CERVICAL ARTERY DISSECTION (CAD)

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OUTLINE

- Introduction
- Pathophysiology
- Clinical Symptoms: ICA, VA Dissection
- Neuroimage
- Treatment & Prognosis

EPIDEMIOLOGY

- Community-based studies in USA & France: annual incidence 2.5-3.0 per 100,000
- Hospital-based studies: 1.0-1.5 per 100,000
- Accounted 2 percent of all ischemic strokes
- One of the most common cause of young stroke (about 15-20%)
- Though common in young adults (5^{th} decade), dissection could also occur in the eldery and children
- Women are on average 5 years younger than men at onset age

INTRODUCTION

- Neck pain and headache preceding or accompanying the focal sign, some are asymptomatic
- Cervical Artery Dissection (CAD):

  ICA and VA, extracranial portions
- Intracranial dissections are uncommon but more serious: mostly V4 and MCA→ SAH, easily fatal
- Aortic arch dissections:

  shock and global cerebral ischemia, loss consciousness; some may with focal sign (contraindication for tPA)

HIS 2ND EDITION

6.5 Carotid or Vertebral Pain

- 6.5.1 Headache or facial or neck pain attributed to arterial dissection
- 6.5.2 Post-endartectomy headache
- 6.5.3 Carotid angioplasty headache
- 6.5.4 Headache attributed to intracranial endovascular procedures
- 6.5.5 Angiography headache

6.5.1 HEADACHE OR FACIAL OR NECK PAIN ATTRIBUTED TO ARTERIAL DISSECTION

A. Any new headache, facial or neck pain of acute onset, with or without other neurological symptoms or signs and fulfilling criteria C and D
B. Dissection demonstrated by appropriate vascular and/or neuroimage investigations
C. Pain develops in close temporal relation to and on the same side as the dissection
D. Pain resolves within 1 month

HEADACHE IN ACUTE STROKE

- 25% had peri-stroke headache (within 3 days)
- Duration: hours ~ weeks or months
- More frequent in posterior circulation stroke
- No correlation between infarct size and headache severity
- Location:

  # ICA: forehead or eyes

  # MCA: temporal

  # PCA: external corner of eyes and eyebrow

  # BA: vertex

  # VA: neck, mastoid, occiput

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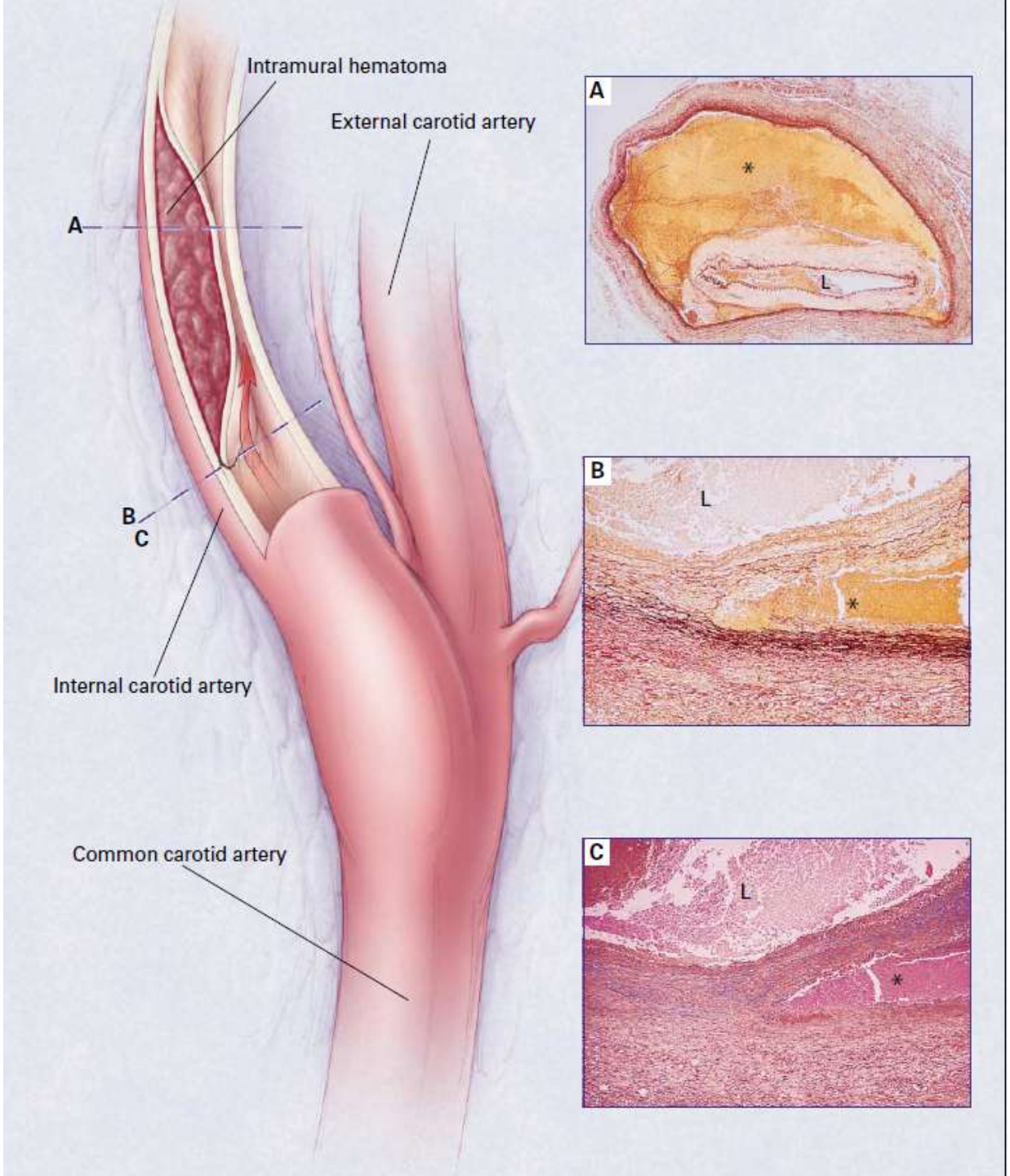

Intramural hematoma
External carotid artery
A
B
C
Internal carotid artery
Common carotid artery
A
*
L
B
L
*
C
L
*

Mechanism

- Blood splits the arterial wall:

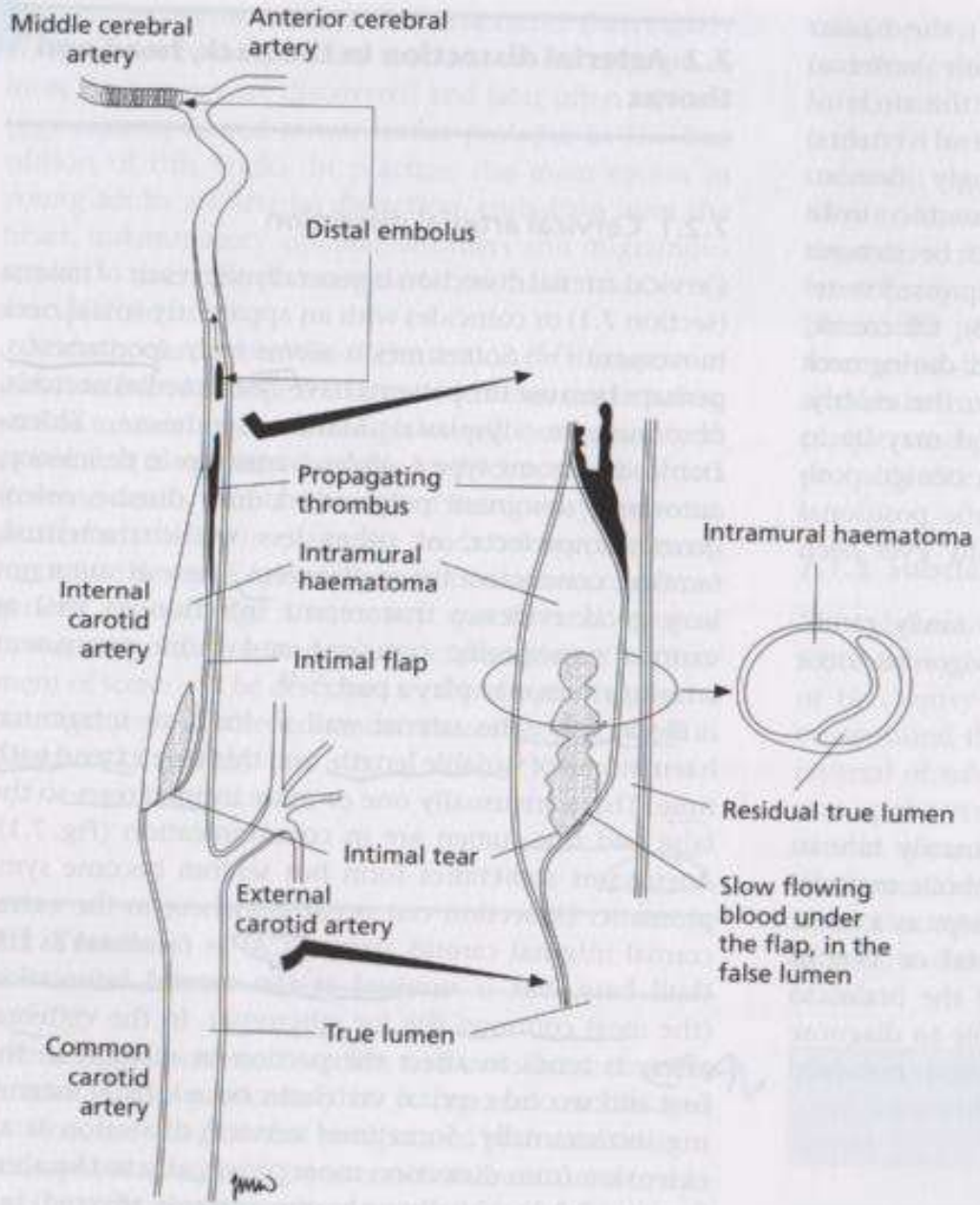
  intima~ media: stenosis

  media~ adventia: pseudoaneurysm

  → intramural hematoma

  variable length, extend with time
- One or more intimal tears

  → false or true lumen
- Form aneurysm but less became symptomatic
- Distal hypoperfusion
- Distal thrombus formation→ embolic stroke


