

Dissection and Stroke

CoxHealth Stroke Conference 2017

Stroke

2017

Thoracic Aorta

Supra-aortic Trunks

Carotid Artery

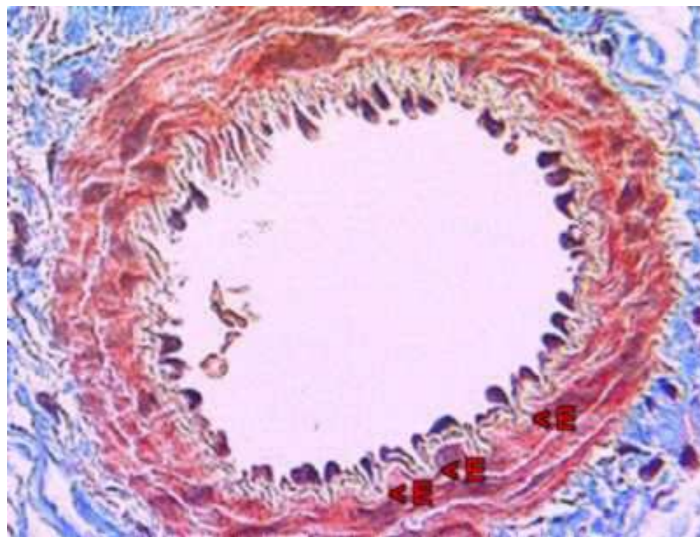
Vertebral Artery

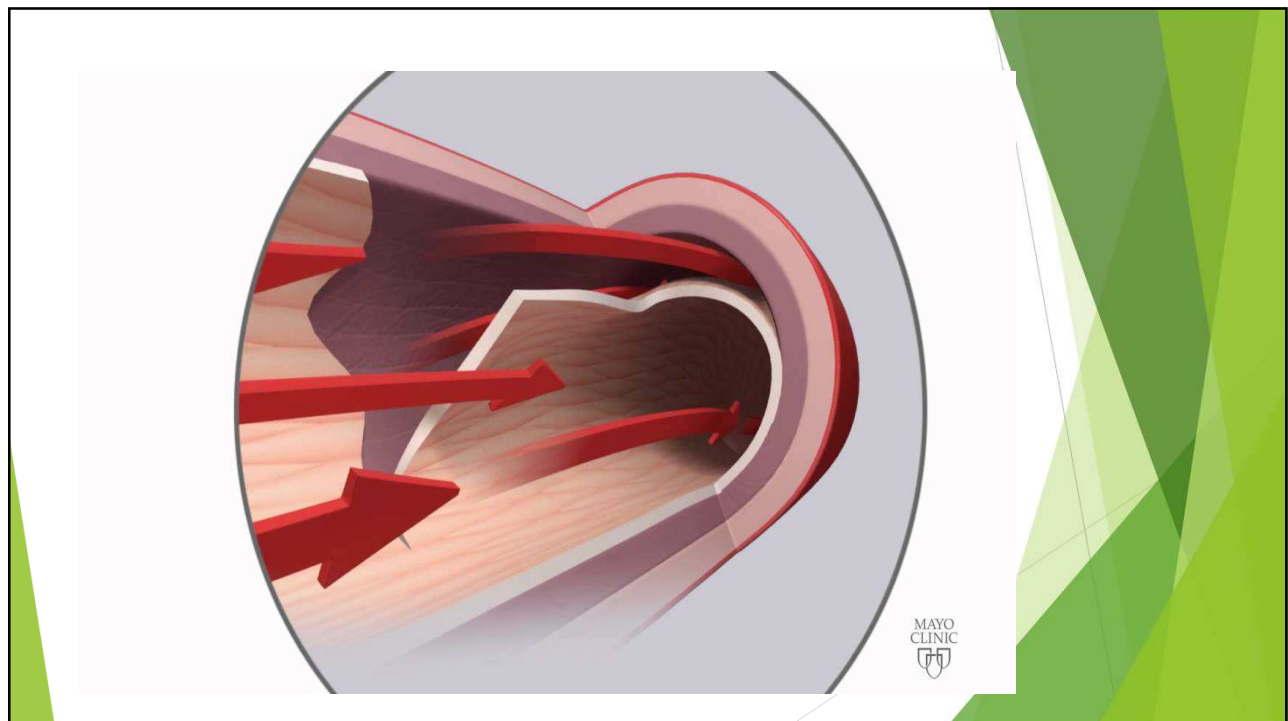
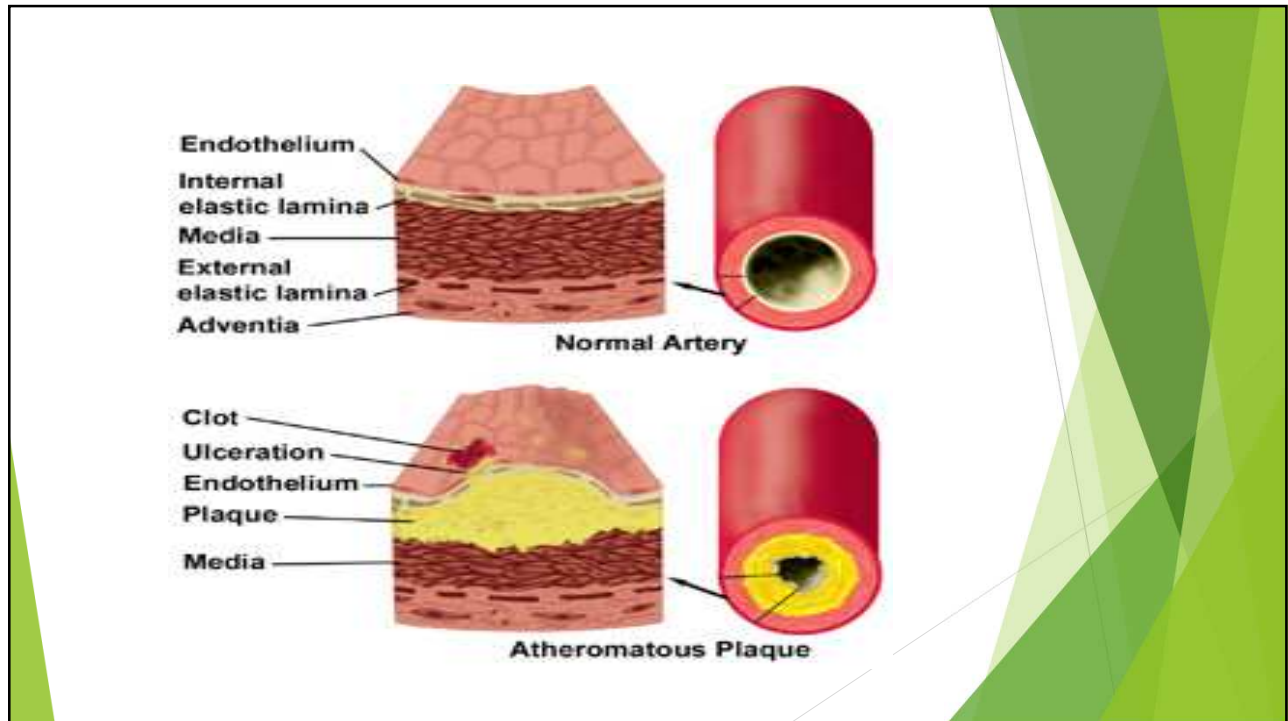
What is a Dissection?

