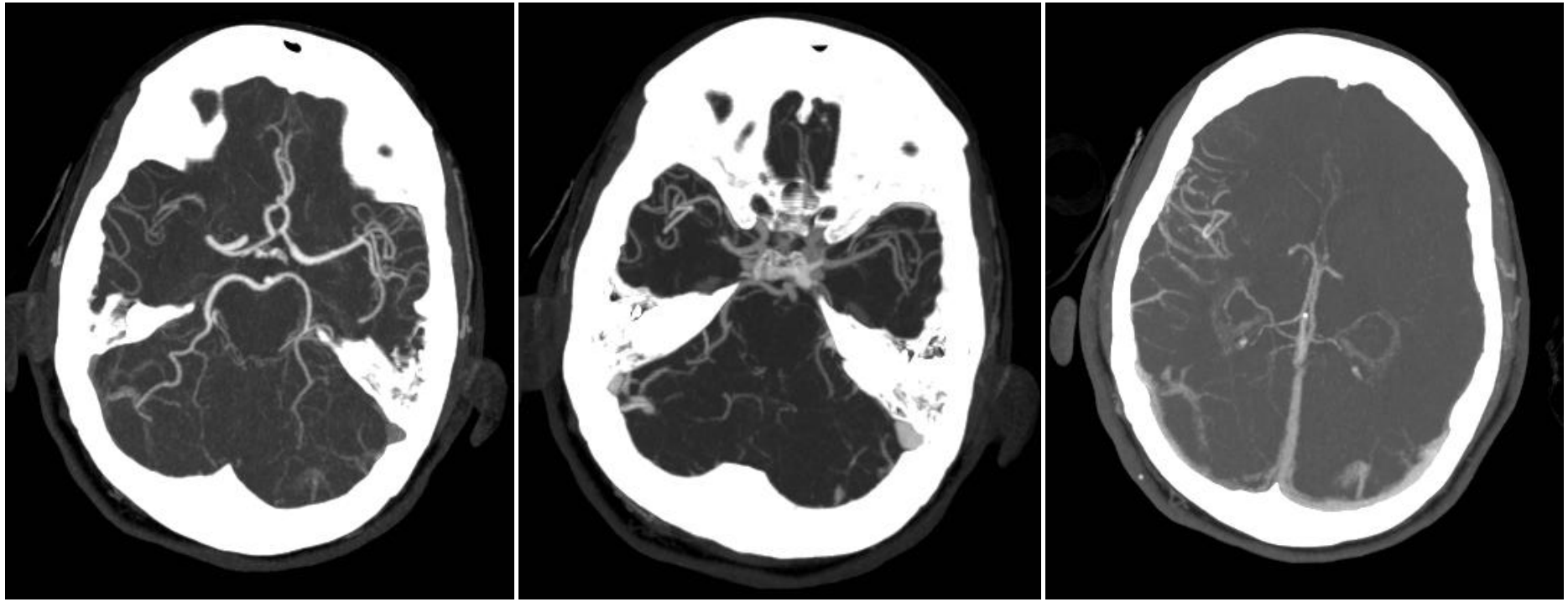


Investigations

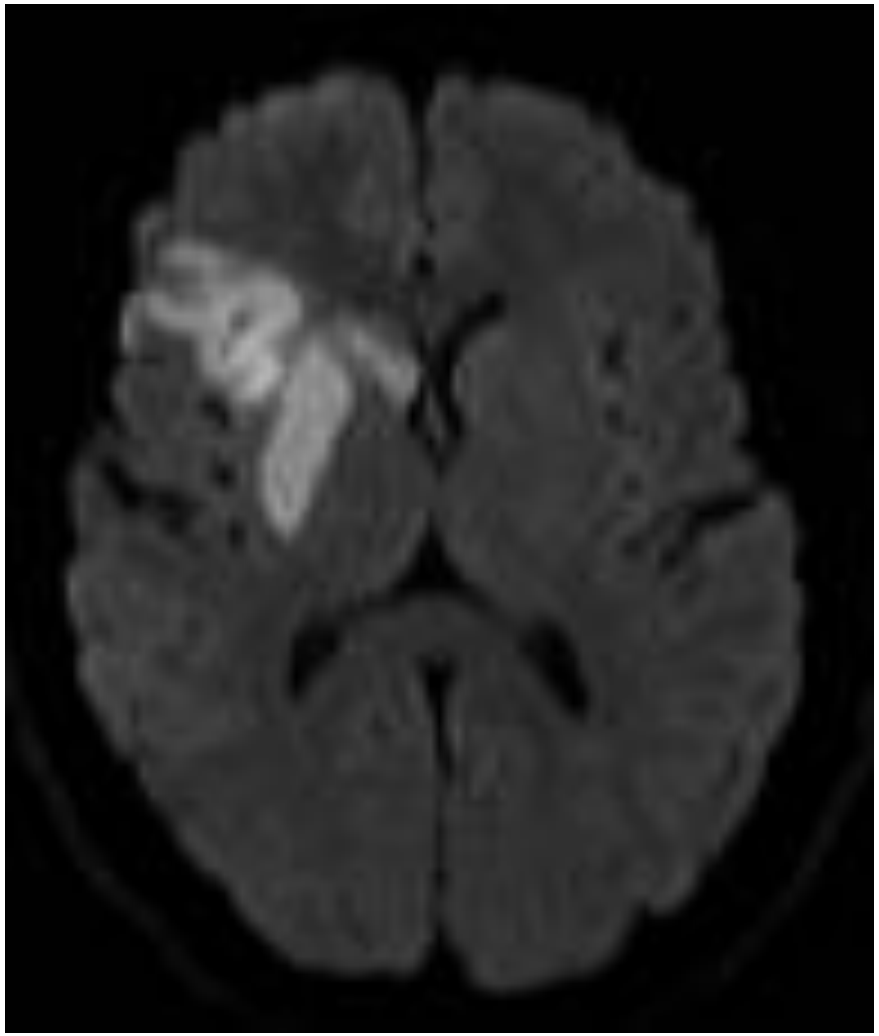
- Transthoracic echocardiogram with bubble study normal
- 24-hour Holter no atrial fibrillation
- Thrombophilia and vasculitis workups negative
- Treated with 3 week of DPAT and then ASA.

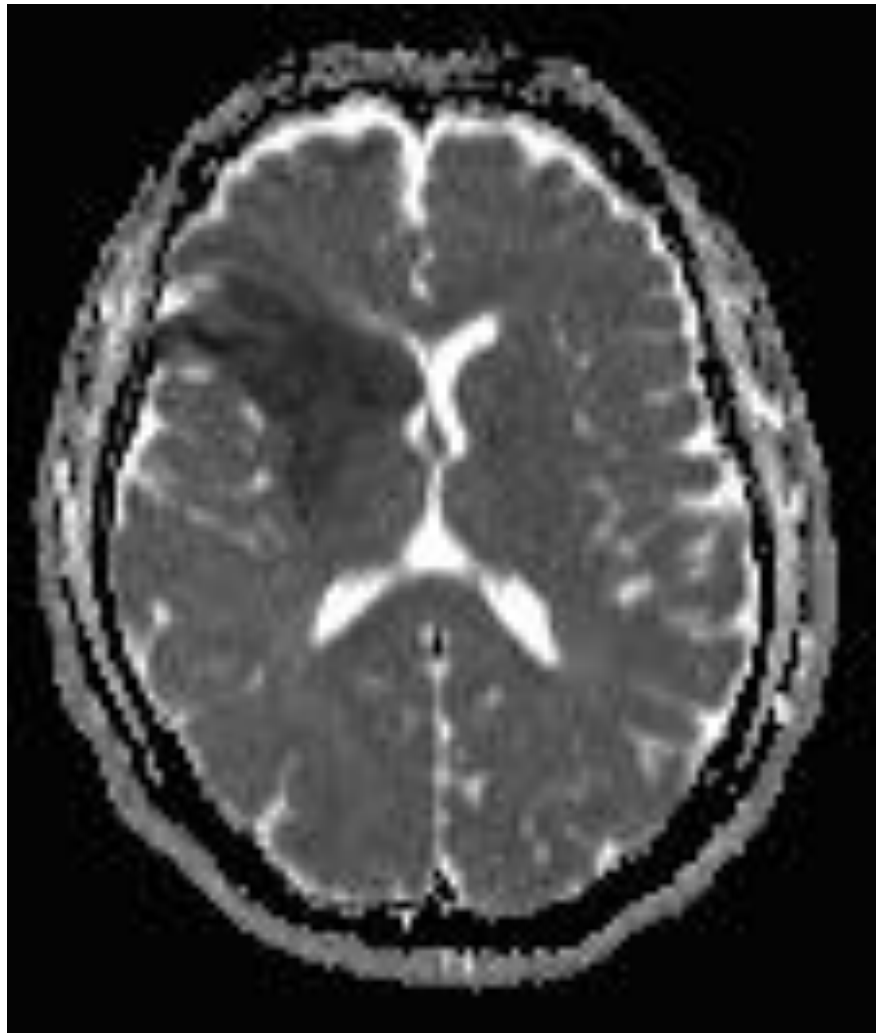
4 years later....

- Sudden onset dysarthria, left sided sensory loss and weakness at 0900h (NIHSS = 15)
- Presenting as “Stat Stroke” at 1010h

