# Vertebral artery dissection

### F1 Nuengruethai



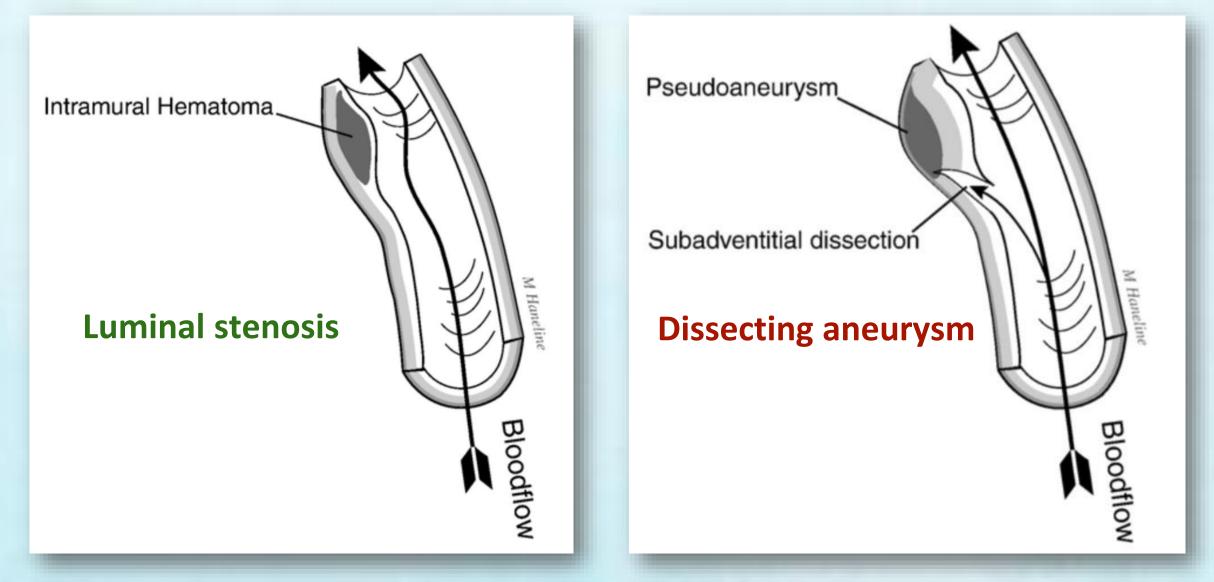


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## Pathophysiology