Middle cerebral artery
Anterior cerebral artery
Distal embolus
Propagating thrombus
Intramural haematoma
Internal carotid artery
Intramural haematoma
Intimal flap
Residual true lumen
Intimal tear
External carotid artery
Slow flowing blood under the flap, in the false lumen
True lumen
Common carotid artery

PATHOGENESIS OF CAD

- Major Trauma
- Minor Trauma or Spontaneous
- Genetic (Connective tissue disease)

  Marfarn syndrome, Fibromuscular dysplasia, Ehlers-Danlos type III-IV, AD polycytoc kidney disease, alpha-I antitrypsin deficiency, others

Fibromuscluar dysplasia

bil. ICA involved (86%), media
15% CAD

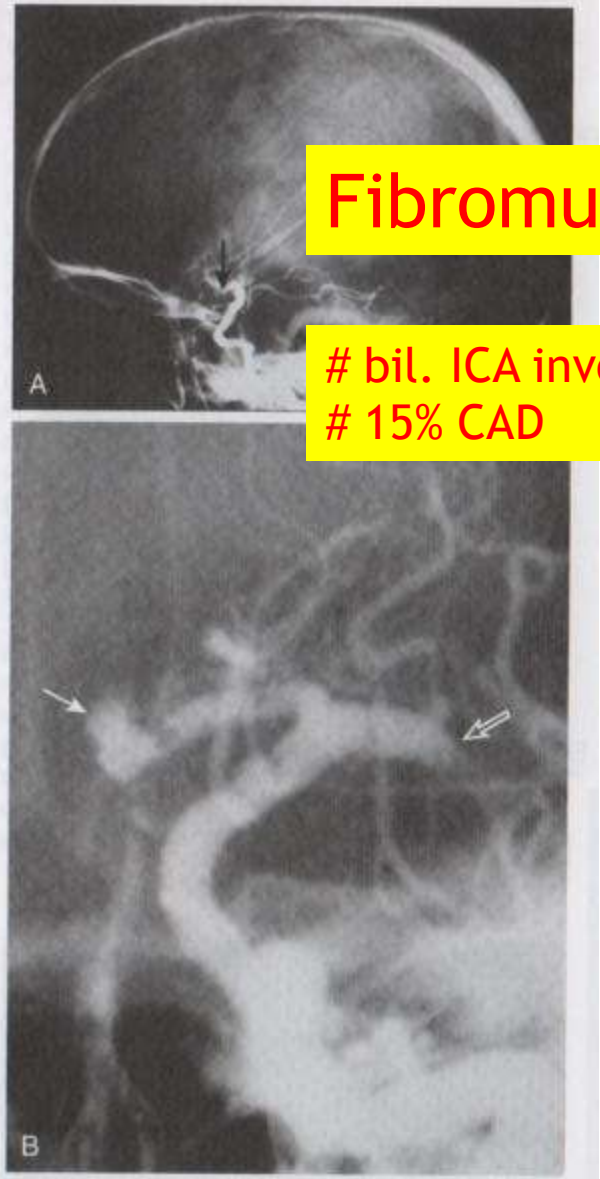


Figure 11-6. Intracranial ICA dissection in a child. **A,** Carotid angiogram, lateral view. The *black arrow* points to the dissection within the intracranial ICA. **B,** A magnified close-up view shows the dissection. The *closed white arrow* on the left shows that the MCA is occluded and the *open arrow* on the right of the picture points to an occluded anterior cerebral artery.

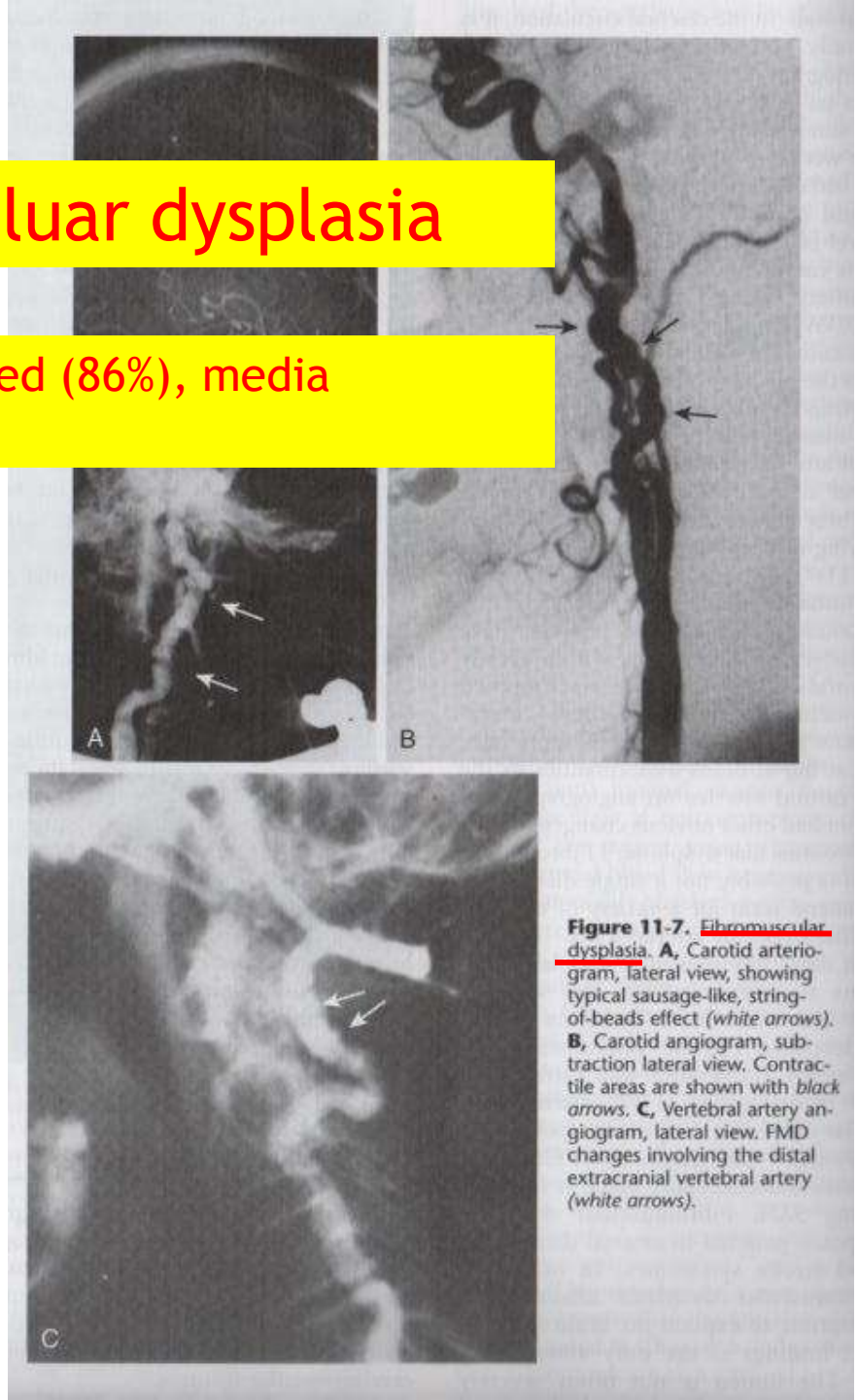


Figure 11-7. Fibromuscular dysplasia. **A,** Carotid arteriogram, lateral view, showing typical sausage-like, string-of-beads effect *(white arrows)*. **B,** Carotid angiogram, subtraction lateral view. Contractile areas are shown with *black arrows*. **C,** Vertebral artery angiogram, lateral view. FMD changes involving the distal extracranial vertebral artery *(white arrows)*.