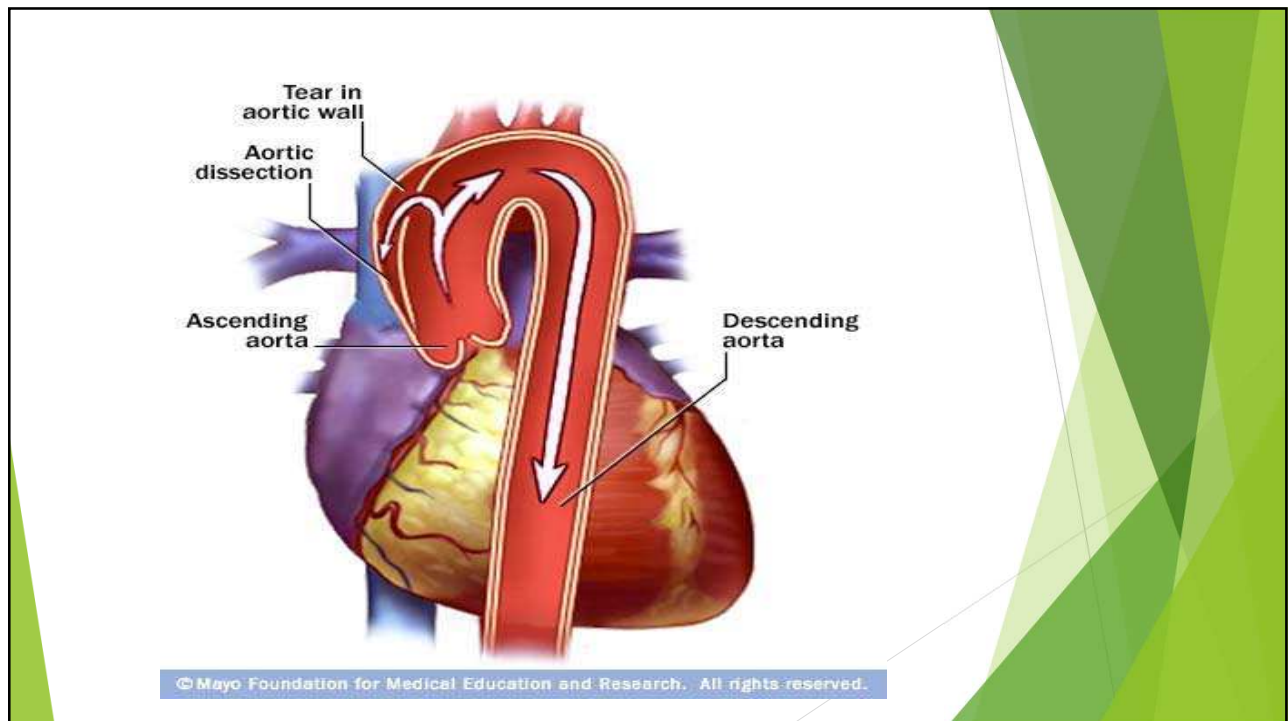
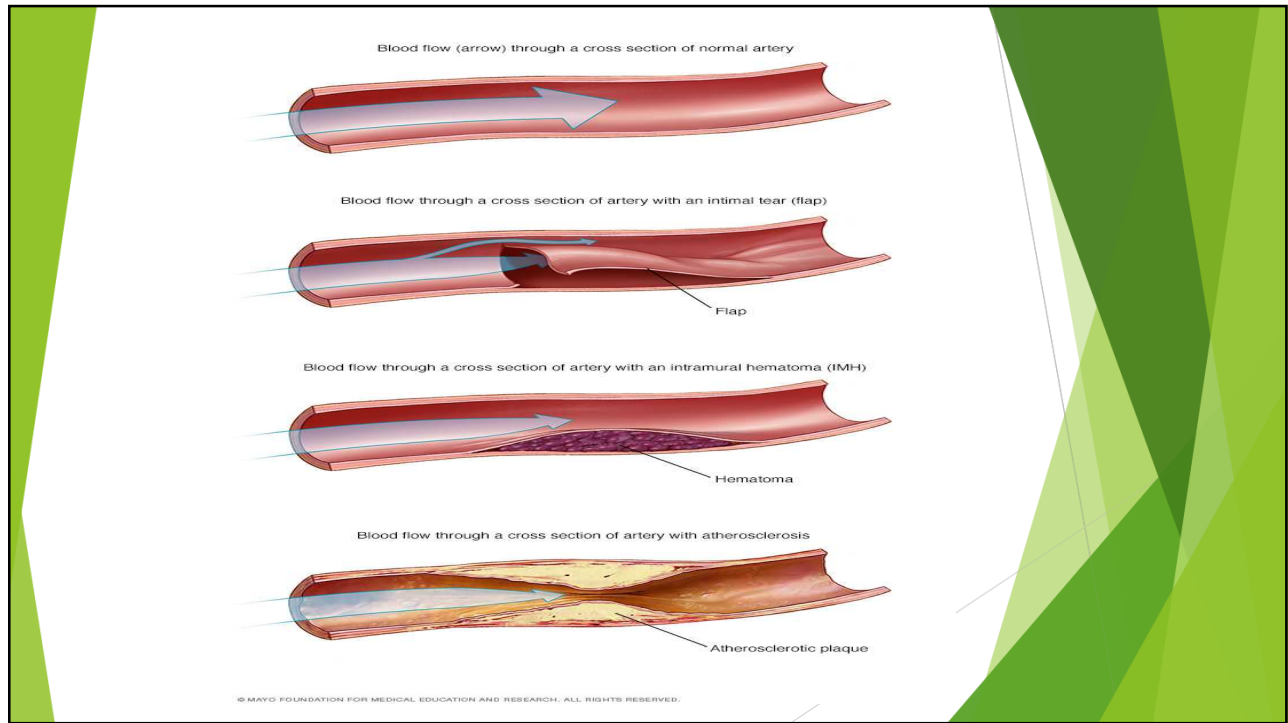
The development of a tear of the inner lining of the artery --the intima-- extending into the media