### **Subintimal dissection Subadventitial dissection**



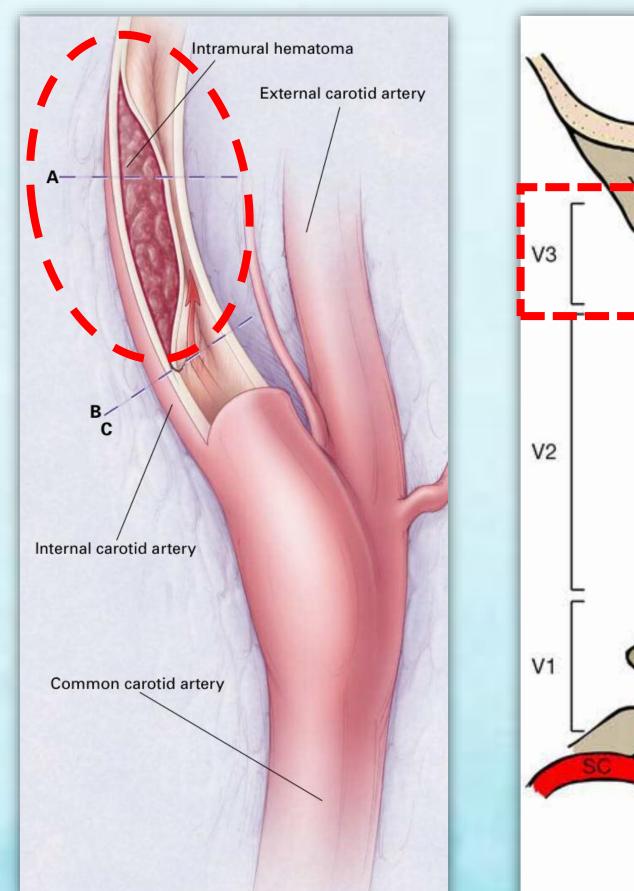


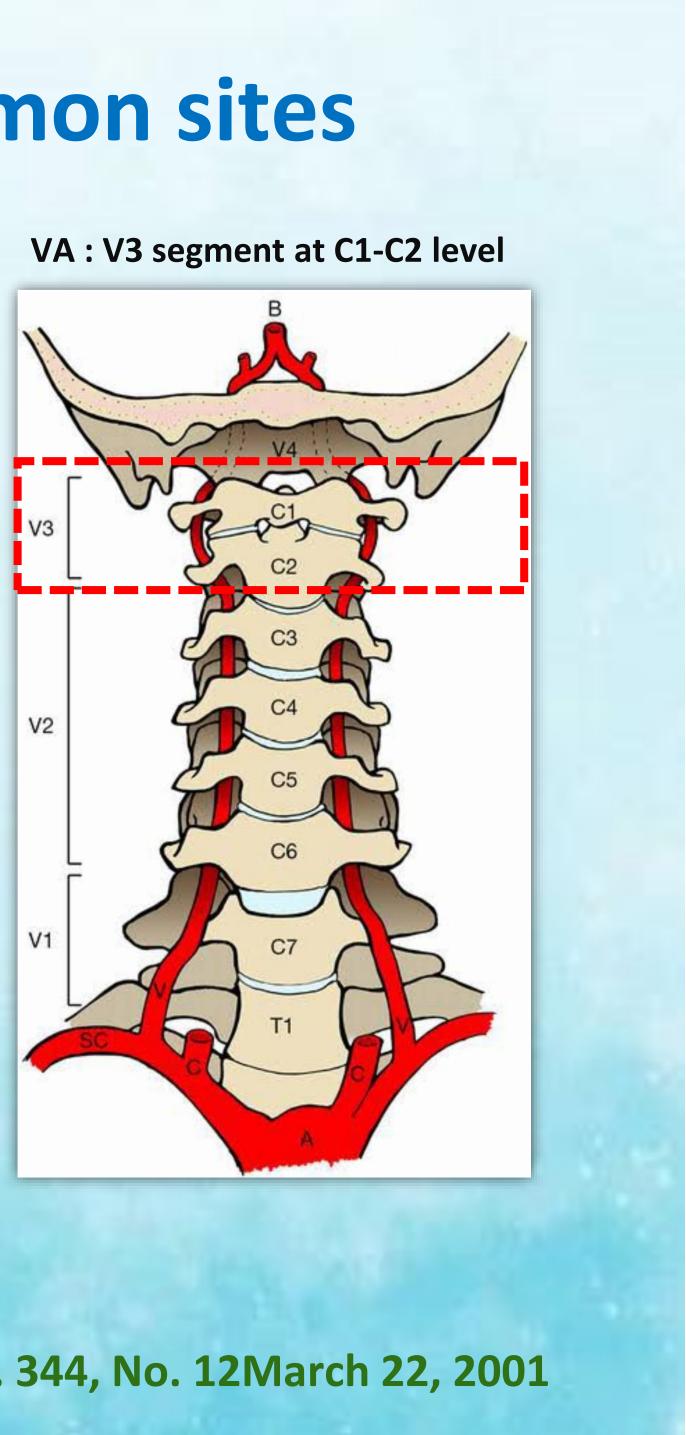
**Thromboembolism** (A to A emboli) **Hypoperfusion** (low flow)