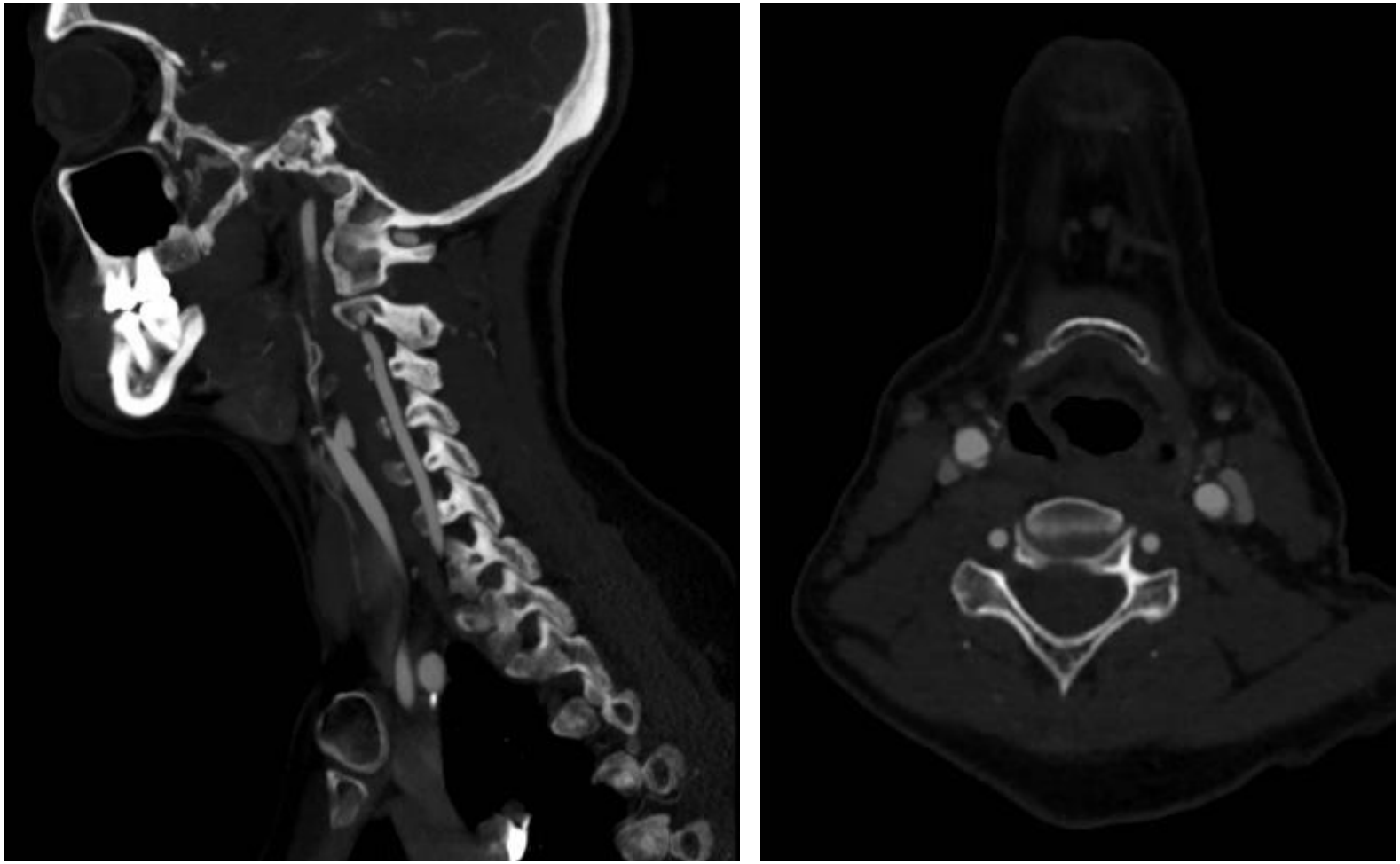


FU MRI....

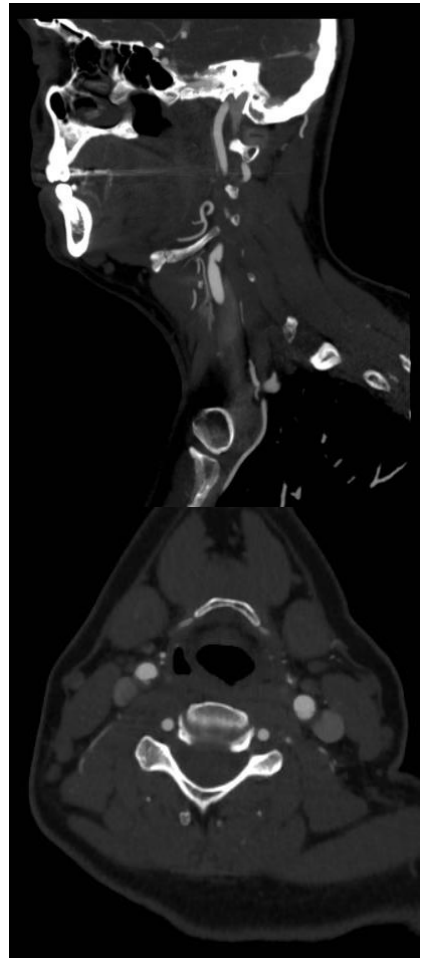




Review of CTA: current and past

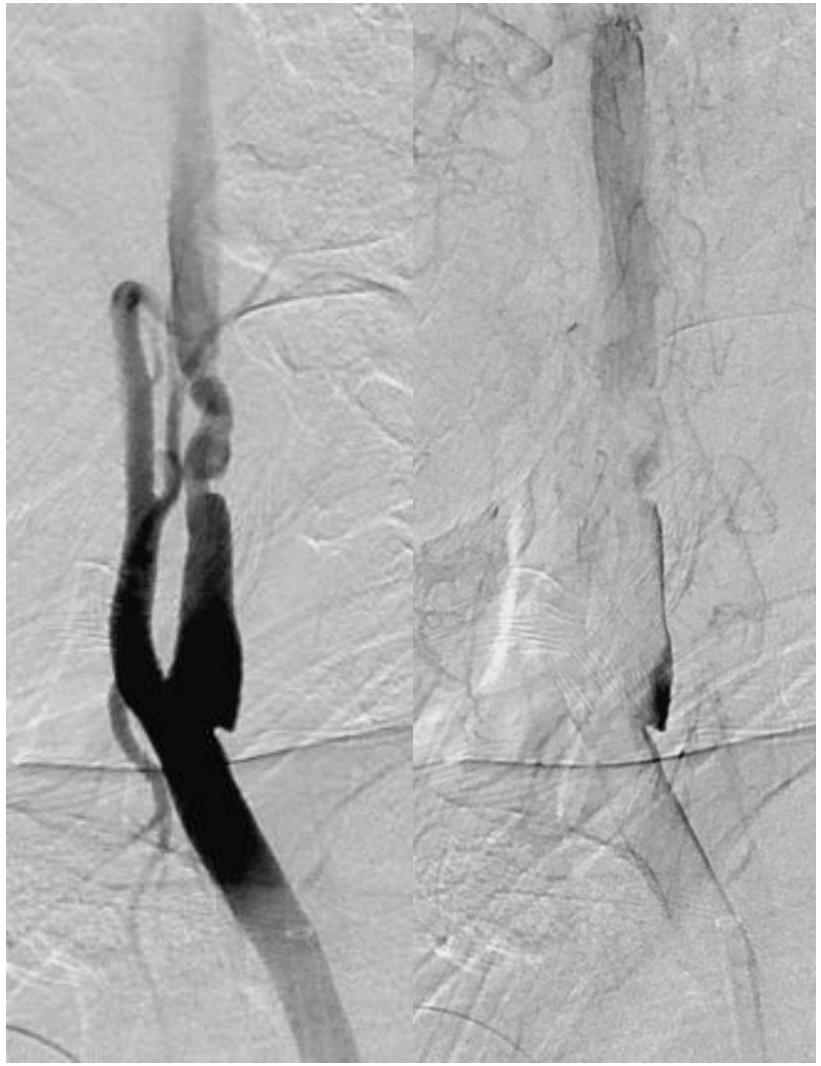


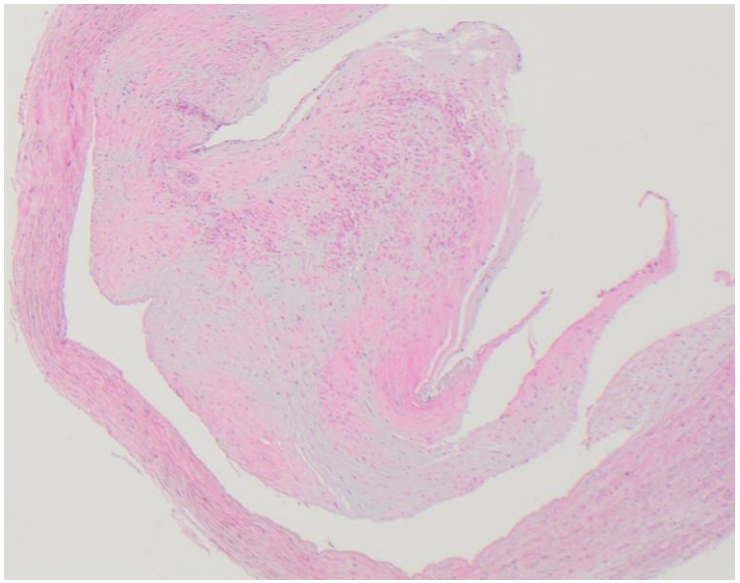
Current CTA

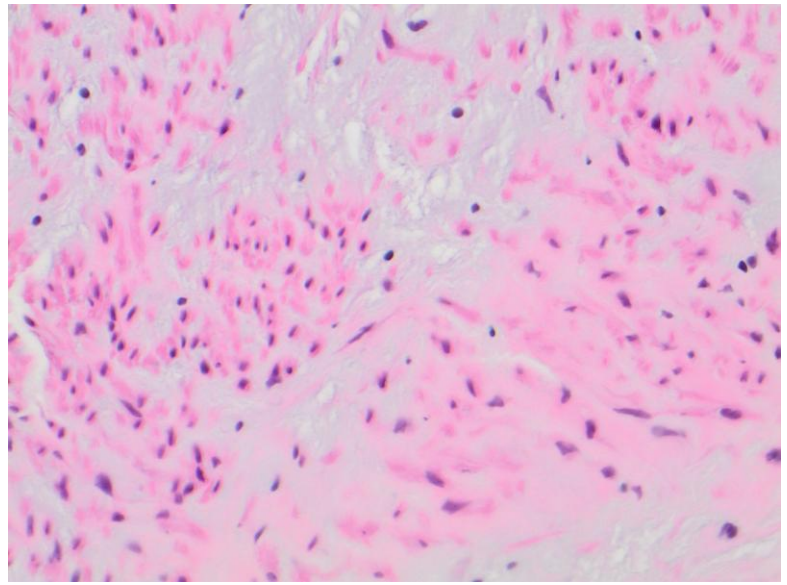


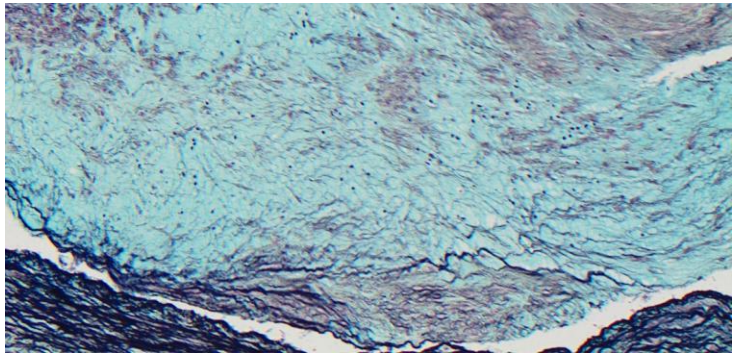
Previous CTA

Carotid web









Courtesy of: Dr. Denise W. Ng, Pathologist, University of Calgary

Non-atherosclerotic carotid pathologies

Luminal pathologies (blood)

- Intraluminal thrombus

Mural pathologies (vessel wall)

- Traumatic cerebrovascular injury
- Non-inflammatory

  - Arterial (fibromuscular) dissection

  - Fibromuscular (arterial) dysplasia: classical and intimal (carotid webs)
- Inflammatory/Vasculitis: large vessel vasculitis, radiation vasculitis, infections
- Vascular tumors: chemodectoma, paragangliomoma (carotid body tumors), regional spread of head/neck cancer