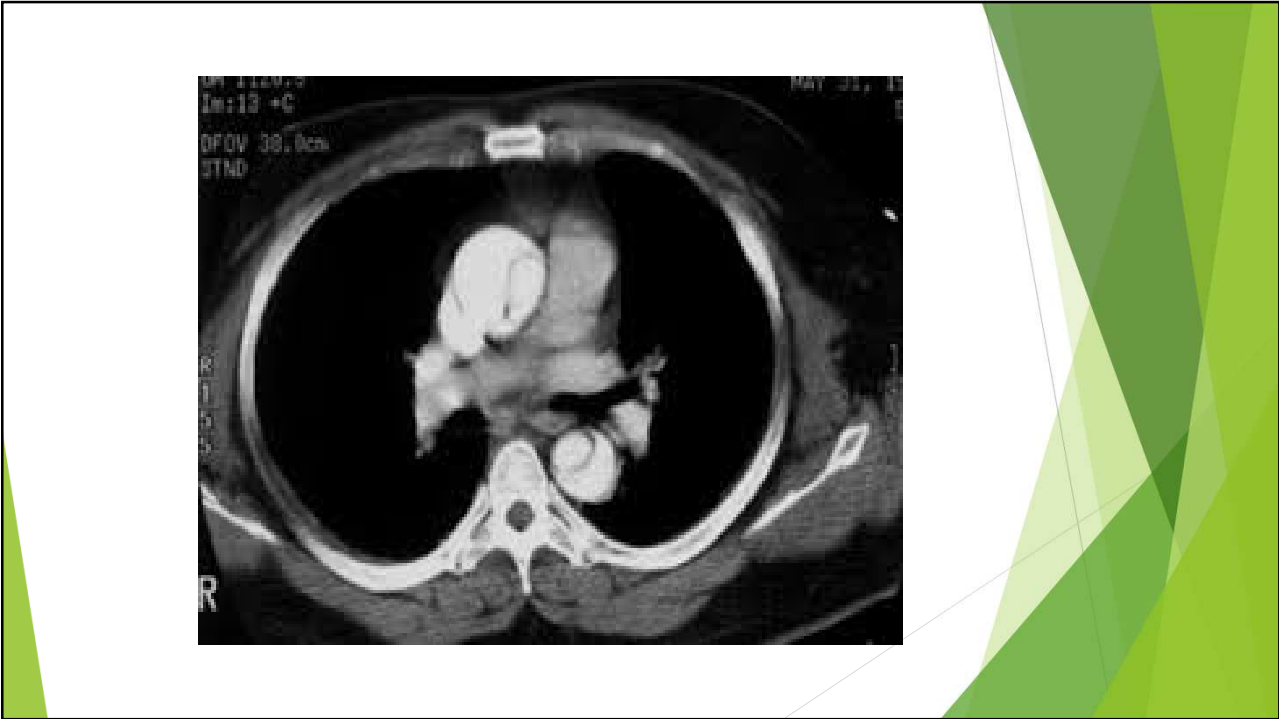
This tear may extend distally, disrupting branches or ultimately leading to aneurysm formation