PATHOGENESIS OF SPONTANEOUS CAD

- Chiropractice:

  previous soreness and numbness
- Sudden or prolonged hyperextesion or torsion of neck:

  yoga, painting a ceiling, coughing/sneezing, vomiting
- Certain sports
- URI: season (fall)
- Migraine
- Atherosclerosis: not severe

  HTN, smoking: no relationship to CAD


中国文摘网 www.wenzhai.org

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- **Clinical Symptoms**
- Neuroimage
- Treatment & Prognosis

HEADACHE/PAIN IN CAD

- Nuchal and/or Headache → focal sign
- Pain is most impressive and initial feature
- Severity: variable
- Radiated pattern

- Dissection of ICA: more frequent
- Dissection of VA

Dissection of ICA

- Triad:

  Pain (neck, facial, or head)

  partial Horner's syndrome

  delayed cerebral or retinal ischemia
- <1/3 compatible with all of three
- Any two symptoms→ strongly suggest dissection

Local Manifestations- Pain

- Neck pain in 25% p’ts,
  upper anterolateral cervical region
- Neck pain as Isolated symptoms: 10%
- Unilateral facial or orbital pain: 50%

Local Manifestations- Pain

- Characteristics unilateral headache develops in 2/3 p’ts
- Frontotemporal region, occasionally occipital region or hemicranium
- Usually gradual onset, but it may be an thunderclap pattern (mimics SAH)
- Usually constant steady aching, may also be throbbing or sharp pain
- Sometimes the pain is distant from the site of dissection

Local Manifestations- Oculosympathetic palsy

- <1/2 patients, partial painful Horner's syndrome
- Facial anhidrosis is not present:

  facial sweat glands are innervated by the sympathetic plexus surrounding the ECA
- Mimic cluster headache
- 10% as isolated sign

Local Manifestations- Cranial nerve palsies

- 12%, especially lower cranial nerves
- Hypoglossal nerve: most common
- Dysgeusia: 10%
- Pulsatile painful tinnitus: 25%, objective

Cranial nerve palsy in spontaneous dissection of the extracranial internal carotid artery

Article abstract—Cranial nerve palsy was present in 23 of 190 consecutive adult patients (12%) with spontaneous dissection of the extracranial internal carotid artery. Ten patients (5.2%) had a syndrome of lower cranial nerve palsies (with invariable involvement of cranial nerve XII with or without additional involvement of cranial nerves XI, X, and IX), seven (3.7%) had palsy of cranial nerve V, and five (2.6%) had a syndrome of ocular motor palsies. Palsy of cranial nerve VIII and ischemic optic neuropathy occurred in one patient each. Three patients had dysgeusia without other cranial nerve involvement, presumably due to involvement of the chorda tympani nerve. Headache or face pain (often unilateral) was present in 83% of patients. Other associated manifestations were cerebral ischemic symptoms, bruits, or oculosympathetic palsy. In one patient, cranial nerve palsy was the only manifestation of internal carotid artery dissection, and in another patient, the disease presented only as a palsy of cranial nerve XII and oculosympathetic palsy. In six patients, a syndrome of hemicrania and ipsilateral cranial nerve palsy was the sole manifestation of internal carotid artery dissection. Cranial nerve palsy is not rare in internal carotid artery dissection. Compression or stretching of the nerve by the expanded artery may explain some but not all of the palsies. An alternative mechanism is likely interruption of the nutrient vessels supplying the nerve.

NEUROLOGY 1996;46:356–359

Ischemic Event

- Cerebral or retinal ischemic symptoms: 50-95%, decreased over years
- TIA or transient monocular blindness precedes
- Multiple acute embolism-like brain infarction (cortical and watershed areas)
- Only 1/5 without warning signs
- Permanent blindness: rare

Dissection of VA

- Posterior neck or head pain, following ischemia in posterior circulation
- More easily misdiagnosed as musculoskeletal problem, mixed-up after chiropractice

Local Manifestations- Pain

- Posterior neck pain: ½
- Occipital Headache: 2/3
- Rarely, involves frontal region or hemicranium
- Neck and occipital can be bilateral pain
- Throbbing, steady, sharp
- Neck pain→ ischemic stroke: 2 weeks
  Headache→ ischemia stroke: 15 hours
- Cervical root involvement, usually at C5-6 level

Ischemic Event

- > 90%
- Wallenberg's syndrome, thalamus, cerebral or cerebellar infarct
- Isolated stroke without pain: uncommon but increasing recognized
- TIA less precedes

Headache and neck pain in spontaneous internal carotid and vertebral artery dissections

Total: 161,

ICAD:135 VAD: 26

NEUROLOGY 1995;45:1517-1522

	ICAD patients		VAD patients	
	No.	%	No.	%
Predissection headache history				
History of migraine	24/135	18	6/26	23
History of muscle contraction headache	69/135	51	11/26	42
Headache of dissection				
Prevalence	92/135	68	18/26	69
Onset				
Preceding other manifestations*	43/92	47	6/18	33
With other manifestations	40/92	43	11/18	61
After other manifestations†	9/92	10	1/18	6
Mode of onset				
Gradual	78/92	85	13/18	72
Abrupt	13/92	14	4/18	22
Gradual/abrupt	1/92	1	1/18	6
Location				
Frontal/frontotemporal	56/92	61	1/18	5.5
Generalized	7/92	8	1/18	5.5
Hemicranial	21/92	23	1/18	5.5
Occipital	0/92	0	12/18	67
Parieto-occipital	8/92	9	3/18	17
Ipsilateral	84/92	92	12/18	67
Character				
Constant	67/92	73	10/18	56
Pulsating	23/92	25	8/18	44
Uniqueness‡	65/105	62	9/18	50

	ICAD patients		VAD patients	
	No.	%	No.	%
Associated pain				
Facial or orbital pain	71/135	53	0/26	0
Neck pain				
Frequency	35/135	26	12/26	46
Onset				
Preceding other manifestations	9/35	26	5/12	42
With other manifestations	25/35	71	7/12	58
After other manifestations	1/35	3	0/12	0
Location				
Anterior lateral	30/35	86	0/12	0
Anterior midline	4/35	11	0/12	0
Posterior bilateral	0/35	0	4/12	33
Posterior lateral	1/35	3	8/12	67

	ICAD patients		VAD patients	
	No.	%	No.	%
Postdissection headache				
Migraine				
Decreased or resolved	6/23	26	2/6	33
Developed	3/111	3	0/20	0
Muscle contraction headache				
Decreased or resolved	10/69	14	0/11	0
Developed	5/66	8	0/11	0
Chronic daily headache developed	4/135	3	0/26	0
Chronic dissection headache developed	4/135	3	0/26	0

Table 2. Clinical findings in 135 patients with dissection of the internal carotid artery

Finding	Patients	
	No.	%
Focal cerebral ischemia (transient ischemic attack or stroke)	66	49
Alone	11	8
With headache	57	42
With neck pain	4	3
With oculosympathetic paresis	18	13
With pulsatile tinnitus	15	11
Oculosympathetic paresis	56	41
Alone	1	1
With headache	52	39
With focal cerebral ischemia	25	19
With transient monocular blindness	6	4
With tinnitus	16	12
Cranial nerve (CN) palsy	22	16
Lower (CN V and CN VII to CN XII)	14	10
Ocular motor (CN III, IV, VI)	5	4
Dysgeusia with other findings	9	7
Dysgeusia alone	3	2
Transient monocular blindness	34	25
Alone	2	1
With headache	19	14
With focal cerebral ischemia	22	16
With tinnitus	6	4

DD OF HEADACHE

- Migraine
- Cluster Headache
- Cervicogenic headache
- SAH

Tobin J, Flitman S. Cluster-like headaches associated with internal carotid artery dissection responsive to verapamil. *Headache* 2008; 48: 461–66.

MIGRAINE & CAD

- ¼ CAD p’ts had migraine history, usually reported unlike previous headache easily
- Pain in CAD: migraine-like with or without aura
- Cases Report: Complicated migraine→ CAD
- ICA dissection:

  Amarousis fugax DD aura

  Horner’s sign DD cluster headache

  (but some scintillation and bright sparkles resembling migraine aura)

IHS criteria for Migraine

- Episodic headache lasting 4-72 hours, attack >5 times, with:

Any 2 of:
- **Unilateral**
- **Throbbing**
- **Moderate to severe**
- **Worsened by Movement**

Any 1 of:
- **Nausea or vomiting**
- **Photophobia & phonophobia**

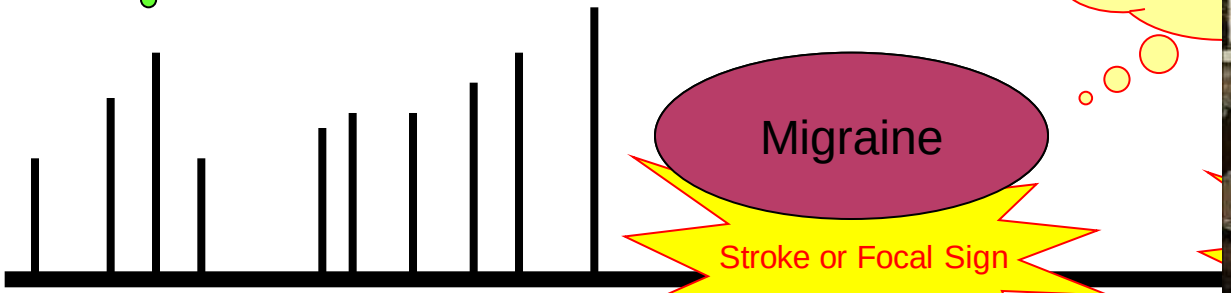
- R/O other organic brain lesion

ICA dissection:

- Young onset
- Orbital and
- Dizziness/sick wit
- TMB→ aura

為什麼頭痛變成中風沒先診斷出來!!