Extramural disorders

- Carotidynia or TIPIC syndrome
- Stylo-carotid syndrome (osseo-vascular conflict)
- Carotid blow out syndrome: radical neck surgery, post-radiation (necrosis), infections of skin/soft tissue

Extension of intracranial or extracranial equivalent of classically intracranial pathologies

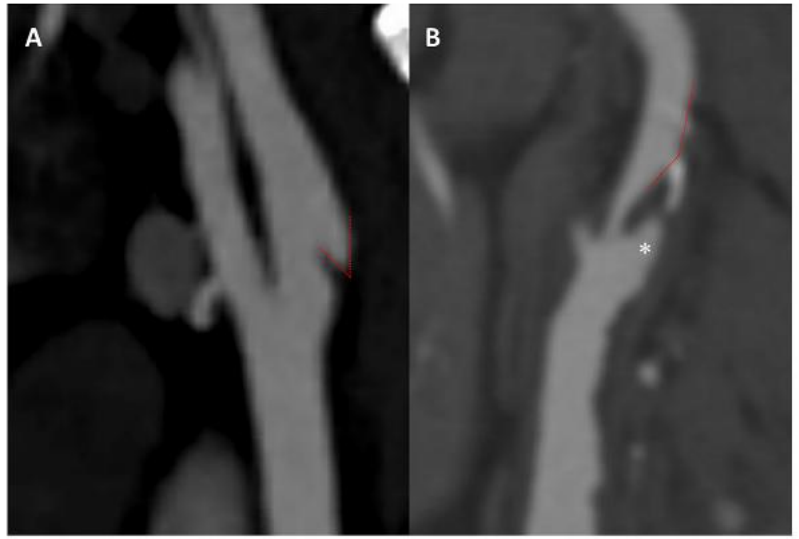
- Carotid spasm (RCVS)
- Carotid Moyamoya
- Carotid spasm (RCVS)

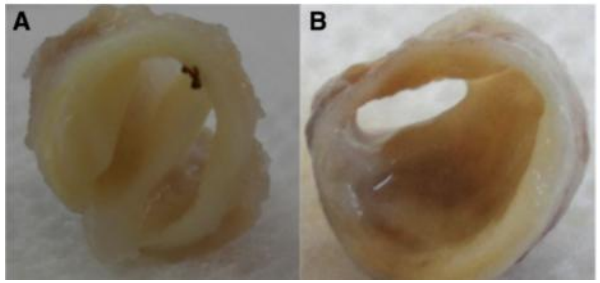
Other conditions

- Aplasia or hypoplasia (Developmental)
- Dilatative arteriopathy or dolichoectasia and related phenotypes (kinking, coiling, loops)

Carotid web

- First angiographic and pathologic description consistent with carotid web: Rainer et al in 1968.
- The term “web-like tissue” was used by Momose and New.
- Other terms: septal fibromuscular dysplasia, atypical FMD, carotid-bulb atypical FMD, carotid artery web, carotid pseudovalvular fold, thrombotic carotid megabulb, carotid bulb diaphragm, and intimal FMD.
- Nonatherosclerotic fibrointimal membrane projecting into the lumen of one or both carotid bulbs. Pathology showed dysplastic tissue- now considered intimal FMD variant.





Singh RJ, Menon BK. A Carotid Web with Atherosclerotic Plaque. *Ann Neurol*. 2023;94:203-204.
Stroke. 2014;45:3711-3713; AJNR. 2015;36:2134-2139

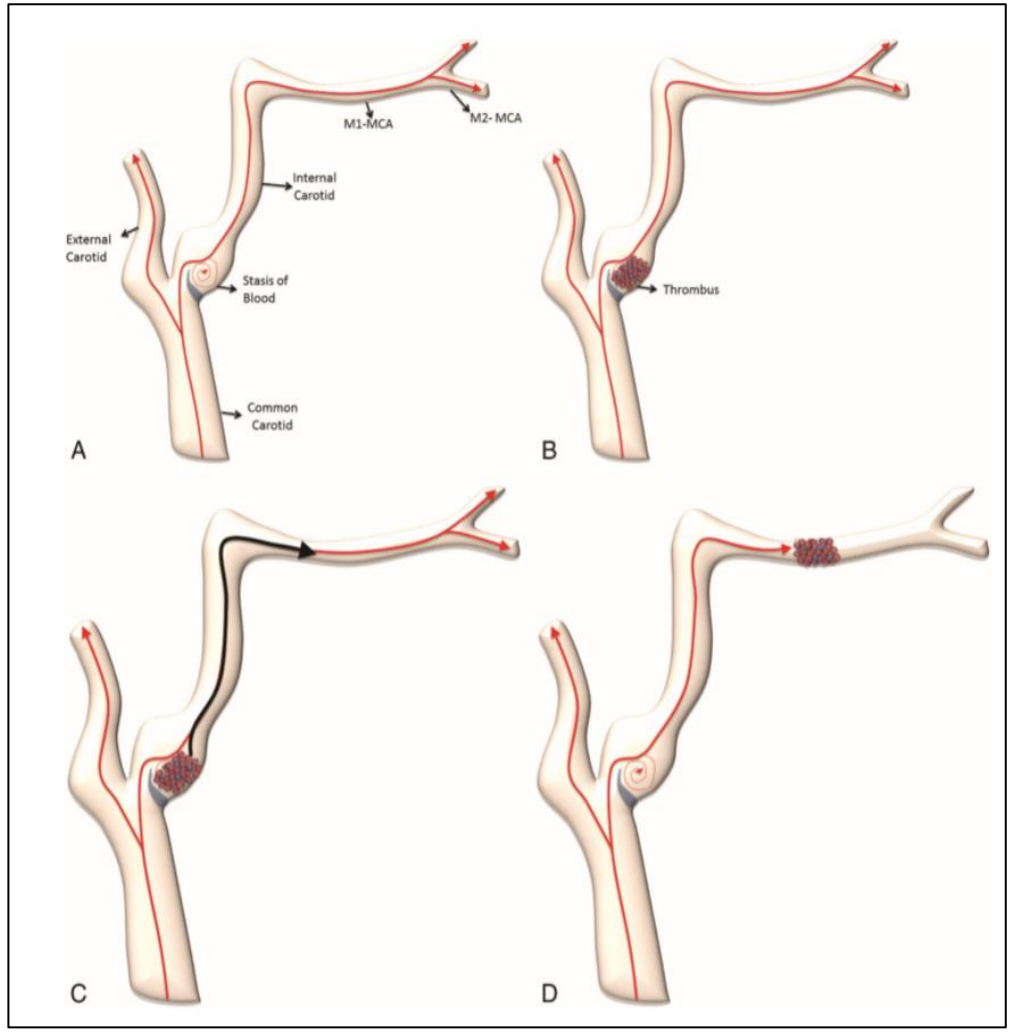
Prevalence

Study	Population; age criteria, y	n=	Prevalence, n (%)
Catheter angiogram			
Momose and New, 1973	CE for various indications; NS	7,000	4 (~0.06)
Osborn and Anderson, 1977	Fibromuscular dysplasia; NS	25	1 (~4.0)
So et al, 1981	CE for various indication; NS	6,100	4* (~0.07)
CT angiogram			
Choi et al, 2015	Suspected stroke; NS	576	7 (1.2)
Joux et al,	Carotid distribution IS; <55	43	Ischemic stroke patients Symptomatic territory: 10 (23.3) Asymptomatic territory: 2 (4.7) Cryptogenic stroke patients: 10/27 (37)
Coutinho et al, 2017	Anterior circulation IS; <60	53	4 (9.4)
Sajedi et al, 2017	Cryptogenic IS; 18-55	33	7 (21.2)
Haussen et al, 2017	Cryptogenic IS; <65	64	11 (17)
Compagne et al, 2018	IS with intracranial large vessel occlusion; ≥18	443	Symptomatic territory: 11 (2.5) Asymptomatic territory: 2 (0.5)

Association between carotid web and stroke

Study	Population	Age, y	Cases n/N (%)	Controls n/N (%)	OR 95% CI	P-value
Joux et al, 2016	Afro-Caribbean cohort Cases= patients with carotid IS Controls= patients with no history of stroke admitted with head trauma	<55	All patients			
			10/43 (23.3)	3/43 (7.0)	9.47* 1.71-52.21	0.035
			Cryptogenic stroke patients			
			10/27 (37)	2/27 (7%)	24.06* 1.78-325.63	0.016
Coutinho et al, 2017	Cases=IS of undetermined cause in the anterior circulation Controls= aneurysm, AVM, ICH	<60	4/53 (9.4%)	1/102 (1.0%)	8.0 1.2-67	0.032
Sajedi et al, 2017	Cases= cryptogenic IS in the anterior circulation Controls= CTA for non-stroke indication (eg trauma)-age, sex, ethnicity matched	18-55	7/33 (21.2%)	1/63 (1.6%)	16.7 2.78-320.3	0.01

Pathophysiology of thromboembolism in carotid web



Clinical presentation

- Demographic features
  - Most patients <60 years (all histopathological confirmed cases <60 years)
  - More common in women
  - More common in Afro-Caribbeans

- Clinical presentation
  - Asymptomatic
  - TIA
  - Stroke (most reported presentation ?selection bias)
  - Conventional risk factors often absent

Nearly 50% of symptomatic web have contralateral web.

Asymptomatic webs are often much smaller and more often seen in males.

Recovery and prognosis

- Functional outcomes after stroke: Generally, appears favorable and mRS 0-2 is achieved in 80-96%.
- Recurrence rate: 17 to 71% patients develop recurrent symptom on medical therapy.

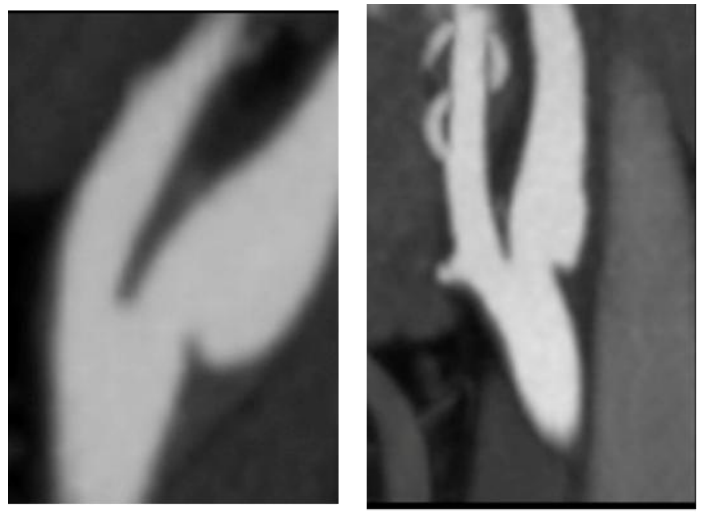
- MR CLEAN registry
  - During 2 years of follow-up, 5 of 30 patients (17%) with CW had a recurrent stroke compared with 5 of 168 patients (3%) without CW (adjusted hazard ratio, 4.9; 95% CI, 1.4-18.1).