Pain **Compression nerve** SAH

## **Common sites**

### ICA: <u>></u> 2 cms distal to carotid bifurcation





N Engl J Med, Vol. 344, No. 12March 22, 2001

# Etiology

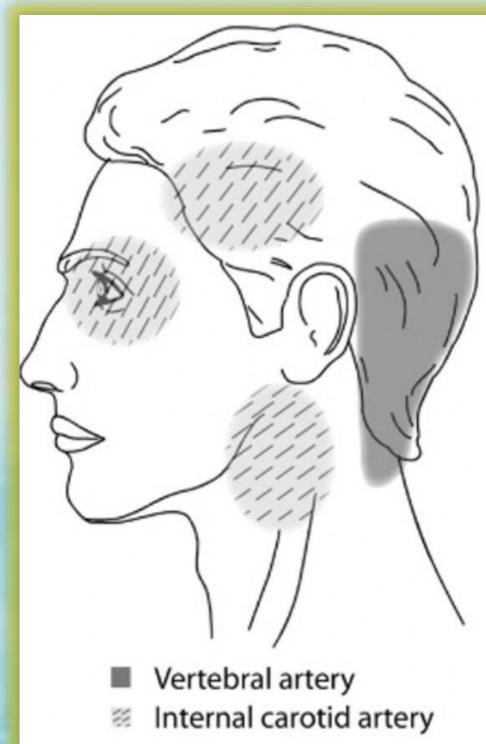
- Blunt trauma to neck
- Cervical cord injury or
  - spine fracture (20-40%)
- Minor trauma
- Coughing/vomiting
- Chiropractic procedure
- Cervical spine manipulation (1 in 20,000)
- Ehlers-Danlos syndrome
- Spontaneous dissections

# **Risk factors**

- Hypertension
- Fibromuscular dysplasia
- Female
- $\circ$  OCP
- Vasculitis
- Sports: Yoga, Judo, wrestling

# Epidemiology

- 2% of all ischemic stroke
- More common causes of stroke in young (10-25%)
- 2.6 cases per 100,000
- Carotid artery dissections
  - 3-5x VS VA dissections



## Presentation

O Headache (60-90%): CAD < 20% thunderclap headache O Neck pain: VAD • TIA/Ischemic stroke: 77% present at time diagnosis **Neurological deficit by** - Occlusion - Emboli: Anterior circulation (CAD) Posterior circulation (VAD) - Compressive aneurysm: Horner syndrome Pulsatile tinnitus CN palsy

Todd B. Britt et al. Vertebral Artery Dissection; July 21, 2020



# Presentation

### Table 3. Presentation Of Spontaneous Cervical Artery Dissection

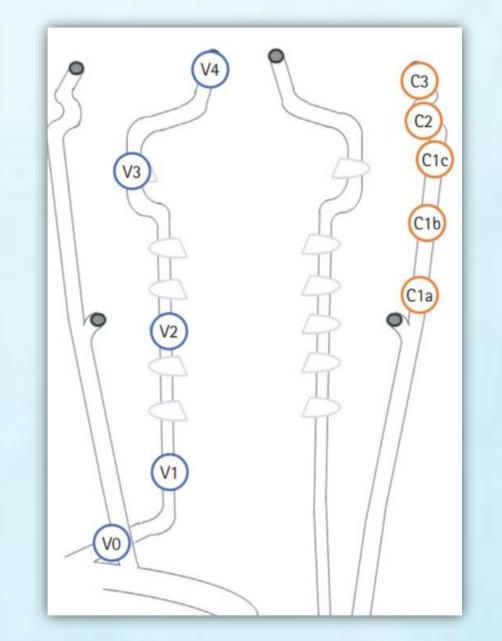
Symptoms	Location of Dissection		
	Carotid	1	
Pain distribution	<ul> <li>Headache or migraine, 36%, 68%,<sup>67</sup> 65%<sup>5,68</sup></li> <li>Occipital headache, 1%<sup>67</sup></li> <li>Frontal headache, 23%<sup>68</sup></li> <li>Facial or orbital pain, 52%<sup>67</sup></li> <li>Neck pain, 16%,<sup>5</sup> 26%<sup>67</sup></li> <li>Chest pain*</li> </ul>		
Neurological deficits, by occlusion	<ul> <li>Hemiparesis and sensory loss</li> <li>Monocular blindness</li> <li>Hemineglect</li> <li>Aphasia (if dominant hemisphere)</li> </ul>		
Neurological deficits, by emboli	<ul> <li>Anterior circulation deficits:</li> <li>Amaurosis fugax (monocular blindness), 17%<sup>5</sup></li> <li>Hemineglect</li> <li>Hemiparesis, 23%<sup>5</sup></li> <li>Aphasia</li> <li>Dysarthria</li> <li>Hemisensory loss, 7%<sup>5</sup></li> </ul>		
Neurological deficits by compressive aneurys- mal dilatation and disruption of adventitia	<ul> <li>Horner syndrome, unilateral miosis, ptosis, anhidrosis, 35%, 20%-48%<sup>68</sup></li> <li>Dysgeusia, 0.5%<sup>68</sup></li> <li>Pulsatile tinnitus, 16%<sup>68</sup></li> <li>Ocular motor palsy: CN III, IV, VI, 2.6%<sup>117</sup></li> <li>Palsy of CN V, 3.7%<sup>116</sup></li> <li>Palsy of CN VII, IX-XII, 7.5%,<sup>74</sup> 12%<sup>116</sup></li> </ul>		