Thrombus formation and embolization is a potential outcome









Dissection of the Ascending Aorta

- ▶ Incidence: At least 30 per million per year
- ▶ Most common manifestation of acute aortic syndrome
- ▶ Mortality of 1% per hour
- ▶ First repair DeBakey, Cooley and Creech 1954

WHO?

Male

Age: 60s and
70s

Hypertension

Prior cardiac
surgery (aortic
valve repair)

Bicuspid aortic
valve

Atherosclerosis
of aorta

WHO?

Less than 10%:

--younger than age 40

--normotensive

--history of cardiac surgery or bicuspid aortic valve

Marfan syndrome, Ehlers-Danlos syndrome, etc

Ascending Dissection: Presentation

- ▶ Gaul C, Dietrich W, Friedrich I et al.
- ▶ Neurological symptoms in type A aortic dissection.
- ▶ Stroke 2007;38:292-7.
- ▶ Dept of Neurology, Nuremburg

Gaul et al, STROKE

- ▶ **Background and Purpose**— Aortic dissection typically presents with severe chest or back pain. Neurological symptoms may occur because of occlusion of supplying vessels or general hypotension. **Especially in pain-free dissections diagnosis can be difficult and delayed.**
- ▶ **Methods**— Clinical records of 102 consecutive patients with aortic dissection (63% male, median age 58 years) ...analyzed for medical history, preoperative clinical characteristics, treatment and outcome with **emphasis on neurological symptoms.**
- ▶ **Results**— Thirty patients showed initial neurological symptoms (29%). **Only two-thirds of them reported chest pain...** Neurological symptoms were attributable to ischemic stroke (16%), spinal cord ischemia (1%), ischemic neuropathy (11%), and hypoxic encephalopathy (2%).

“In aortic dissections, neurological symptoms are often dramatic and may dominate the clinical picture and mask the underlying condition.”

“The frequency of neurological involvement varies from 17% to 40%. **Many neurological findings have supposedly been overlooked because of incompleteness of neurological examination in critically ill patients.** ”

“Remarkably, pain is not an obligatory symptom of aortic dissection.”

“13.9% of patients... noted no pain, matching reported ranges of pain-free dissections between 5% and 15%.

“Approximately half of patients who did not report pain solely showed neurological symptoms.”

Presentation:

? Chest Pain ?