Neurology. 1999;52:883-886; Stroke. 2014;45:3711-3713; AJNR. 2015;36:2134-2139; Interv Neurorad. 2016;22:432-437; Stroke. 2017;48:3134-3137, JAMA Neurol. 2021 1;78:826-833.

Diagnosis

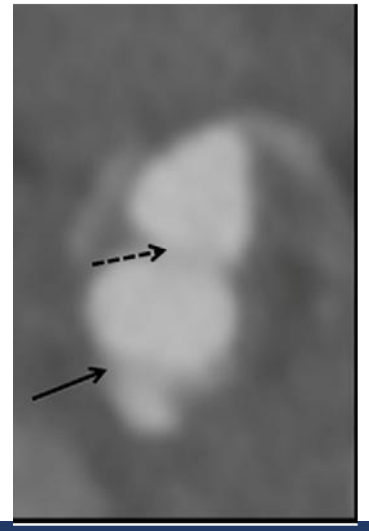
CT angiography

- Oblique sagittal section
- thin intraluminal filling defect along the posterior or posterolateral wall of the carotid bulb just beyond the carotid bifurcation



Axial section

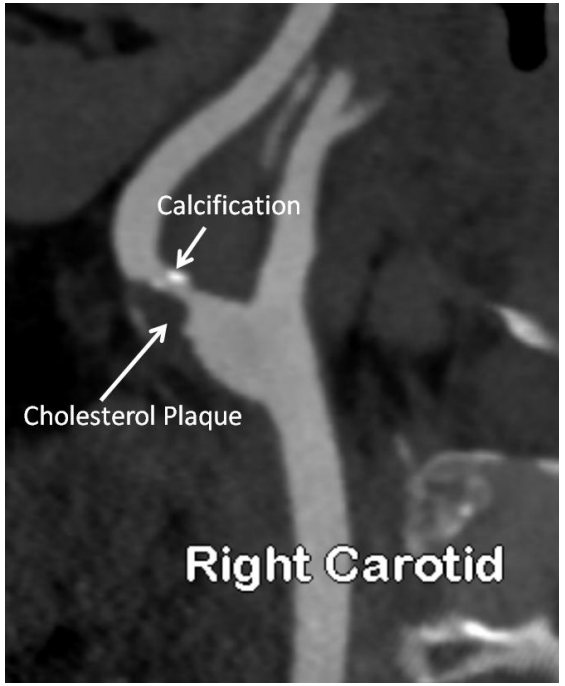
- Septum: three lumen sign

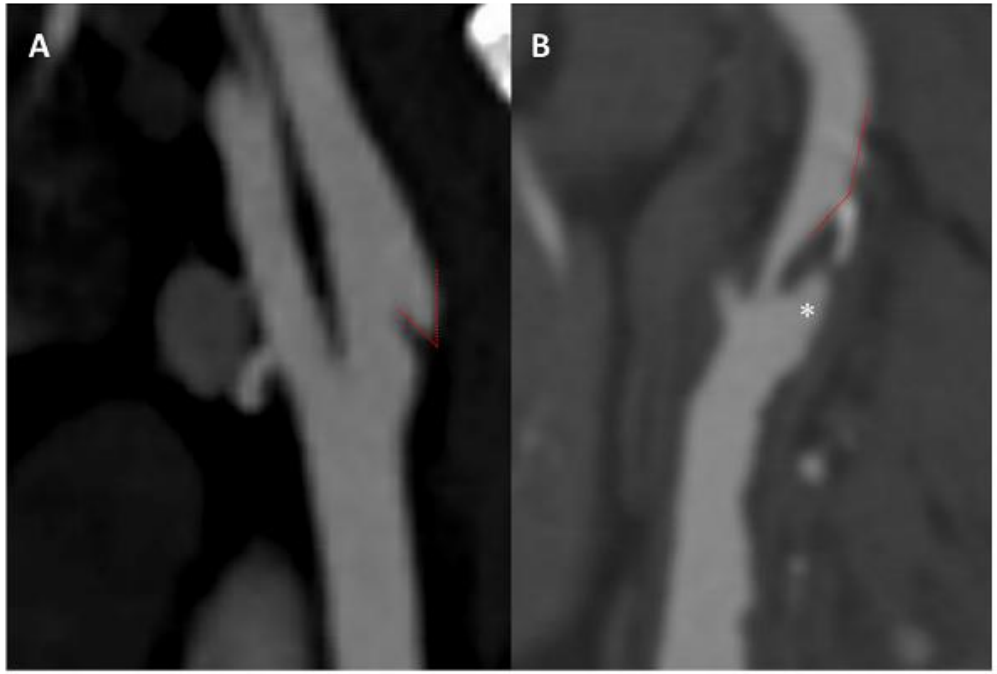


Differential

- Protruding plaque
- Atherosclerotic diaphragms

Differentials



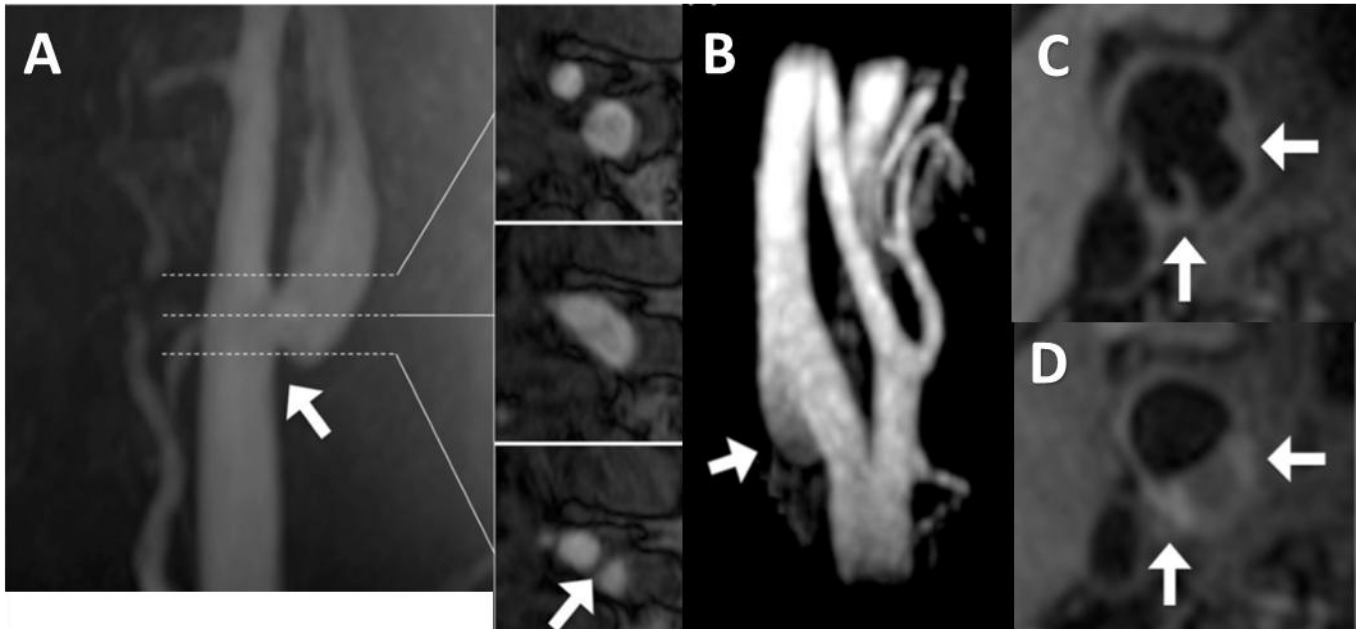


Singh RJ, Menon BK. A Carotid Web with Atherosclerotic Plaque. *Ann Neurol*. 2023;94:203-204.

http://neuroangio.org

MRI

- Conventional MRI
  - mildly hyper-intense signal on T1W, homogenously iso-intense signal on T2W, minimal enhancement of inner layers on post-gadolinium imaging; pooling contrast sign
- MR angiography
  - Typical appearance as in CTA

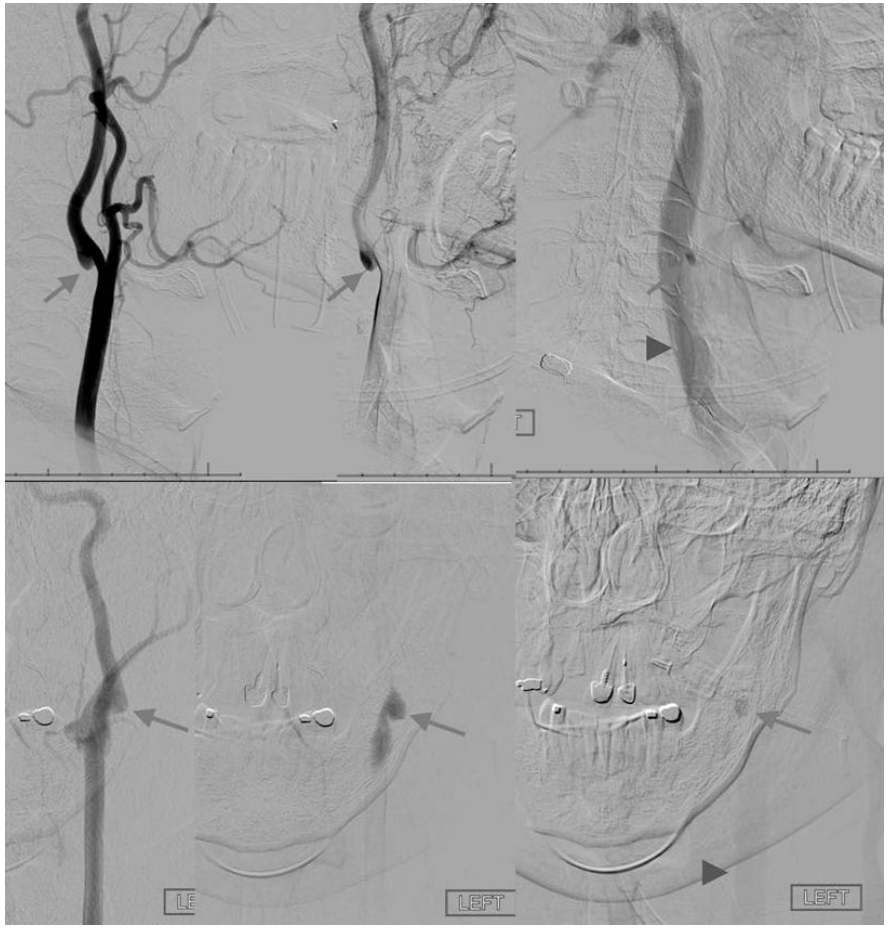


Neuroradiology. 2017;59:361-365

DSA

- Catheter angiography: septum or shelf-like filling defect in the carotid bulb immediately after the bifurcation