### Vertebral

- · Headache or migraine
- Neck pain
- Neck pain posteriorly
- Chest pain\*
- · Locked-in syndrome
- Respiratory failure
- Hemianopsia or bilateral visual field loss

Posterior circulation deficits:

- · Hemianopsia or unilateral field deficit
- Ataxia, 20%<sup>40</sup>
- Diplopia
- Dysarthria
- Upside-down vision
- Lateral medullary syndrome (Wallenberg syndrome), 32%40: dysphagia, hemiparesis, diplopia, facial weakness, unilateral tinnitus
- Pulsatile tinnitus, 5%
- Unilateral radicular weakness (C5-C6 most common), 1%123,125,127,128



Vertebral artery	⁰⁄₀ V0-V4 (n=141)
Headache	84.4 (119)
Horner's	0.0
Cranial nerve palsy	0.0
Tinnitus	3.5 (5)

Internal carotid artery	% C1a-C3 (n=127)
Headache	74.8 (95)
Horner's	33.1 (42)
Cranial nerve palsy	10.2 (13)
Tinnitus	13.4 (17)

Journal of Stroke 2019:21(1)112-115 **Emerge Med Prac. 2012; 14(4)** 



## Neuroimaging

### • **CTA & MRA**: equal Se/Sp

**Digital subtraction** 0 angiography (DSA) (Gold standard)

**TCCD/CDUS**: 0 Se/Sp 68-95/20%



**CTA** 

### **Flame-shape occlusion**

# Diagnosis

## Angiographic finding (Se/Sp 64-100%/51-98%)



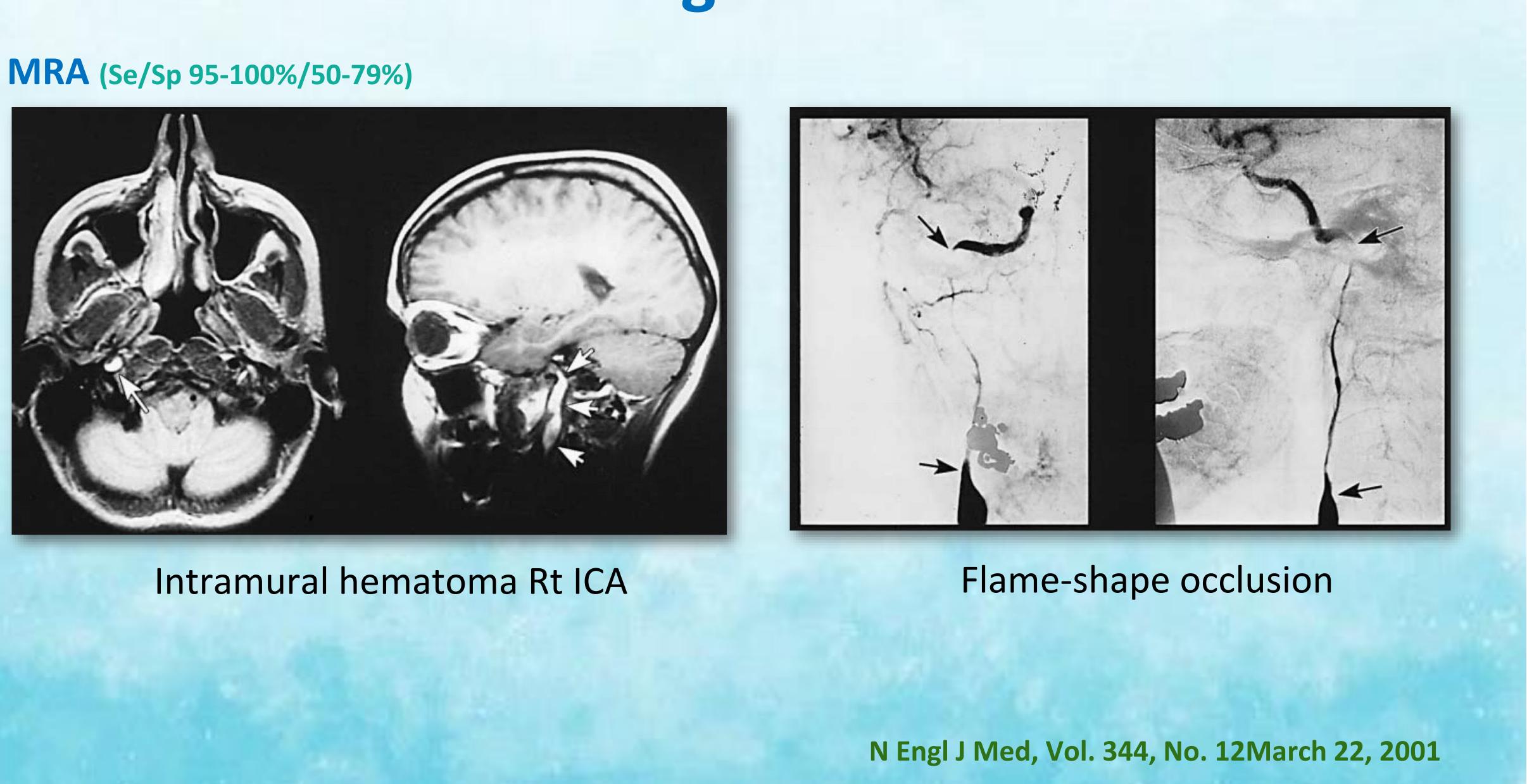