Neuro symptoms in 29% of patients

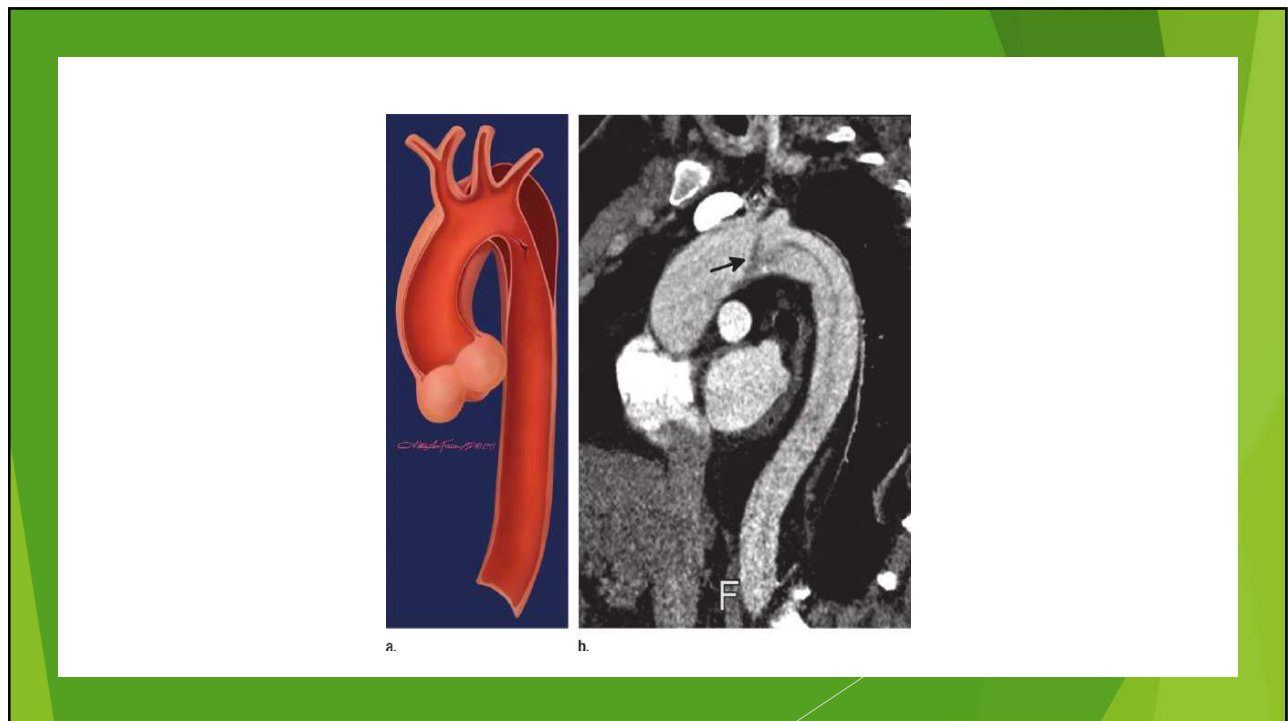
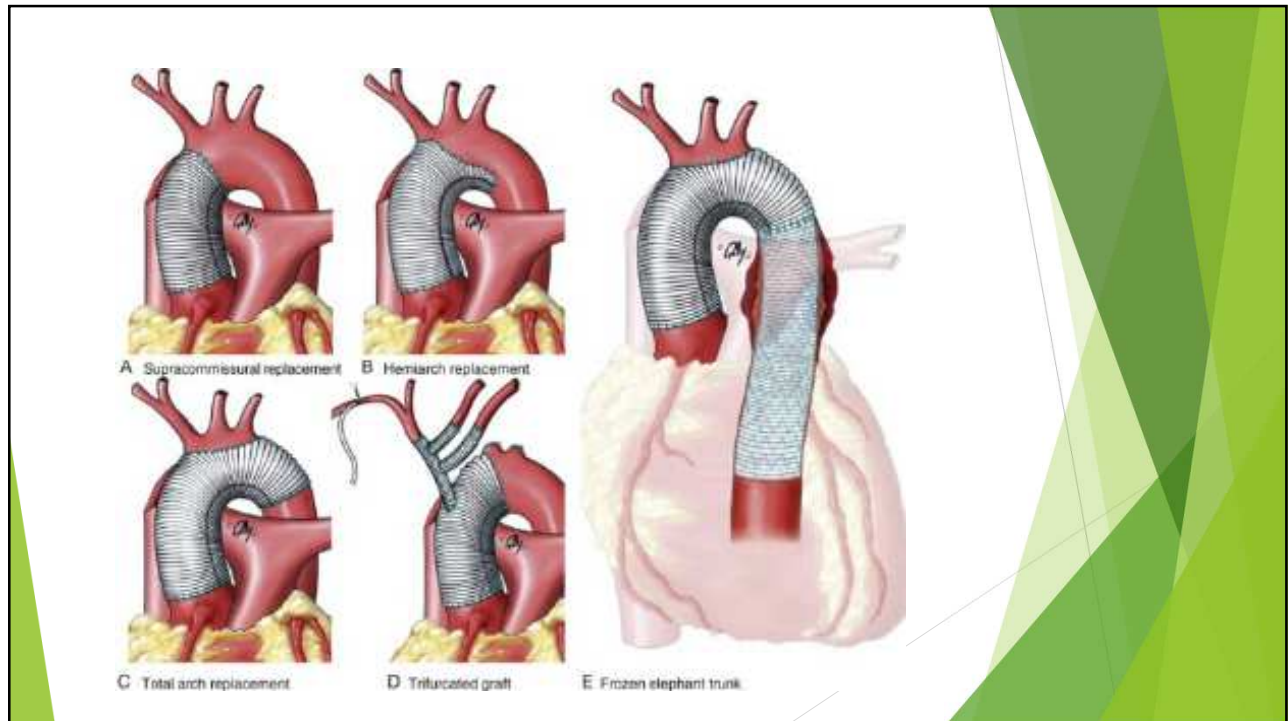
--ischemic stroke 16%