Migraine



Migraine

Stroke or Focal Sign

Neck pain

Migraineurs also had muscle tension pain
78% described neck pain

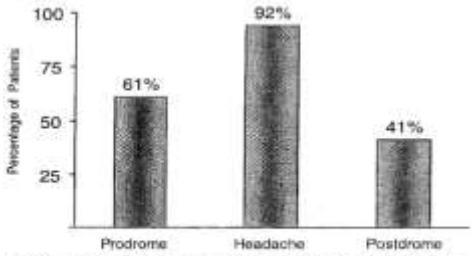


Figure 1. Percentage of patients who met IHS criteria for migraine reporting neck pain during the prodrome, headache phase, or postdrome of their migraine attacks.

THUNDERCLAP HEADACHE -CASE REPORT

44y/oF, night snack

- Sudden onset right posterior neck severe pain accompanied general dyscomfort, whirling sensation, nausea in this early morning (2-3AM)
- Almost unable to sit up and walk
- Pain radiated to ipsilateral parietoocipital region, quite excruciating

- Never had vertigo/ headache before
- No trauma, fever, URI, insomnia, tinnitus, double vision, dysarthria, altered mental state
- No obvious focal sign, Fundus:OK, FNF:OK
- Neck: unable to touch, Kernig sign(-)
- Brain CT: normal
- Mx:

cephadol 1# bid, sibelium 1# hs, deanxit 1# qd,

APAP 1# bid, motilium 1# bid, voren gel, keto iv prn

DAY 2

- Could sit, headache improved
- Right neck extremely soreness without tenderness, mild dizziness remains
- Obviously deviated to right while walking

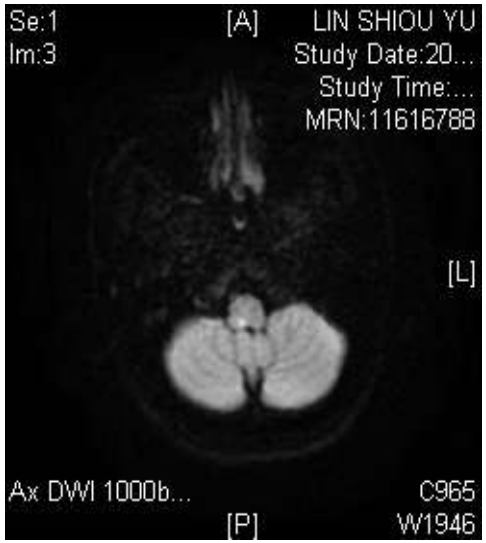
  and sitting

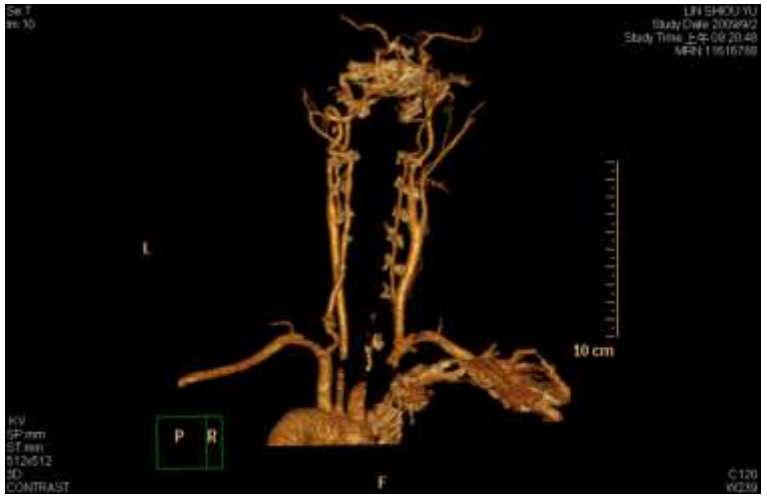
  no nystagmus, diplopia, Horner's sign

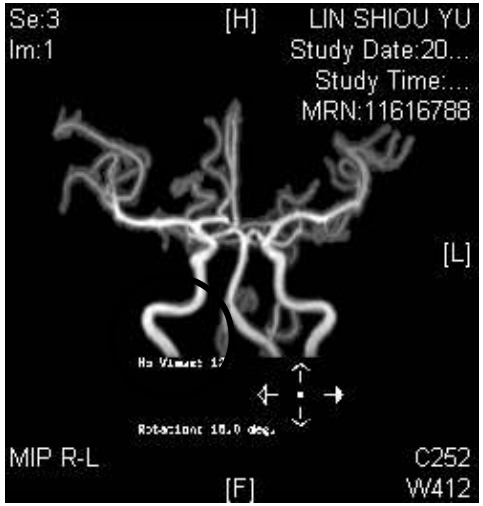
  no dysathria, focal weakness

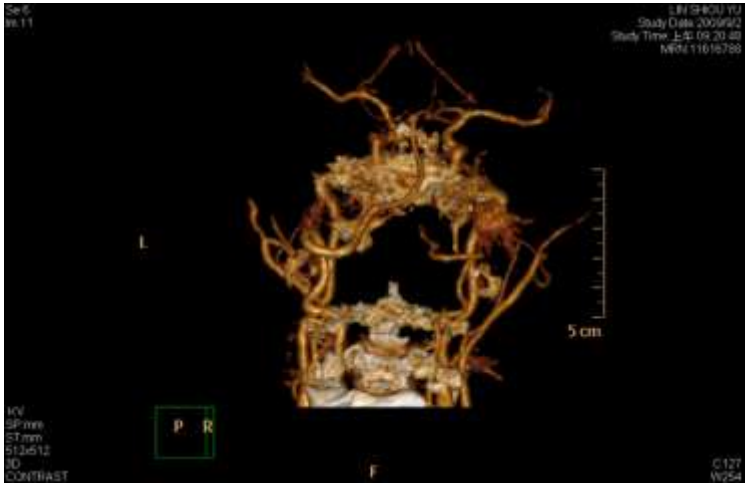
  sensory: normal FNF/HKS: normal

DX: RIGHT VA DISSECTION WITH MEDULLARY INFARCTION









KEYNOTES

- Occipital headache with or without ipsilateral neck pain, tenderness(++)
- Nuchal pain located in posterolateral aspect of the neck, bruit(-)
- Followed by delayed ischemic symptoms in the vertebrobasilar distribution

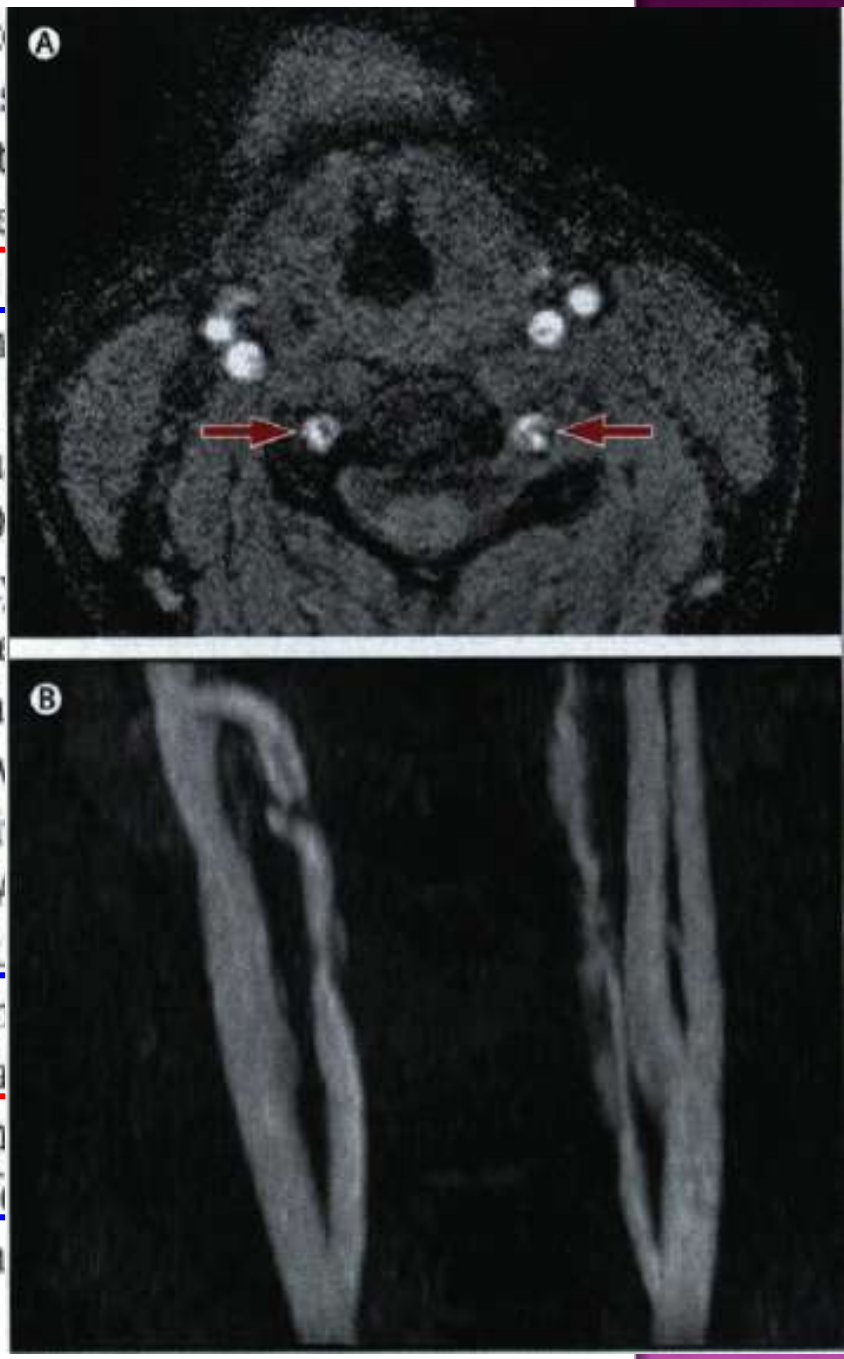
  (cerebellar, Wallenberg', or Horner's sign)
- Young patient
- MRA with contrast, CTA, or angiography
- Headache/vertigo improved after day 3