### **Pseudoaneurysm**

### **Intimal flap**

N Engl J Med, Vol. 344, No. 12March 22, 2001

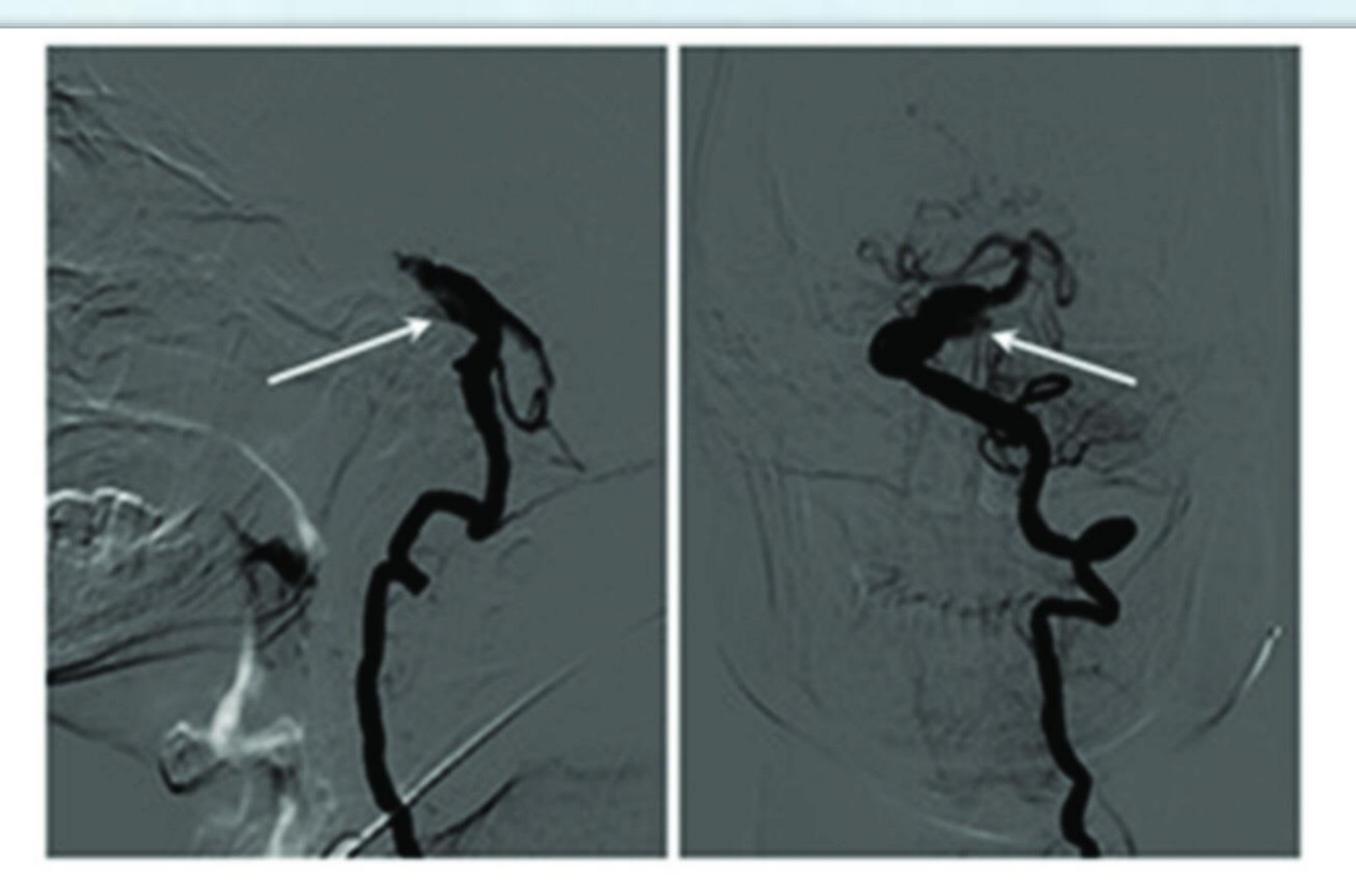


# Diagnosis



# Diagnosis

## **Digital subtraction angiography (DSA):** Gold standard



| Digital subtraction angiography (DSA) examination identified a basilar artery dissection indicating the delayed image of distal basilar artery and the stratification and retention of contrast agent (arrow).



## Treatment

## **AHA/ASA** Guideline

## **Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association**

## Acute treatment

Extracranial cervical dissections	IV alteplase in AIS known or suspect within 4.5 h and probably recomme
Intracranial arterial dissection	IV alteplase usefulness and hemorr remain unknown, uncertain and not



Aortic arch dissection

IV alteplase in AIS known or suspected to be associated with a ortic arch dissection is potentially harmful and should not be administered. † (COR III: Harm; LOE C-EO)§ (Recommendation wording modified to match COR III stratifications.)

ected to be associated with extracranial cervical arterial dissection is reasonably safe ended. † (COR IIa; LOE C-LD)§

rhagic risk in AIS known or suspected to be associated with intracranial arterial dissection ot well established.† (*COR IIb; LOE C-LD*)§



# Treatment

### AHA/ASA Guideline

Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke

A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association

## Secondary prevention

6.6.3. Arterial Dissection	COR	LOE
<ol> <li>For patients with AIS and extracranial carotid or vertebral arterial dissection, treatment with either antiplatelet or anticoagulant therapy for 3 to 6 months is reasonable.</li> </ol>		B-NR
<ol> <li>For patients with AIS and extracranial carotid or extracranial vertebral arterial dissection who have definite recurrent cerebral ischemic events despite medical therapy, the value of extracranial EVT (stenting) is not well established.</li> </ol>	llb	C-LD

### Antiplatelet treatment compared with anticoagulati treatment for cervical artery dissection (CADISS): a randomised trial