--ischemic neuropathy 11%

--hypoxic encephalopathy 2%

--spinal cord ischemia 1%





Brachiocephalic Dissection

Isolated dissecting aneurysm of the brachiocephalic artery associated with contained rupture

Tomoaki Hirose, MD · et al

Gen Thorac Cardiovasc Surg (2012) 60:225-22

A 79-year-old man with no history of trauma with sudden onset of headache and backache. CT showed an isolated dissection associated with contained rupture of... the brachiocephalic artery. The proximal aortic arch was successfully replaced using antegrade selective cerebral perfusion. The patient...recovered uneventfully.

Brachiocephalic Artery Dissection

- ▶ [BMJ Case Rep](#). 2015; 2015: bcr2014208815.
- ▶ Published online 2015 Aug 27.
- ▶ [10.1136/bcr-2014-208815](https://doi.org/10.1136/bcr-2014-208815)
- ▶ **Isolated brachiocephalic artery dissection presenting as acute stroke**
- ▶ [Hariharasudan Mani](#) and [Sharat Ahluwalia](#)

Brachiocephalic Dissection

A 41-year-old obese man with **undiagnosed hypertension**

Presented with ataxia, slurred speech and left facial weakness

Onset: acute, but the day before

Sought medical help the next morning for persistence of symptoms

Non smoker, no other medical history or trauma

Family history: grandparents had strokes in their 60s, cause unknown.

No family history of coagulopathy, aortic dissection or connective tissue disorders