Thunderclap headache—and a tender neck

In May, 2007, a 22-year-old woman was driven to the emergency room by her mother, after developing a severe headache. The headache had begun suddenly at the top of her forehead, before radiating, then moving, to the back of her head. When the headache started, the patient had seen "lightning bolts", then lost the vision in her right eye for several seconds; her vision had since been blurred, with diplopia. She had also been feeling sick and vomiting. She had previously felt well. There was no recent head injury or strenuous activity. She was not drowsy. She did not take any medications, alcohol, or illicit drugs. Her medical history was notable only for migraine—she had five to six episodes per year, each lasting about 12 h. Although her migraines manifested as headache, blurred vision, and nausea, this episode felt very different, since the symptoms were more severe and were accompanied by neck pain and diplopia.

Lancet 2007; 370: 1974

With her right eye, the patient was able t
ment but was unable to count fingers at a di
60 cm. Fundoscopy showed nothing of not
and back of the neck were exquisitely tend
but Kernig's and Brudzinski's signs were
rest of the physical examination was unrem
over, vision returned to normal within 2–3
including the ESR (20 mm/h), gave norm
CSF was clear, and contained only 1×10
cells per L, 82% of which were lymphoc
centrations of protein and glucose were
2·8 mmol/L respectively (plasma concentra
4·6 mmol/L). Microscopy and culture show
in the CSF. MRI of the brain with and wi
and magnetic-resonance angiography (MR
showed nothing abnormal. However, MR
(figure) showed intraluminal flaps and r
osis, signifying dissection of both vertebra
patient was treated with heparin, then with
headache and vomiting ceased within a f
was discharged 5 days after admission. Wh
July, 2007, she was well.



Expert Opinion

Headache in Cervical Artery Dissections

CLINICAL HISTORY

A 36-year-old woman was eating dinner when she developed acute stabbing and throbbing pain going from the left anterior neck up to the left side of her face. There was no associated nausea, light sensitivity, visual symptoms, nor sensorimotor complaints. The pain was severe for about 5 hours. She went to an emergency room and received a narcotic injection, with benefit. The next morning the pain was still present but slight and then resolved that day. There was no history of head or neck trauma. For several months before admission she had been intermittently painting her house but had not done any painting of the house or ceilings for a number of weeks. Four days before the onset of pain she had her hair washed in a salon with her neck in an hyperextended position in the basin but had no associated symptoms. There was a history of intermittent tension-type headaches but no migraine and also of cervical and lumbar myofascial pain occurring intermittently. She had no history of hypertension, diabetes mellitus, ischemic heart disease, or cerebrovascular disease.

(*Headache*. 2002;42:1061-1063)

When I examined her the next day the general physical examination was normal except for left carotid bulb tenderness and left posterior cervical paraspinal tenderness, with full range of motion of the neck. Neurological examination was entirely normal. A magnetic resonance image of the brain with magnetic resonance angiogram (MRA) sequence of the brain and neck revealed dissection of the upper cervical left internal carotid artery with marked stenosis of the arterial lumen and normal intracranial, vertebral, and basilar arteries. A cerebral arteriogram confirmed dissection of a long segment of the left internal carotid artery above the bulb and also demonstrated dissection of both vertebral arteries in their distal portions, with poor anterograde flow in the distal basilar artery. She was placed in a cervical collar and treated with heparin and warfarin. An ultrasound of the thoracic aorta and computed tomography of the chest were normal. An antinuclear antibody titer was elevated at 1/160 with a speckled pattern. Anti-DNA, Anti-SSA, SSB, and ANCA IgG, IgM levels were all negative. C3 and C4 levels were normal. An RPR was nonreactive. A urine homocysteine level was normal. At discharge her neurologic examination remained normal.

Pain as the only manifestation of internal carotid artery dissection

Cephalalgia 1992;12:314-17.

Patient I

A 40-year-old woman with a past history of migraine without aura fell while skiing without sustaining head or neck trauma. Five hours later she complained of headache and 1 h later of cervical pain. The cervical pain was severe and right-sided, going from the mid-cervical region to the angle of the mandible. The headache was moderate, bilateral and diffuse, and unassociated with nausea, vomiting, photophobia or phonophobia. There was neither pulsatile tinnitus nor symptom of retinal or cerebral ischemia. Neurological and fundoscopic examinations one day later were normal. Palpation of the neck revealed tenderness over the right internal carotid artery without swelling or increased pulsations. She had no cervical bruits.

Patient 2

A 37-year-old woman with a history of mild arterial hypertension and migraine without aura was eating when she abruptly experienced a left-sided excruciating and throbbing temporal headache, described as "something exploding in the head". The headache was accompanied by phonophobia, photo-phobia and vomiting. Twenty-four hours later the headache became bilateral and less severe. Four days later, while the headache was still present, she developed severe bilateral cervical pain and neck stiffness. There was no Horner's syndrome, pulsatile tinnitus or symptoms of retinal or cerebral ischemia. Neurological and general examinations were normal except for tenderness over both internal carotid arteries in the neck, without swelling, increased pulsation or bruit. Computed tomography of the head and cerebrospinal fluid were normal. Carotid angiography (Fig. 2) disclosed a tapered occlusion of the right internal carotid artery distal to the bifurcation, and a long tight stenosis of the left internal carotid artery, indicating bilateral extracranial internal carotid artery dissections.

Symptoms less likely to happen in Migraine

- Not like previous migraine
- Prominent neck pain and tenderness (Carotidynia)
- Facial pain

- Horner's sign
- Cranial nerve palsy

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- Introduction
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ANATOMY

- ICA dissection:

  starts at 1.5-2.0 cm above carotid bifurcation

  (different from atherosclerosis, which characteristically affects the carotid bulb)

  ends at skull base, before penetrates the petrous bone

ANATOMY

- VA dissection:

  V1 Segment:

  subclavian ~ before entering V6

  V3 segment

  originate at C1-2 level as the artery leaves the transverse foramen of the axis (C1)

NEUROIMAGE STUDY

- MRI+MRA of neck: fat suppressed T1-weighted sequence→ identified intramural hematoma

  eccentric: crescent

  concentric: doughnut, easily stroke
- CTA
- Ultrasound: ICA dissection, f/u
- Pseudoaneurysm→ SAH (very rare)

ANGIOGRAPHIC SIGNS OF CAD

- Tapered luminal narrowing (string sign) with stenosis, with occlusion
- Pseudoaneurysm (segmental dilatations)
- Oval segmental dilatation of the lumen
- Extraluminal pouch
- Small dilatation at the end of a string sign (rat's tail)
- Intimal flap
- Double lumen