The CADISS trial investigators\*

	Intention-to-tr	ntention-to-treat population		Per-protocol population			
	Antiplatelet group (n=126)	Anticoagulant group (n=124)	OR (95% CI)*	p value	Antiplatelet group (n=101)	Anticoagulant group (n=96)	OR (95% CI)*
Ipsilateral stroke or death	3 (2%)	1 (1%)	0.335 (0.006–4.233)	0.63	3 (3%)	1 (1%)	0.346 (0.006–4.390)
Secondary endpoints							
Any stroke or death	3 (2%)	1 (1%)	0·335 (0·006–4·233)	0.63	3 (3%)	1 (1%)	0·346 (0·006–4·390)
Any stroke, death, or major bleed	3 (3%)	2 (2%)	0.673 (0.055-5.983)	1.00	3 (3%)	2 (2%)	0.696 (0.057-6.220)
Any stroke	3 (2%)	1 (1%)	0.335 (0.006–4.233)	0.63	3 (3%)	1 (1%)	0·346 (0·006–4·390)
Ipsilateral stroke, TIA, or death	4 (3%)	5 (4%)	1.280 (0.268–6.614)	0.98	4 (4%)	4 (4%)	1.054 (0.190–5.835)
Any stroke or TIA	5 (4%)	5 (4%)	1.017 (0.228–4.540)	1.00	5 (5%)	4 (4%)	0.836 (0.161–4.015)
Major bleeding	0 (0%)	1 (1%)			0 (0%)	1 (1%)	
Death	0 (0%)	0 (0%)			0 (0%)	0 (0%)	

Data for presence of residual stenosis (>50%) at 3 months have not yet been analysed. OR=odds ratio. TIA=transient ischaemic attack. \*Tested with exact logistic regression.

Table 2: Outcomes within 3 months

Patient	250 Pts (Extracranial carotid (118) and VAD (132) with Onset of symptoms within past 7 days (39 centers in UK
Intervention	Antiplatelet (126)
Control	Anticoagulant (124)
Outcome	No difference efficacy to prevent stroke & death But low stroke rate and events

ion	
p value 0.66 1.00 0.66 1.00 1.00 1.00  	
K)	



AMERICAN COLLEGE of CARDIOLOGY FOUNDATION American Heart Stroke Association Association

### **ACCF/AHA Pocket Guideline**

Based on the 2011 ASA/ACCF/AHA/AANN/AANS/ACR/ CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS

Guideline on the Management of Patients With Extracranial Carotid and Vertebral Artery Disease

Developed in Collaboration With the American Academy of Neurology and Society of Cardiovascular Computed Tomography

January 2011

### 18. Management of Patients With Cervical Artery Dissection

Class I	1. Contrast-enhanced CTA, MRA and catheter-based
	angiography are useful for diagnosis of cervical
	artery dissection. (Level of Evidence: C)

Class IIa 1. Antithrombotic treatment with either an anticoagulant (heparin, low molecular weight heparin or warfarin\*) or a platelet inhibitor (aspirin, clopidogrel or the combination of extended-release dipyridamole plus aspirin\*) for at least 3 to 6 months is reasonable for patients with extracranial carotid or vertebral arterial dissection associated with ischemic stroke or TIA. (Level of Evidence B)

\* Drugs are not listed in order of preference.

Class IIb 1. CAS might be considered when ischemic neurological symptoms have not responded to antithrombotic therapy after acute carotid dissection. (Level of Evidence: C)

**2.** The safety and effectiveness of therapy with a-adrenergic antagonist, angiotensin inhibitor, or nondihydropyridine calcium channel antagonist to lower BP to normal and reduce arterial wall stress are not well established. (Level of Evidence: C)