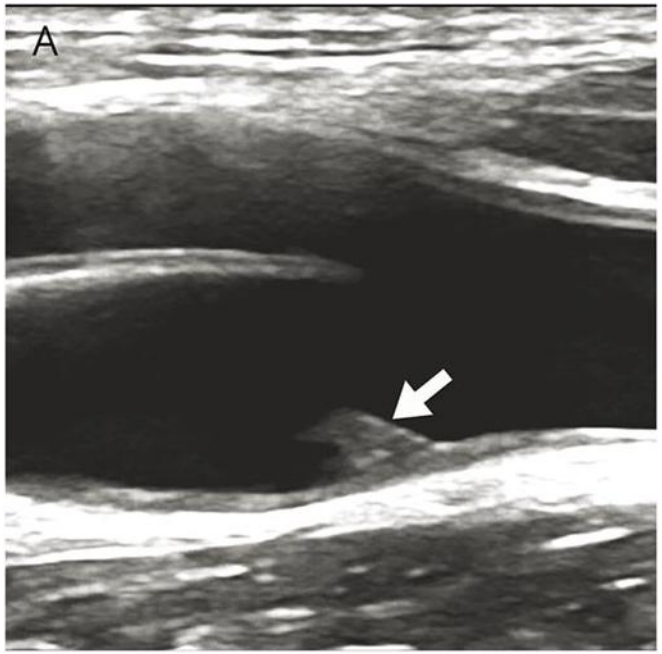
- Pooling contrast sign



Stroke. 2017;48:3134-3137

US

- On carotid ultrasound the web is seen as a mild bulbar growth.
- The changes might be subtle and could be easily missed.
- In one study, 18 of 21 (~86%) patients who underwent ultrasound had inconspicuous bulbar growth
- Differential: fibrous plaque
- False negative: superimposed thrombus causing mixed echogenicity



Neurology Apr 2018, 90 (17) e1541

Treatment

Treatment	Patients With Symptomatic Carotid Web, n (%)	Recurrent Stroke, n (%)	Months to Recurrent Stroke (Median, Range)	Months Stroke Free If No Stroke Recurrence (Median, Range)
Medical management*	47/97 (53)	25/45 (56)	12 (0–97)	24 (14–100)
Antiplatelet	43/47 (91)	22/41 (54)	12 (0–97)	22.5 (14–100)
Anticoagulation	4/47 (9)	3/4 (75)	0.2 (0.2–6)	48 (48–48)
Carotid revascularization (all)†	70/97 (72)	0/42 (0)	‡	14 (3–144)
Carotid artery stent	35/70 (50)	0/25 (0)	‡	10.7 (3–144)
Carotid endarterectomy	35/70 (50)	0/17 (0)	‡	14 (6–120)

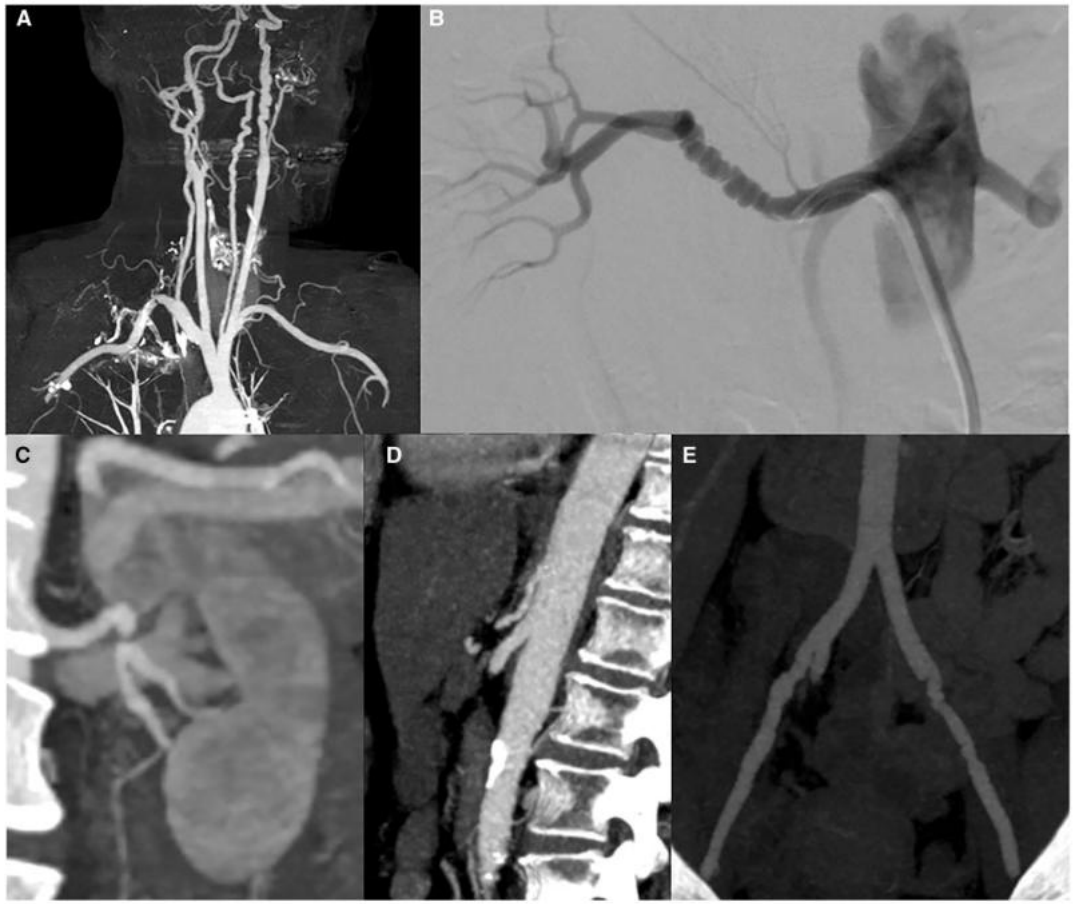
Zhang AJ, et al. A Systematic Literature Review of Patients With Carotid Web and Acute Ischemic Stroke. Stroke. 2018;49:2872-2876..

Fibromuscular dysplasia

- Fibromuscular dysplasia (FMD) is nonatherosclerotic,noninflammatory vascular disease.[1]
- Prevalence: Estimated at 12.0 per 100,000 but much higher prevalence of renal FMD in other studies.[2] In one of the largest series, 58% involved the renal artery, 32% involved the carotid/vertebral artery, and 10% involved other arteries - iliac artery or intracranial vessels.
- More common in younger women.
- Etiology: genetic, environmental or multifactorial.
- Mechanism of stroke:
  - Ischemic- arterial stenosis, occlusion, dissection and webs
  - Hemorrhagic: aneurysm or dissection.
- Clinical presentation
  - Usually asymptomatic or non-ischemic symptoms- headache, dizziness, tinnitus or bruit.
  - Vascular presentation is variable: TIA/IS, SAH

1. Olin JW, Gornik HL, Bacharach JM, et al. Fibromuscular dysplasia: state of the science and critical unanswered questions: a scientific statement from the American Heart Association. Circulation. 2014;129:1048-1078.
2. Rana MN, Al-Kindi SG. Prevalence and manifestations of diagnosed fibromuscular dysplasia by sex and race: Analysis of >4500 FMD cases in the United States. *Heart Lung*. 2021;50:168-173.

Diagnosis: CUS, CTA, MRA or DSA



Pappaccogli M, Di Monaco S, Warchoł-Celińska E, et al. The European/International Fibromuscular Dysplasia Registry and Initiative (FEIRI)-clinical phenotypes and their predictors based on a cohort of 1000 patients. Cardiovasc Res. 2021;117:950-959. Stroke. 2014;45:3711-3713; AJNR. 2015;36:2134-2139

Management

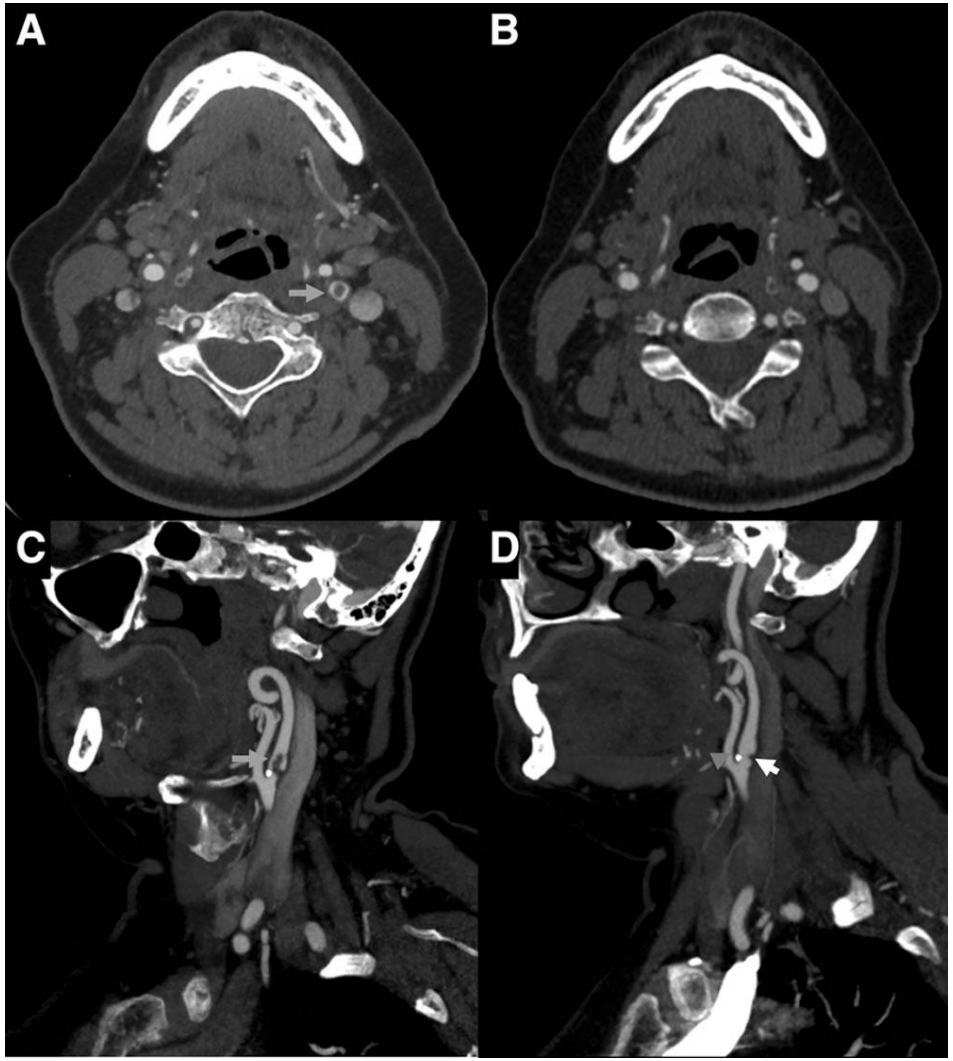
- No randomized data to guide management.
- General approach:
  - Prevention: avoid triggers for dissections
  - Medical therapy: antiplatelet or anticoagulation
  - Endovascular: stenting
  - Surgical: occasionally needed

Intraluminal thrombus (Free floating thrombus or nonocclusive thrombus)

- Prevalence ranging from 0.4% to 3.2%. Most common location of ILT is carotid bifurcation (~2/3rd).