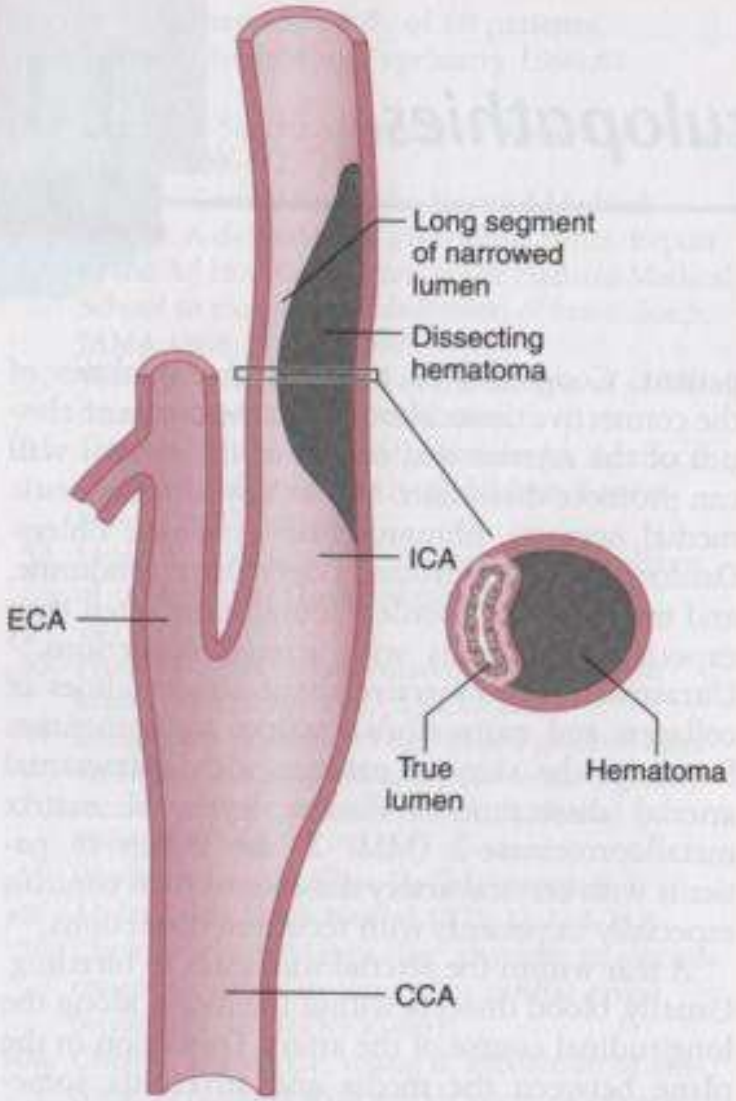


Figure 11-1. Cartoon showing dissection of the internal carotid artery. The inset is a cross-section view showing the hematoma and the luminal compromise. CCA, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery.

spells often occur in rapid succession over hours or a few days leading Fisher to coin the term

Figure 11-2 is a cartoon that illustrates various

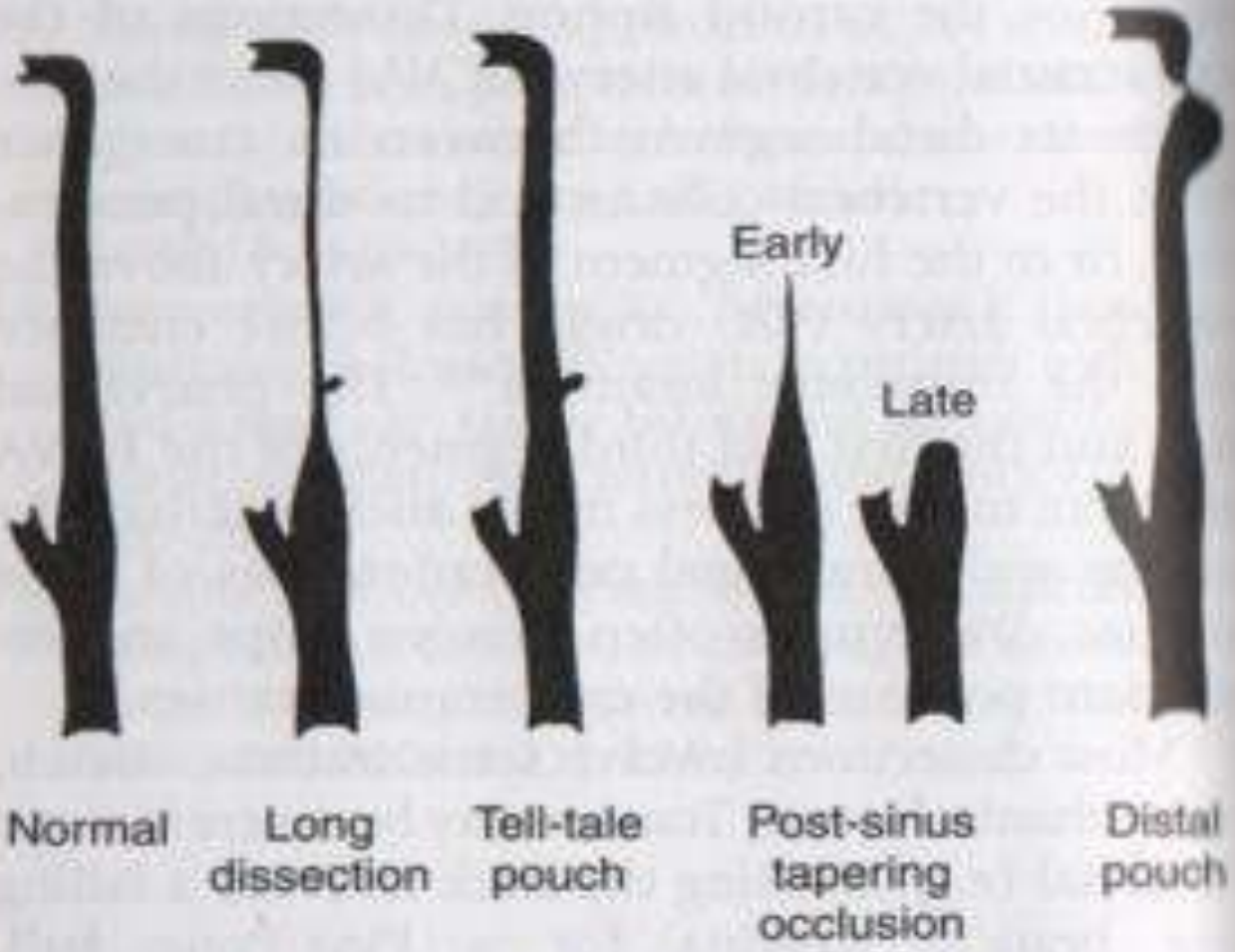


Figure 11-2. Drawings of various abnormal carotid arteriograms in patients with carotid-artery dissection. (From Fisher CM, Ojemann RG, Robertson GH: Spontaneous dissection of cervicocerebral arteries, Can J Neurol Sci 1978;5:9-19, with permission.)