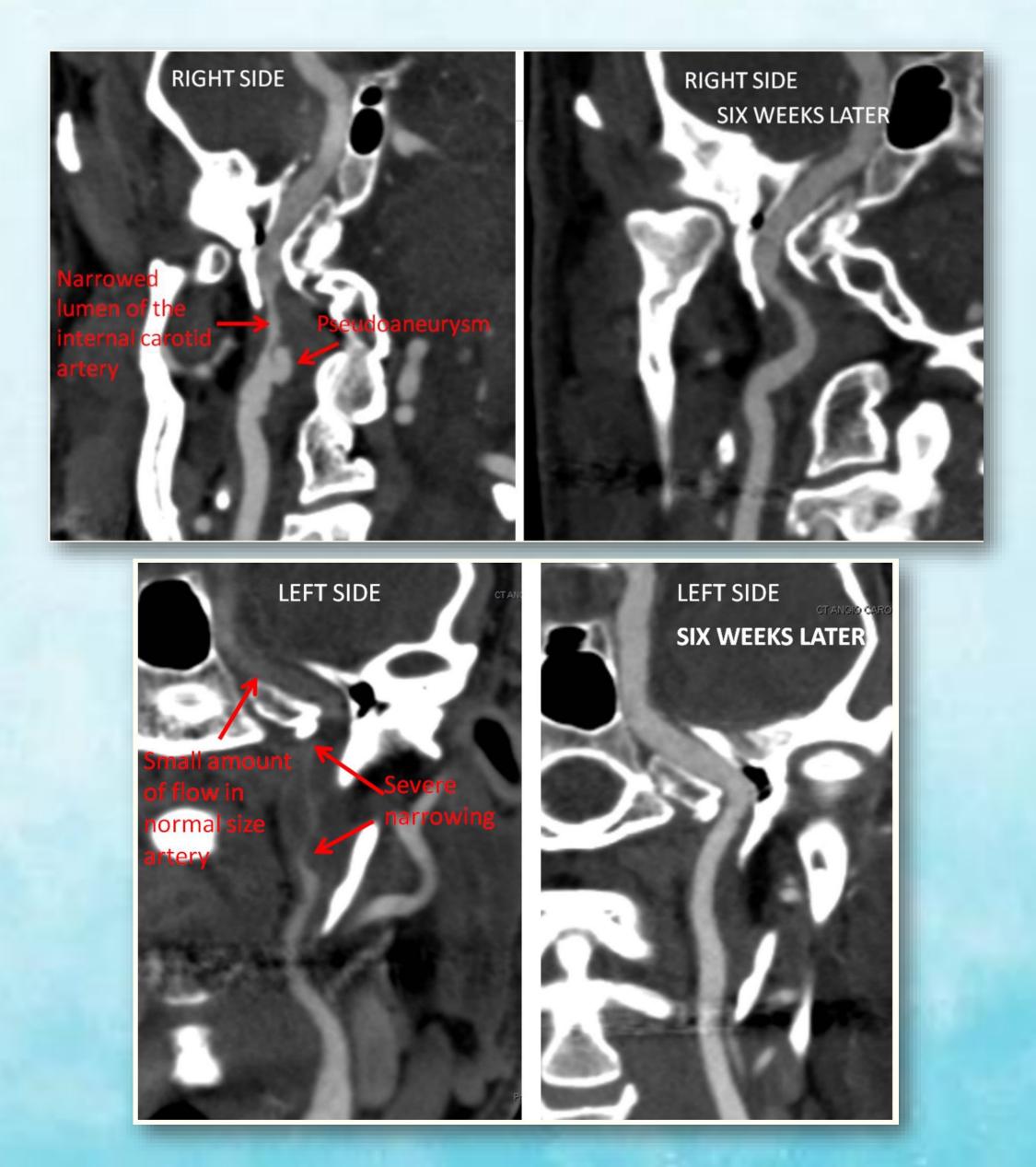
## **Vessel healing**

60-70 % **Complete recanalization of** vertebral artery 6-9 mo

## Recurrence

- $\circ$  0.3-1% annually
- o 2% at 3 mo
- Asians higher recurrent rate
- Not related aneurysm formation

# Prognosis





# Take Home Message

- Common sites: ICA > 2 cms distal to carotid bifurcation, VA at V3 segment (C1-2)
- $\circ$  More common cause of stroke in the young 10-25%, CAD (3-5x) > VA
- Presentation: Headache more common, neck pain, TIA and AIS (occlusion, emboli, compressive aneurysm)
- CTA, MRA equal Se/Sp, DAS is gold standard (but rare to used)
- Extracranial cervical dissection rtPA in 4.5 hrs AIS is safe but unknown

harm in a ortic arch dissection

 AIS with extracranial carotid or VA dissection treatment with antiplatelet or anticoagulant for 3-6 mo

usefulness and hemorrhagic risk in AIS with intracranial arterial dissection and





# Thank you