- This phenomenon has also been termed intraluminal clot, intraluminal nonocclusive thrombus, free-floating thrombus, and colloquially the doughnut sign.

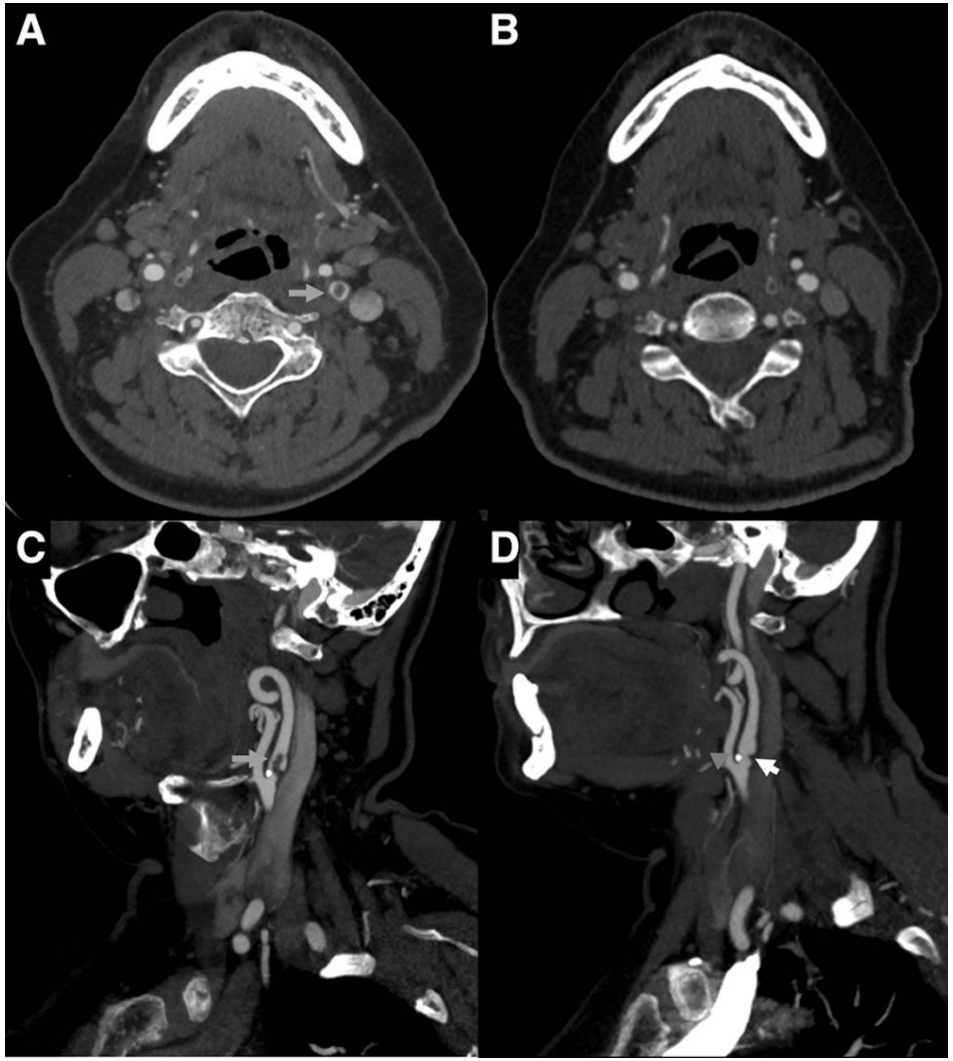
TOAST classification, n (%)	
Large-artery atherosclerosis	42 (71.2)
Cardioembolism	1 (1.6)
Small-vessel occlusion	0 (0)
Other determined cause	4 (6.6)
Undetermined cause	10 (16.4)



Singh RJ, Chakraborty D, Dey S, et al. Intraluminal Thrombi in the Cervico-Cephalic Arteries. Stroke. 2019;50:357-364.

Intraluminal thrombus (Free floating thrombus or nonocclusive thrombus)

- Mechanism of stroke: embolic (>90%), hemodynamic or mixed.
- Diagnosis: CUS, CTA, MRA or DSA



Singh RJ, Chakraborty D, Dey S, et al. Intraluminal Thrombi in the Cervico-Cephalic Arteries. Stroke. 2019;50:357-364.

Treatment

- Treatment variable: anticoagulation (UFH or LMWH) without or with single antiplatelet.
  - The initial treatment strategy was primarily a combination antithrombotic regimen of intravenous unfractionated heparin and 1 antiplatelet agent (either aspirin or clopidogrel) in 93.4% patients
- Prognosis: almost 75% of thrombi resolve either partially or completely within the first week. Recurrent stroke risk is low on combination antithrombotic therapy.

› Can J Neurol Sci. 2024 Nov 27:1-8. doi: 10.1017/cjn.2024.348. Online ahead of print.

Management Approaches to Intraluminal Thrombi in Acutely Symptomatic Carotid Stenosis

Davis MacLean [1 2], Benjamin Beland [2], Gordon A E Jewett [2], Luca Bartolini [3], David J T Campbell [1 4 5], Malavika Varma [1 2], Ravinder-Jeet Singh [6], John H Wong [1], Bijoy K Menon [1 4 7 8], Aravind Ganesh [1 4 7]

Affiliations + expand

PMID: 39592265 DOI: 10.1017/cjn.2024.348

Results of this study demonstrate a preference for anticoagulation and delayed revascularization after reimaging to examine for clot resolution, though much equipoise remains.

Outcome

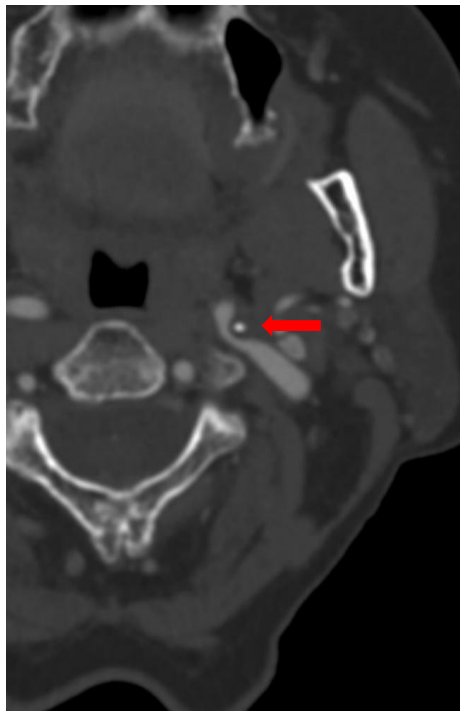
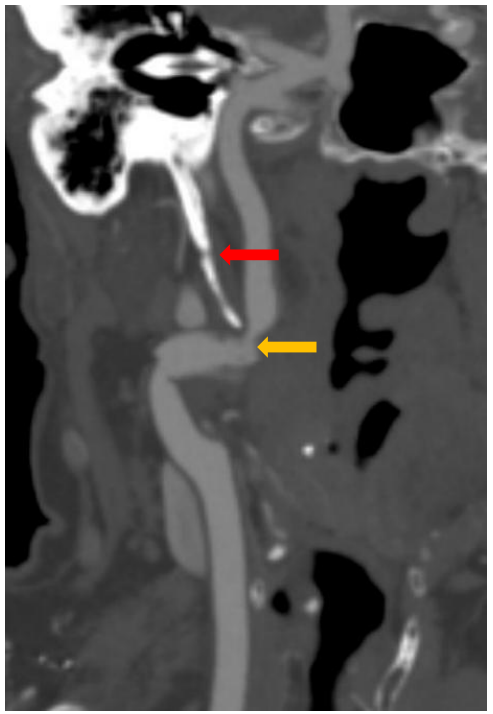
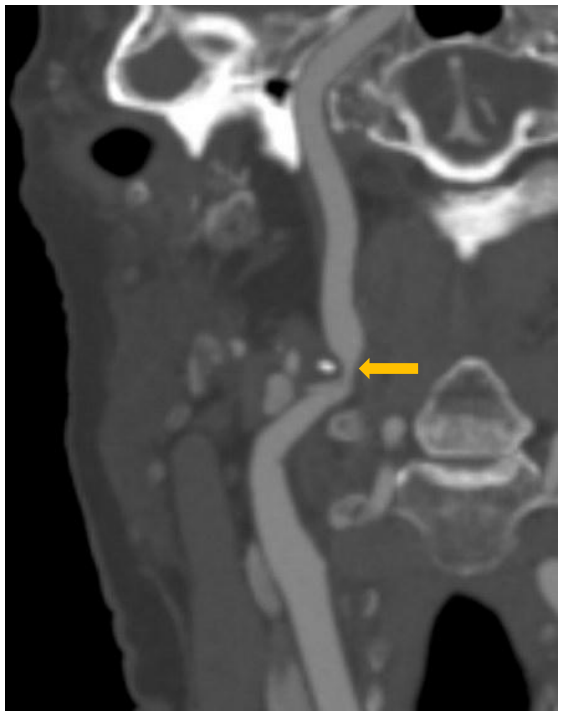
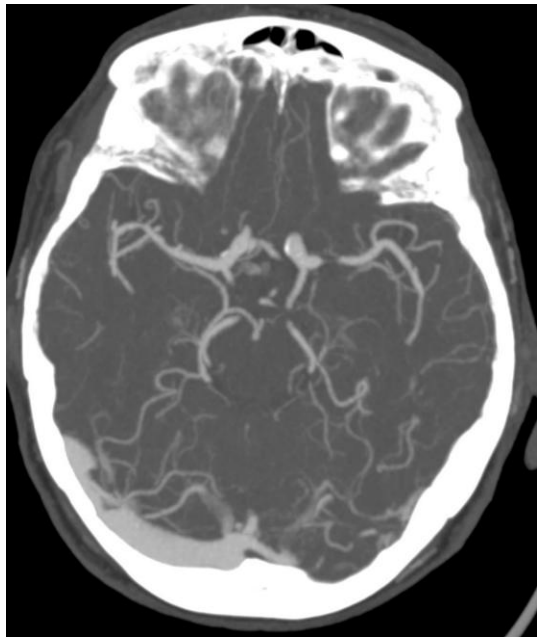
Table 2. Clinical and Imaging Outcomes (Table view)

Clinical outcome	N=61
Length of stay, median (IQR)	8 (6–13)
In-hospital stroke recurrence, n (%)	4 (6.6)
Discharge NIHSS, median (IQR)	1 (0–3)
Discharge mRS, median (IQR)	1 (0–2)
Imaging outcome	N=59
Any hemorrhagic transformation, n (%)	7 (11.9)
Thrombus resolution, n (%)	
No change	15 (25.4)
Partial	
<50%	6 (10.2)
50%–99%	22 (37.3)
Complete	16 (27.1)
Degree of stenosis	
Median (%, IQR)	61 (38–68)
≥50%, n (%)	42 (71.2)
≥70%, n (%)	13 (22)
Occluded, n (%)	5 (8.2)
Carotid revascularization	N=61
Any, n (%)	24 (39.4)
CEA	15 (24.6)
CAS	9 (14.8)

Singh RJ, et al. Intraluminal Thrombi in the Cervico-Cephalic Arteries. Stroke. 2019;50:357-364.