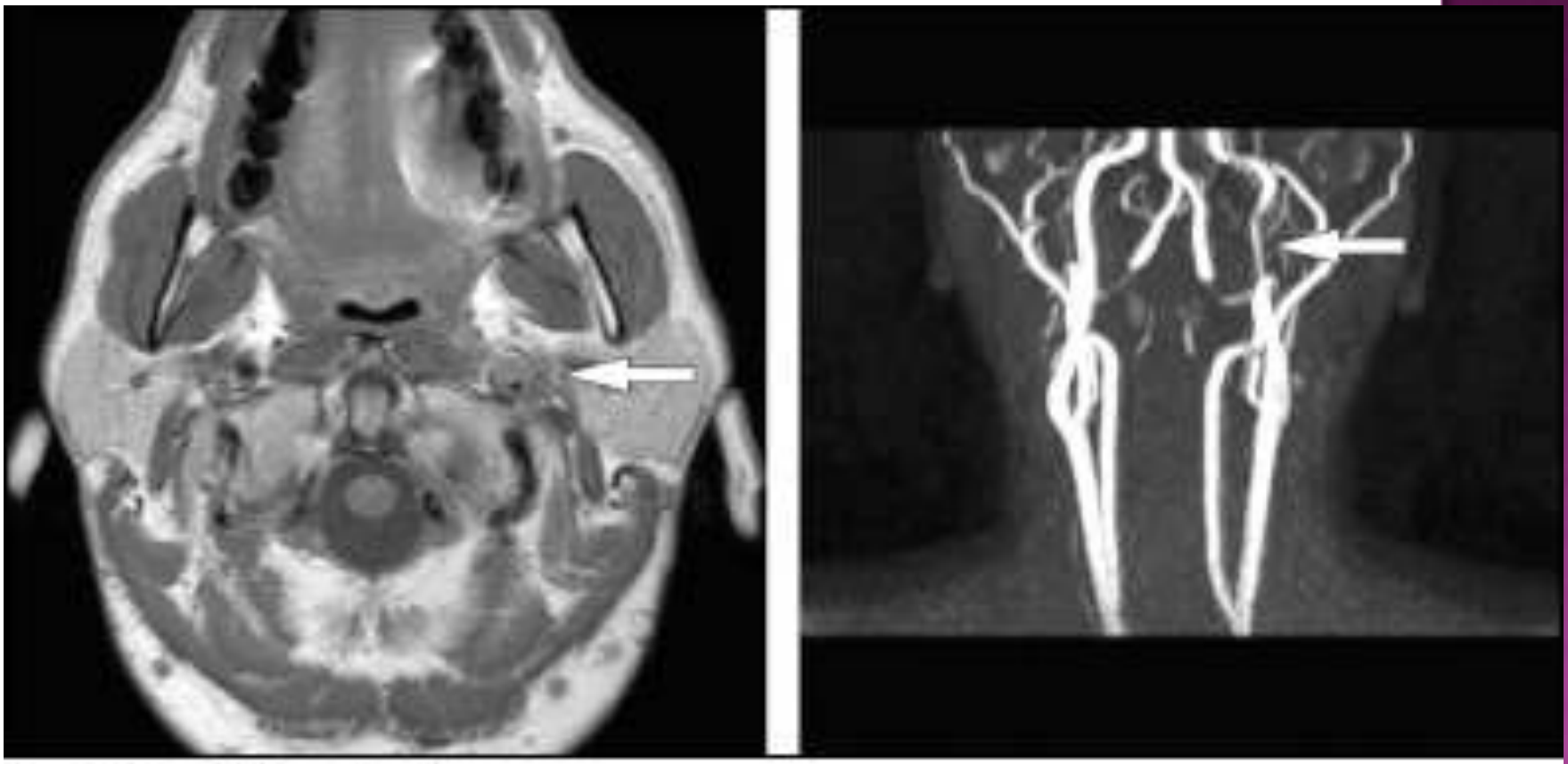
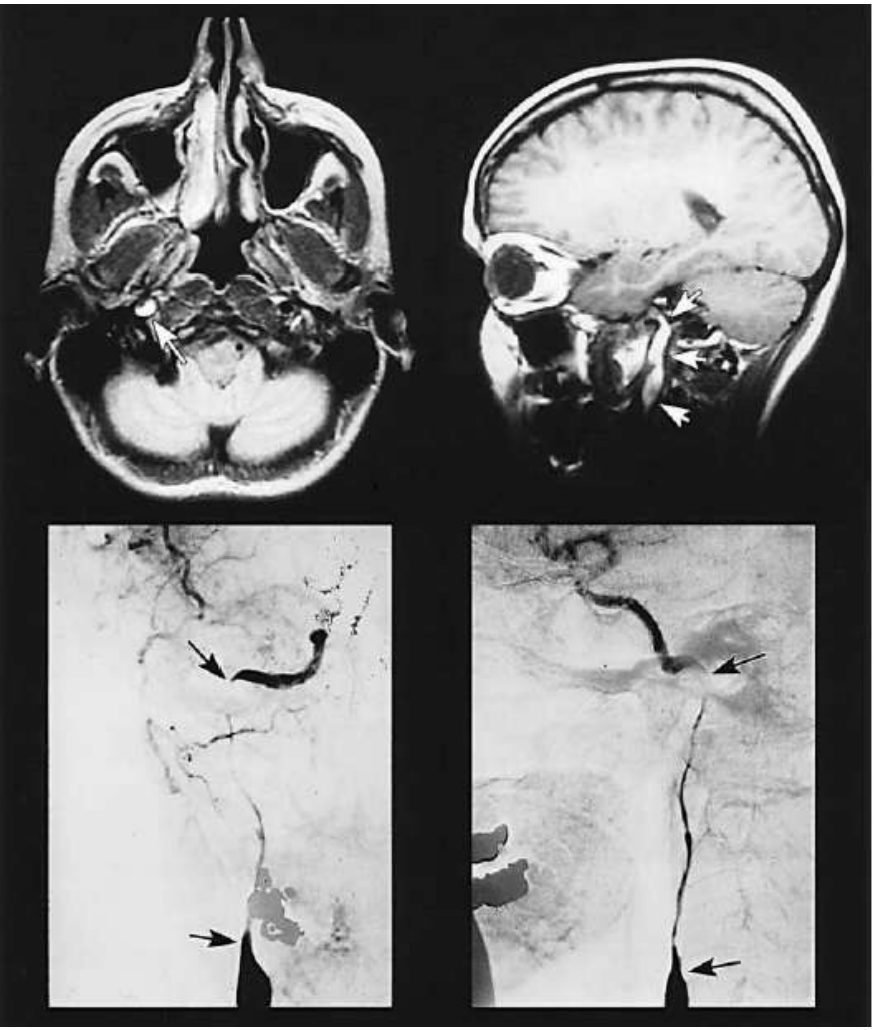
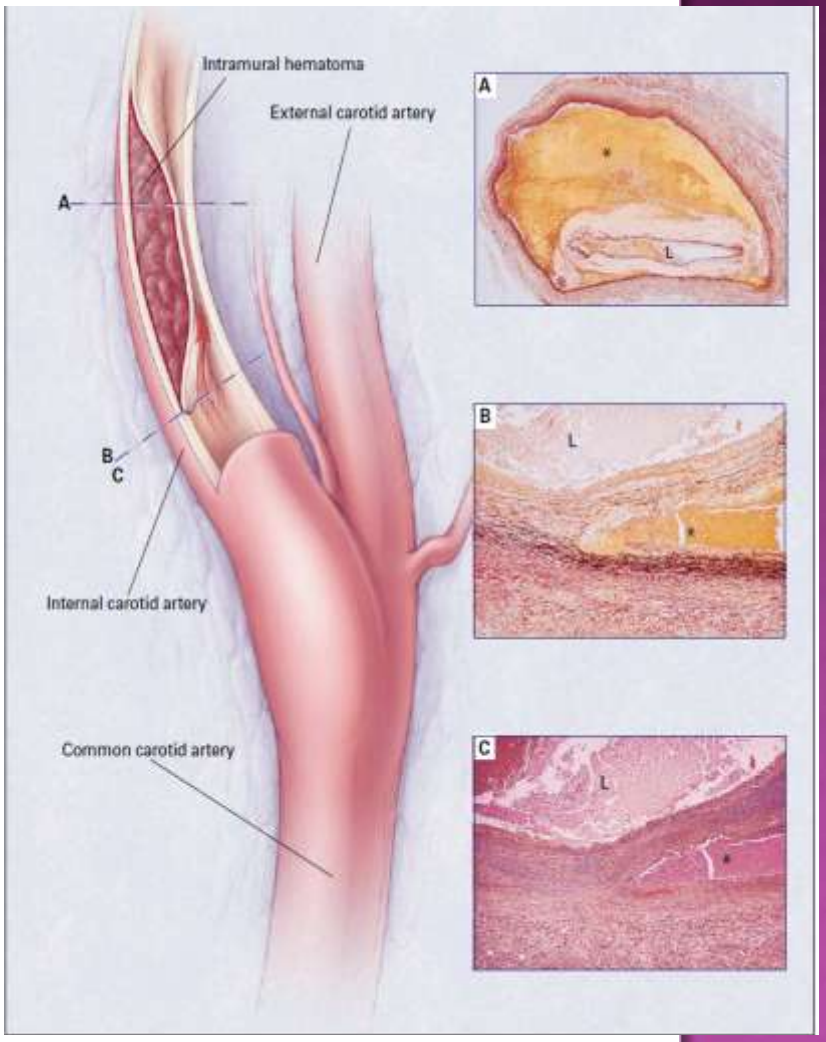


Figure 2: Cervical artery dissection
Left: Axial FLAIR MRI reveals a false lumen and crescent sign in the left internal carotid artery (arrow). Right: Gadolinium bolus magnetic resonance angiogram reveals tapered stenosis of the mid-cervical portion of the left internal carotid artery.




Intramural hematoma
External carotid artery
A
B
C
Internal carotid artery
Common carotid artery
A
L
B
L
C
L

OUTLINE

- Introduction
- Pathophysiology
- Clinical Symptoms
- Neuroimage
- Treatment & Prognosis

TX

- Controversial, lack randomized trial
- Do nothing
- Aspirin
- Heparin→ warfarin → aspirin
  after following MRA

Tx

- A growing minority of clinicians are using aspirin instead of anticoagulation to prevent stroke in dissection (100~300mg/qd)
- 2003 Cochrane review: 26 studies, total 327 p’ts: no difference between aspirin and anticoagulants groups
- The fear that anticoagulant or intravenous tPA therapy to extend he dissection: unfounded
- Plavix, enoxaparin: also used, but no evidence
- Angioplasty in complicated case
- Repeated vascular image 2-3 months later

PROGNOSIS

- Lack large-scaled study
- Prognosis of stroke:

  ¾ good functional recovery, <5% death
- Recanalization is possible within the first few weeks, more common in VA dissection
- Aneurysm formation as consequence of dissection, but their prognosis is benign

- Recurrence of dissection: possible but low risk(1% per annum), particularly after the first two months (2%)

  → exception: connective tissue disease

Prognosis

- Headache spontaneously resolves within a few days, >90% in one week, few persisted for years
- Previous migraine➔ also improved in ¾ p’ts
- Dissection (dynamic process)& pain

  → days later ischemic stroke symptoms occur

  → 2/3 rapid recanalization and resolution of image and clinical finding

  → good recovery of pain and less recurrent possibility

RECURRENT STROKE OR TIA AFTER CAD

	Sample size*	CAD type	Mean follow-up (years)	Recurrence rate (%)†	Type of event (n)
Guillon et al[31]	16	ICAD (aneurysmal)	3·1	0	--
Touzé et al[105]	35	VAD and ICAD (aneurysmal)	3·5	0	--
Touzé et al[9]	457	VAD and ICAD	2·6	2·7	Stroke (4), TIA (8)
Bogousslavsky et al[114]	23	ICAD (with stroke)	3·2	4·2	Stroke (1)
Arauz et al[11]	130	VAD and ICAD	1·6	4·6	Stroke (6)‡
Leys et al[81]	105	VAD and ICAD	3·0	4·8	Stroke (2), TIA (3)
Gonzales-Portillo et al[106]	27	VAD and ICAD	4·8	7·4	Stroke (2)§
Benninger et al[107]	38	ICAD (aneurysmal)	6·5	7·9	Stroke (3)
Treiman et al[108]	24	ICAD	9·3	8·3	TIA (2)
Engelter et al[109]	33	ICAD	2·3	9·1	Stroke (2), TIA (1)
Kremer et al[110]	92	ICAD	6·7	11·9	Stroke (5), TIA (6)¶
Beletsky et al[9]	105	VAD and ICAD	1·0	13·3	Stroke (9), TIA (5)‖