Stylocarotid or vascular Eagle syndrome

- 81 F presented with 6 months history of “near passing out” and 2 months history of transient neurological symptoms: two episodes of expressive aphasia- on turning her head to left and two episodes of right sided (visual field) blurred vision, also on turning her head to right

Stylocarotid or vascular Eagle syndrome

- Eagle syndrome refers to a constellation of symptoms caused by an abnormally elongated styloid process or calcified stylohyoid ligament compressing adjacent neurovascular structures.
- Anatomical studies suggest roughly 4% of the general population have an elongated styloid process (>30 mm) but most are asymptomatic. Only about 4% of individuals with an elongated styloid actually develop Eagle syndrome symptoms.
- Two variants
  - Classical: throat pain
  - Stylocarotid or vascular variant
- Stylocarotid syndrome was first described by American otolaryngologist Watt W. Eagle in 1949. The classical Eagle syndrome characterized by throat pain was described earlier in 1927.

Eagle WW. Elongated styloid processes: report of two cases. Arch Otolaryngol. 1937;25(5):584–587.
EAGLE WW. Symptomatic elongated styloid process; report of two cases of styloid process-carotid artery syndrome with operation. Arch Otolaryngol (1925). 1949;49:490-503.

Stylocarotid or vascular Eagle syndrome

- Typical demography: middle-aged adult although described at all ages, slight female predominance
- Key features
  - Incidental
  - TIA/stroke
  - Syncope
  - Neck pain or headache especially if there is dissection
  - Horner syndrome
  - Pulsatile tinnitus especially if IJC compressed

- Diagnosis relies on cross-sectional imaging; CTA is optimal imaging approach. MRA is alternative but CT better at assessing styloid process.
- Dynamic assessment can be done with doppler, dynamic CTA or DSA.

Eagle WW. Elongated styloid processes: report of two cases. Arch Otolaryngol. 1937;25(5):584–587.
EAGLE WW. Symptomatic elongated styloid process; report of two cases of styloid process-carotid artery syndrome with operation. Arch Otolaryngol (1925). 1949;49:490-503.
Raser JM, Mullen MT, Kasner SE, Cucchiara BL, Messé SR. Cervical carotid artery dissection is associated with styloid process length. Neurology. 2011;77:2061-6.

Management

- Medical
  - Observation in asymptomatic cases
  - Antiplatelet therapy if ischemic symptoms, dissection or thrombus

- Surgical: Definitive treatment of Eagle syndrome is surgical shortening (partial resection) of the elongated styloid process, known as styloidectomy. Can be either transoral or transcervical approach.

Carotidynia or TIPIC syndrome

- Carotidynia was a clinical entity described by Fay in 1927, characterized by tenderness and pain at the level of the carotid bifurcation.
- It was recognized in the first International Classification of Headache Disorders in 1988 but was subsequently removed as a distinct entity in 2004.
- In 2017, Lecler proposed TIPIC syndrome (TransIent Perivascular Inflammation of the Carotid artery syndrome) to be a clinic-radiological entity and proposed a criteria.

Fay T. Atypical neuralgia. Arch Neurol Psychiatry 1927;18:309–5
Lecler A, et al. TIPIC Syndrome: Beyond the Myth of Carotidynia, a New Distinct Unclassified Entity. AJNR Am J Neuroradiol. 2017;38:1391-1398.

Carotidynia or TIPIC syndrome

- 2.8% prevalence among patients presenting with acute neck pain.
- Typical demography: middle-aged adult, slight female predominance
- Key features
  - Acute, unilateral neck pain localized to the carotid artery region.
  - Pain is usually continuous and throbbing pain worse with head movement
  - Point tenderness over the carotid bifurcation
  - Can be preceded by viral illness

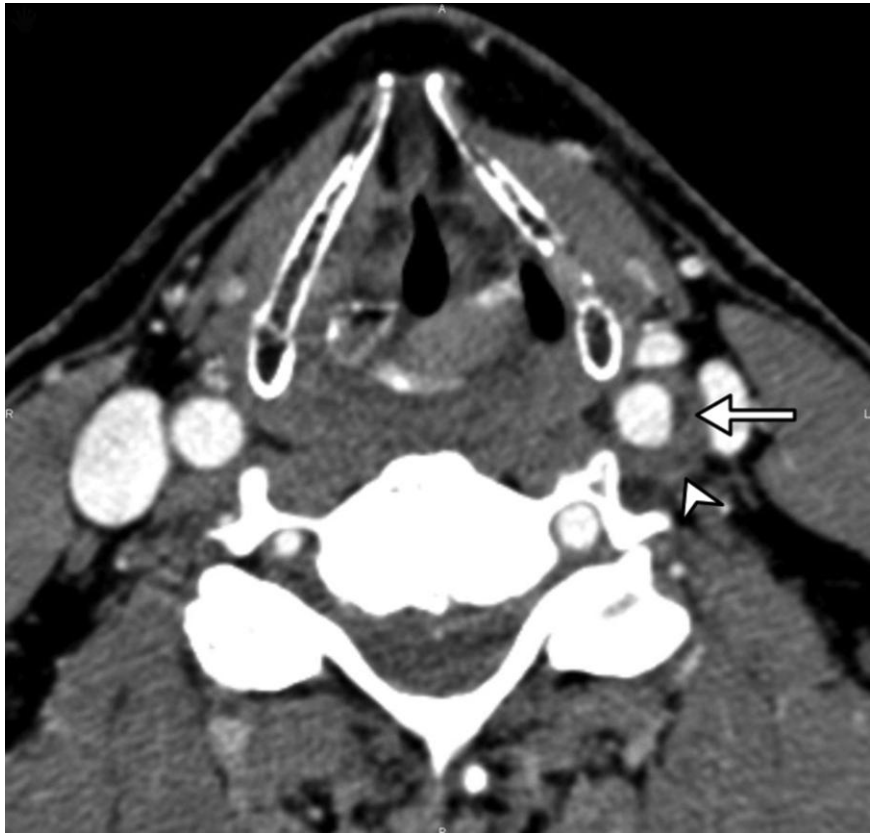
Fay T. Atypical neuralgia. Arch Neurol Psychiatry 1927;18:309–5
Lecler A, et al. TIPIC Syndrome: Beyond the Myth of Carotidynia, a New Distinct Unclassified Entity. AJNR Am J Neuroradiol. 2017;38:1391-1398.

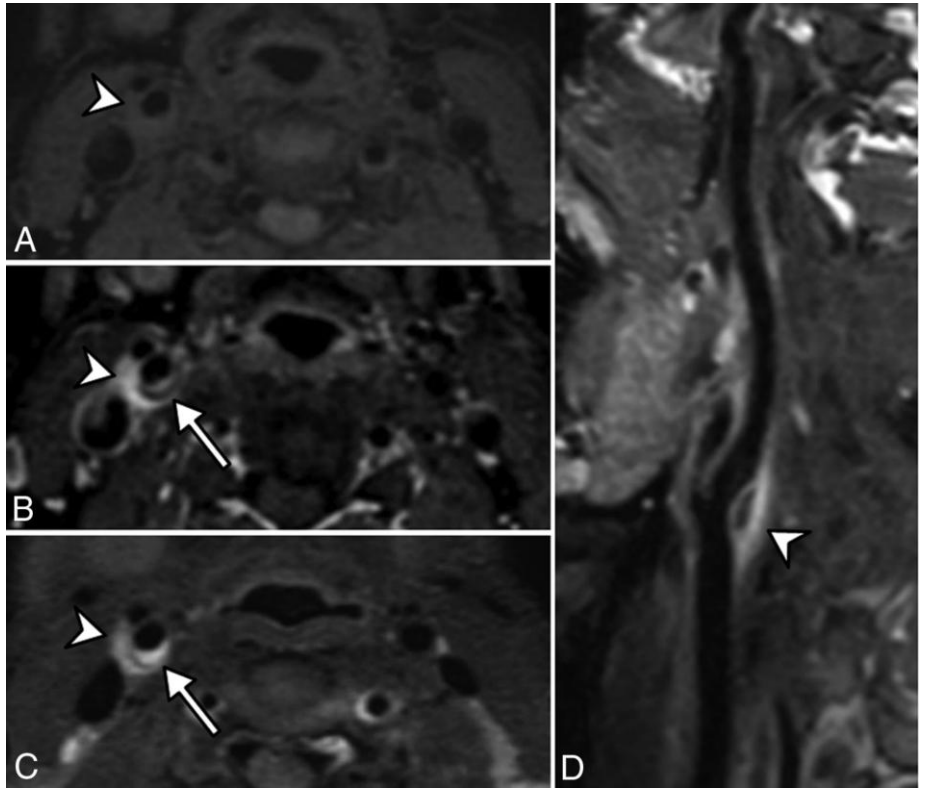
Carotidynia or TIPIC syndrome

- In 2017, Lecler proposed TIPIC syndrome (TransIent Perivascular Inflammation of the Carotid artery syndrome) to be a clinic-radiological entity and proposed a criteria as follows:
  - Presence of acute pain overlying the carotid artery, which may or may not radiate to the head
  - Eccentric perivascular infiltration (soft amorphous tissue replacing the fat surrounding the carotid artery, with a hazy aspect of the fat) on imaging
  - Exclusion of another vascular or nonvascular diagnosis with imaging
  - Improvement within 14 days either spontaneously or with anti-inflammatory treatment.
  - Additionally, a minor criterion could be the presence of a self-limited intimal soft plaque.

Fay T. Atypical neuralgia. Arch Neurol Psychiatry 1927;18:309–5
Lecler A, et al. TIPIC Syndrome: Beyond the Myth of Carotidynia, a New Distinct Unclassified Entity. AJNR Am J Neuroradiol. 2017;38:1391-1398.

Diagnosis





Lecler A, et al. TIPIC Syndrome: Beyond the Myth of Carotidynia, a New Distinct Unclassified Entity. AJNR Am J Neuroradiol. 2017;38:1391-1398.

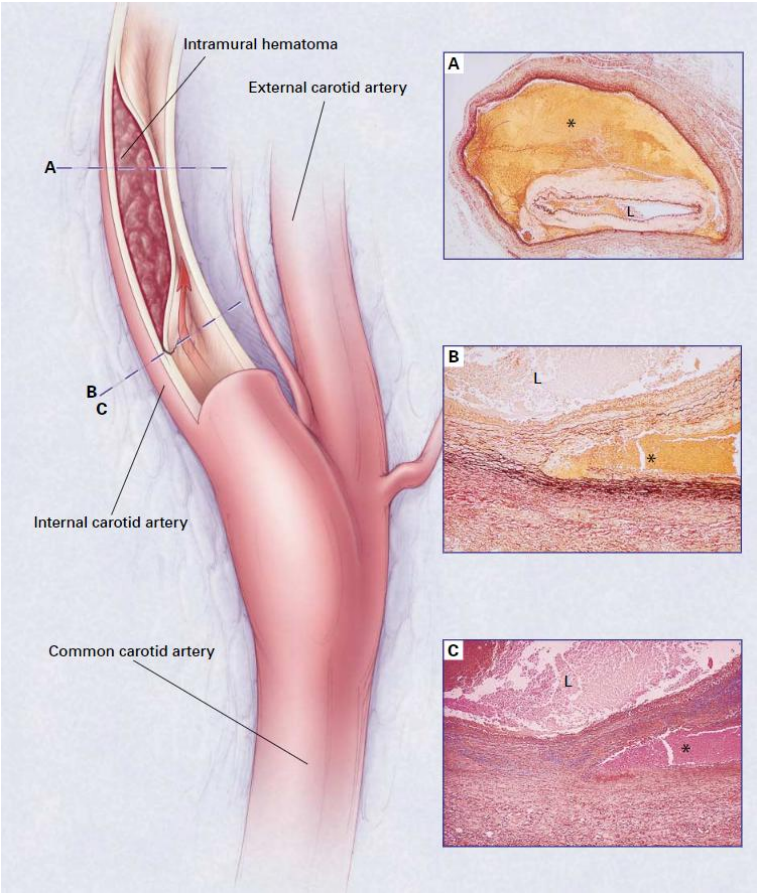
Management

- Treatment
  - There have been no randomized controlled trials for treating carotidynia/TIPIC syndrome.
  - In general, TIPIC syndrome is a benign, self-resolving condition, so treatment aims to relieve symptoms and perhaps shorten the episode. The mainstay therapies are analgesic and anti-inflammatory medications. Rest and avoiding excessive neck movement or strain can help.
  - First-line treatment typically involves NSAIDs (such as ibuprofen, naproxen, or aspirin) given on a short-term basis. NSAIDs address both pain and inflammation.
  - In more severe cases or those not responding to NSAIDs, a short course of corticosteroids can be given. Oral prednisone (e.g. 30–60 mg daily) for a week or two often produces rapid relief of pain and reduction of inflammation. A dramatic improvement within 24–48 hours of steroid therapy is typical.
- Follow-up: Follow-up imaging in few weeks might provide reassurance if the perivascular inflammation improves.
- Prognosis:
  - In the short term, about 19% of patients have a relapse within the first 6 months after the initial episode and ~29% at longer follow-up upto ~5 years.

Lecler A, et al. TIPIC Syndrome: Beyond the Myth of Carotidynia, a New Distinct Unclassified Entity. AJNR Am J Neuroradiol. 2017;38:1391-1398.
Obadia M, et al. Long-term clinical and ultrasound follow-up after transient perivascular inflammation of the carotid artery (TIPIC) syndrome: a multicenter study. Cephalalgia. 2024;44:3331024241230247.

Carotid dissections

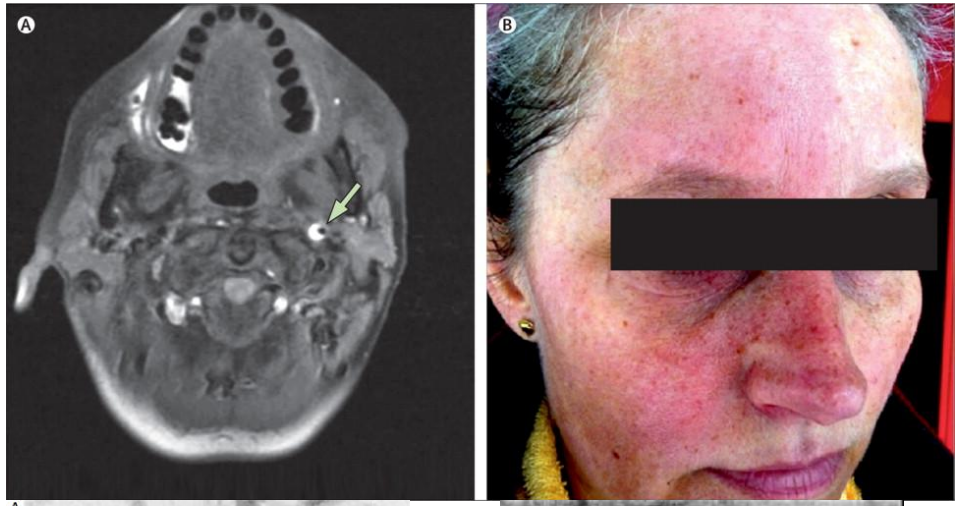
- Arterial dissection is presence of intramural hematoma causing splitting of arterial wall layers. Can result from intimal tear (inside out hypothesis or rupture of vasa vasorum (outside in hypothesis).
- Prevalence: Spontaneous carotid artery dissection ranged from 2.5 per 100,000 to 3 per 100,000.
- Cervical artery dissection although infrequent cause of ischemic stroke overall accounting for ≈1% to 2% of ischemic strokes and much more common in young adult (<50 years) causing up to 25% ischemic strokes.
- Etiology: genetic factors (connective tissue disorders- Marfan, Ehler Danlos etc.) or environmental (infections, low energy trauma/precipitating factors).
- Mechanism of stroke: embolic, hemodynamic or mixed. Can have intracranial extension and cause SAH.

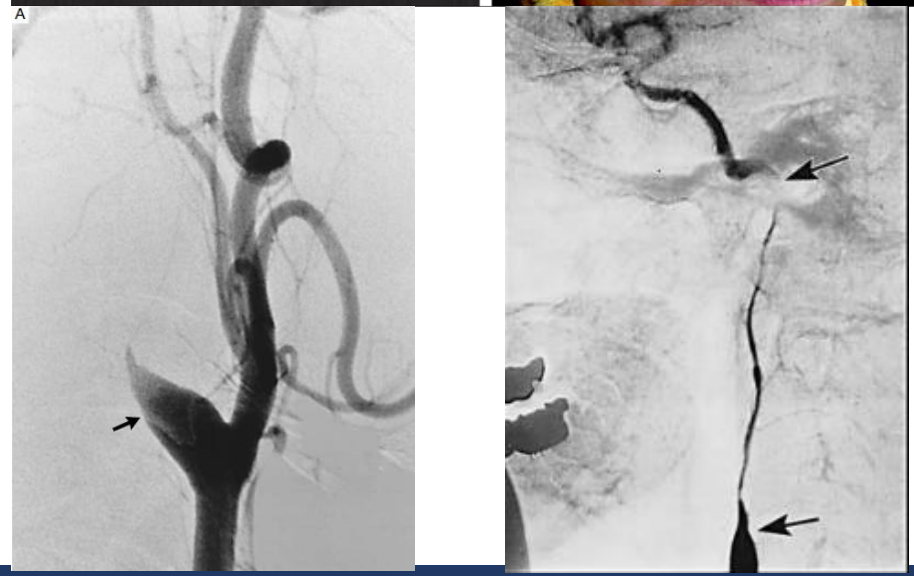


Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med. 2001;344:898-906.
Antiplatelet therapy vs anticoagulation therapy in cervical artery dissection: the Cervical Artery Dissection in Stroke Study (CADISS) Randomized Clinical Trial Final Results. JAMA Neurol. 2019; 76:657–664.

Clinical features and diagnosis

- Clinical features:
  - Local: Pain (most common, ipsilateral and generally constant), Horner syndrome (~50% or less) or lower cranial signs (~12%, especially taste impairment or hypoglossal involvement).
  - Ischemic: retinal or hemispheric deficits in 50-95%, generally within the first week or two of dissection.
  - Hemorrhagic: SAH.
- Diagnosis: CUS, CTA, MRA or DSA





Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med. 2001;344:898-906.
Biousse V, D'Anglejan-Chatillon J, Touboul PJ, Amarenco P, Bousser MG. Time course of symptoms in extracranial carotid artery dissections. A series of 80 patients. *Stroke*. 1995;26:235-239.
Morris NA, Merkler AE, Gialdini G, Kamel H. Timing of Incident Stroke Risk After Cervical Artery Dissection Presenting Without Ischemia [published correction appears in Stroke. 2018 Oct;49(10):e308]. *Stroke*. 2017;48:551-555.
Sarikaya H. Hemifacial sweating after carotid artery dissection. *Lancet*. 2011;378(9791):606. doi:10.1016/S0140-6736(11)61102-1

Management

Antiplatelet Therapy vs Anticoagulation Therapy in Cervical Artery Dissection
The Cervical Artery Dissection in Stroke Study (CADISS) Randomized Clinical Trial Final Results

Hugh S. Markus, FMedSci[1]; Christopher Levi, MD[2]; Alice King, PhD[3]; et al
Author Affiliations | Article Information
JAMA Neurol. 2019;76(6):657-664. doi:10.1001/jamaneurol.2019.0072

- Carotid dissection within 1 week
- Compared antiplatelet vs heparin/VKA (INR 2-3) for 90 days
- Primary endpoint was ipsilateral stroke or death
- Only 4 strokes (among 126) in APT and 1 in OAC (of 124).

- Cervical artery dissection within 2 weeks
- Compared ASA 300 mg vs heparin/VKA (INR 2-3) for 90 days
- Primary endpoint was a composite of clinical (ischemic stroke, major extracranial or intracranial hemorrhage, death
- Primary endpoint 23.1 % in ASA and 14.6% in VKA (p=0.54)
- Ischemic stroke: 7.7 vs 1.2%

Prognosis: about 90% stenosis resolve and 2/3 occlusion recanalize usually within 2-3 months. In CADISS trial- 3rd had normal arteries in fu, ~3rd had irregular or residual stenosis a quarter remains occluded while other developed pseudoaneurysm.
Recurrence: 2% in first months and 1%/year thereafter. Depends on the underlying predisposition.

Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med. 2001;344:898-906.

Conclusion

- Non-atherosclerosis carotid disease is important but heterogenous group causing stroke.
- It can occur asymptomatically and presents with local, ischemic (TIA/stroke) or hemorrhagic manifestations with fatal consequences.
- Diagnosis is possible by use of non-invasive vascular imaging.
- Managements is evidence based only in few conditions. Trigger prevention (when applicable) and medical therapy is primary management strategy. Endovascular or surgical options are used in select patients.
- Futures trials to risk stratify patients with non-atherosclerotic disease would help to personalize management approaches.

Evaluation

For the **Provincial Stroke Rounds Planning Committee**:

- To plan future programs
- For quality assurance and improvement

- For **You**: Reflecting on what you've learned and how you plan to apply it can help you enact change as you return to your professional duties

For **Speakers**: The responses help understand participant learning needs, teaching outcomes and opportunities for improvement.

https://forms.office.com/r/71LXRtm1b5

Please take 2 minutes to fill the evaluation form out. Thank you!

Thank you