Studies describing recurrent events without details on the type of event were not included in this table.[101] de Bray and co-workers[14] reported a rate of stroke recurrence of 0·4% per year, but did not mention the exact number of events and the type of stroke. Wessels and co-workers[111] reported a rate of stroke recurrence of 6%, but the population included both extracranial and intracranial vertebral-artery dissections. CAD=carotid-artery dissection. ICAD=internal carotid-artery dissection. TIA=transient ischaemic attack. VAD=vertebral artery-dissection. *With follow-up data available. †Absolute rate. ‡In the first 2 weeks after the diagnosis. §One ischaemic stroke occurred after percutaneous balloon angioplasty for treatment of a persistent vertebral-artery pseudoaneurysm. ¶One patient also had a subdural haematoma during follow-up. ‖Of these events, five stroke and three TIA recurrences occurred before enrolment in the study (the rate of recurrence was 5·7% after enrolment).

Table 1: Risk of recurrence of ischaemic stroke or TIA in patients with CAD

RECURRENT CAD

	Sample size*	CAD type	Mean follow-up (years)	Recurrence rate (%)†
Mokri et al[8]	36	ICAD	4·9	0 (0)
Guillon et al[21]	16	ICAD (aneurysmal)	3·1	0 (0)
Engelter et al[22]	33	ICAD	2·3	0 (0)
Touzé et al[23]	35	VAD and ICAD (aneurysmal)	3·5	0 (0)
Gonzales-Portillo et al[24]	27	VAD and ICAD	4·8	0 (0)
Beletsky et al[9]	105	VAD and ICAD	1·0	0 (0)
Arauz et al[25]	130	VAD and ICAD	1·6	0 (0)
Lee et al[4]	48	VAD and ICAD	7·8	0 (0)
Nedeltchev et al[26]	154	ICAD	1·0	1 (0·6)
Touzé et al[5]	457	VAD and ICAD	2·6	4 (0·9)
Kremer et al[27]	92	ICAD	6·7	1 (1·1)
Desfontaines et al[28]	60	ICAD	3·1	1 (1·7)
Leys et al[29]	105	VAD and ICAD	3·0	3 (2·9)
d'Anglejan Chatillon et al[30]	62	ICAD	3·4	2 (3·2)
Bassetti et al[31]	81	VAD and ICAD	2·8	3 (3·7)
Bogousslavsky et al[32]	23	ICAD (with stroke)	3·2	1 (4·3)
Dziewas et al[33]	126	VAD and ICAD	0·5	6 (4·8)‡
de Bray et al[34]	103	VAD and ICAD	4·0	5 (4·9)§
Schievink et al[7]	200	VAD and ICAD	7·4	14 (7·0)¶
Treiman et al[35]	24	ICAD	9·3	2 (8·3)
Dittrich et al[36]	36	VAD and ICAD	0·6	9 (25·0)‖

Studies describing recurrent events without details on the type of event were not included in this table.[37] Wessels and co-workers[10] reported a rate of CAD recurrence of 3%, but the population included both extracranial and intracranial vertebral-artery dissections. CAD=cervical-artery dissection. ICAD=internal carotid-artery dissection. VAD=vertebral-artery dissection. *With follow-up data available. †Absolute rate. ‡Within 1 month in four patients, and within 1 year for two patients. §Two recurrences occurred 1 year after the first dissection and the remaining recurrences occurred 4, 9, and 10 years later.[34] ¶Two patients also had a renal-artery dissection during follow-up. ‖The recurrence occurred within 1 to 4 weeks after the initial CAD in seven patients.[36]

Table 2: Risk of recurrence of CAD

MORTALITY DURING ACUTE CAD

	Sample size*	Dissection type	Mean follow-up (years)	Mortality rate (%)†
Touzé et al[108]	35	VAD and ICAD (aneurysmal)	3·5	0 (0)
Touzé et al[5]	459	VAD and ICAD	2·6	0 (0)‡
Kremer et al[109]	92	ICAD	6·7	0 (0)
Mokri et al[8]	36	ICAD	4·9	0 (0)
Mokri et al[110]	25	VAD	3·8	0 (0)
Schievink et al[6]	200	VAD and ICAD	7·4	1 (0·5)
Ast et al[111]	68	ICAD	1·0	1 (1·5)
d'Anglejan Chatillon et al[112]	62	ICAD	3·4	1 (1·6)
de Bray et al[14]	103	VAD and ICAD	4·0	2 (1·9)
Lee et al[4]	48	VAD and ICAD	7·8	1 (2·1)
Engelter et al[113]	33	ICAD	2·3	1 (3·0)
Arauz et al[11]	130	VAD and ICAD	1·6	4 (3·1)
Bassetti et al[17]	81	VAD and ICAD	2·8	3 (3·7)
Treiman et al[19]	24	ICAD	9·3	3 (12·5)
Pozzati et al[21]	19	ICAD (occlusive)	8·2	3 (15·8)
Bogousslavsky et al[114]	30	ICAD (with stroke)	3·2	7 (23·3)

CAD=cervical-artery dissection. ICAD=internal carotid-artery dissection. VAD=vertebral-artery dissection. * Studies not included in this table were those that included only patients with follow-up data available,[121] as they are biased towards patients who survived the acute stage; those that reported mortality rates after CAD, but that did not mention the delay between CAD occurrence and death; and those that did not report the duration of follow-up.[122] †Within the first month or during the initial hospitalisation. ‡Two patients died after the acute phase (at 2·4 and 23·7 months).

Table 3: Mortality during the acute stage of CAD

CONCLUSION

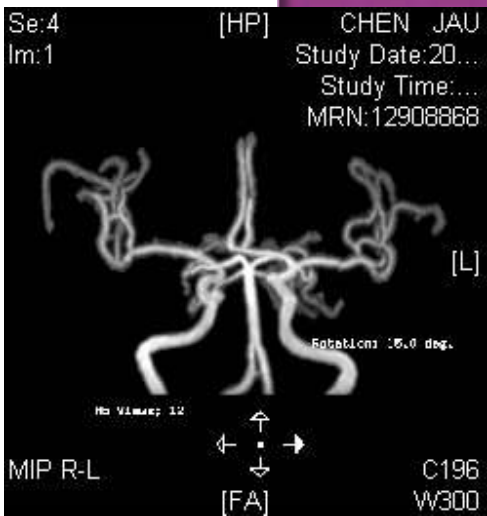
- CAD: well-recognized cause of young stroke, related to distal thromboembolism
- Spontaneous CAD: true mechanism unknown
- Pain with minor focal sign→ delayed stroke
- Outcome: generally favorable, but permanent neurologic deficits and even death may result
- Early initiation of antiplatelet or anticoagulation therapy→ possibly preventing more serious cerebral ischemic complication

CONCLUSION: CAD DX

- Pain/Headache is frequently the earliest symptom (60-75%)
- Neck pain associated headache:
  25% in carotid dissection and 50% in VA dissection
- Headache mimicking CAD:
  migraine, cluster headache,
  primary thunderclap headache, SAH
- Headache may be sole symptoms of disseciton
- Carotidynia help DD dissection from migraine

CONCLUSION: CAD DX

- Young patient with painful Horner syndrome or Wallenberg's syndrome may hint cervical arterial dissection
- Brain CT, MRI+MRA and L.P are unrevealing
- Ultrasound, Neck CTA/MRA, conventional angiography



	ICAD	VAD
Neck Pain	Anterolateral (25%)	Posterior (50%) Some bilateral
Headache	Frontotemporal	Occipital
Facial Pain	(+) 10% Eye, facial, ear pain	(-)
Other focal sign	Partial Horner's sign Cranial nerve palsy	Cervical radiculopathy
Pain to Stroke time	4 days	14.5 hours
Stroke	Anterior circulation	Posterior circulation
Typical syndrome	TIA/ TMB precedes	Wallernberg's syndrome

Headache	ICAD	VAD
Location	Frontotemporal 60%	Occipital 83%
Initial symptoms	47%	33%
Onset	Gradual	Gradual
Nature	Steady 73% Pulsating 25%	Steady 56% Pulsating 44%
Severity	Varied	varied
Misdiagnosed as	Migraine, Cluster headache TCH/SAH	Musculoskeletal dz

6.5.1 HEADACHE OR FACIAL OR NECK PAIN ATTRIBUTED TO ARTERIAL DISSECTION

A. Any new headache, facial or neck pain of acute onset, with or without other neurological symptoms or signs and fulfilling criteria C and D
B. Dissection demonstrated by appropriate vascular and/or neuroimage investigations
C. Pain develops in close temporal relation to and on the same side as the dissection
D. Pain resolves within 1 month

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