Brachicephalic Dissection: Exam

BP 190/119 mm Hg LEFT, 170/110 mmHg
RIGHT

Left facial asymmetry, upper motor facial palsy

Minimal dysarthria

No extremity motor or sensory deficits

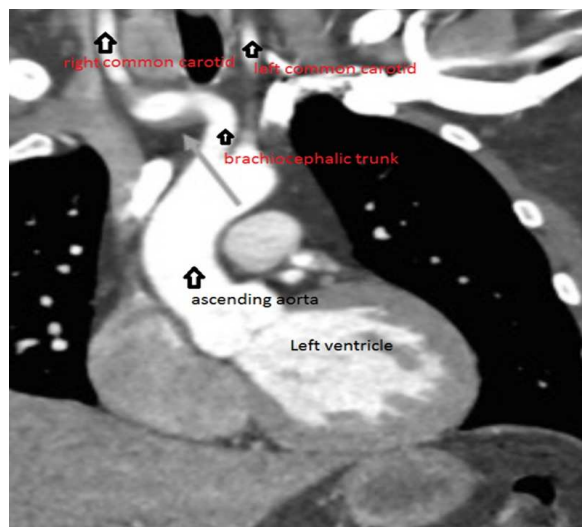
No cerebellar signs

On standing, tendency to drift left

Brachiocephalic Dissection: Imaging

CT: acute ischemic infarct of the right basal ganglia.

Brachiocephalic Dissection: Imaging



Now What?

- Other work-up: hypercoagulopathy lab and echo are normal
- Treated with heparin anticoagulation, bridged to Coumadin
- Long Term: Minimal residual dysarthria and facial asymmetry

- ▶ **Aortic dissection** most commonly presents with tearing chest pain, but **14% of patients discussed here had no pain and only neurologic symptoms**
- ▶ **Supra Aortic Trunk Dissection** may present with minimal or misleading symptoms (headache, backache, ataxia, dysarthria)
- ▶ Subsequent ischemic stroke is believed to be due to thromboembolism
- ▶ **Antithrombotic therapy**, with either full anticoagulation or antiplatelet agents, **is the treatment of choice**
- ▶ Anticoagulation may prevent occlusion of a stenotic vessel and minimise distal embolisation.

Cervical Artery Dissection

- ▶ Carotid
- ▶ Vertebral
- ▶ [Arch Neurosci. 2015 Oct; 2\(4\): e26670.](#)
- ▶ Published online 2015 Oct 17.
doi: [10.5812/archneurosci.26670](https://doi.org/10.5812/archneurosci.26670)
- ▶ **Cervical Artery Dissection: A Review of the Epidemiology, Pathophysiology, Treatment, and Outcome**
- ▶ [Christina A. Blum](#)¹ and [Shadi Yaghi](#)²

Cervical Artery Dissection

- ▶ Cervical artery dissection is a common cause of stroke in young adults, with a prevalence of up to 20% in this population and an annual incidence rate of 2.6 to 2.9 per 100,000^{1, 2}. The true incidence is likely higher because many cases with dissection may go undiagnosed due to minor self-limited clinical symptoms. In population-based studies, the mean age of occurrence is approximately 45 years and there appears to be a slight gender predisposition favoring males (53-57%)^{3, 4}. Furthermore, there seems to be seasonal variation, with dissection more likely to occur in the winter⁵.
- ▶ Cervical artery dissection is classified based on the artery involved (vertebral vs. carotid) and the location of involvement (intracranial vs. extracranial). The most common type is extracranial internal carotid dissection which typically occurs 2-3 cm above the bifurcation and accounts for up to 2.5% of all first strokes.⁶

- ▶ Heritable connective-tissue disorders
- ▶ Ehlers-Danlos syndrome type IV
- ▶ [Fibromuscular dysplasia](#)
- ▶ Cystic medial necrosis
- ▶ [Marfan syndrome](#)
- ▶ Autosomal dominant polycystic kidney disease
- ▶ [Osteogenesis imperfecta type I](#)
- ▶ Oral contraceptives
- ▶ Hypertension
- ▶ Neck manipulation or strain - This can result from intentional manipulation or from other strain that may occur during sports activities, yoga, or even apparently minimal activity (eg, overhead painting)
- ▶ Blunt trauma from high impact and seemingly minor mechanisms of injury
- ▶ Penetrating trauma
- ▶ Wearing a three-point restraint seat belt during a motor vehicle crash
- ▶ Smoking
- ▶ Respiratory tract infection (related to seasonal distribution?)





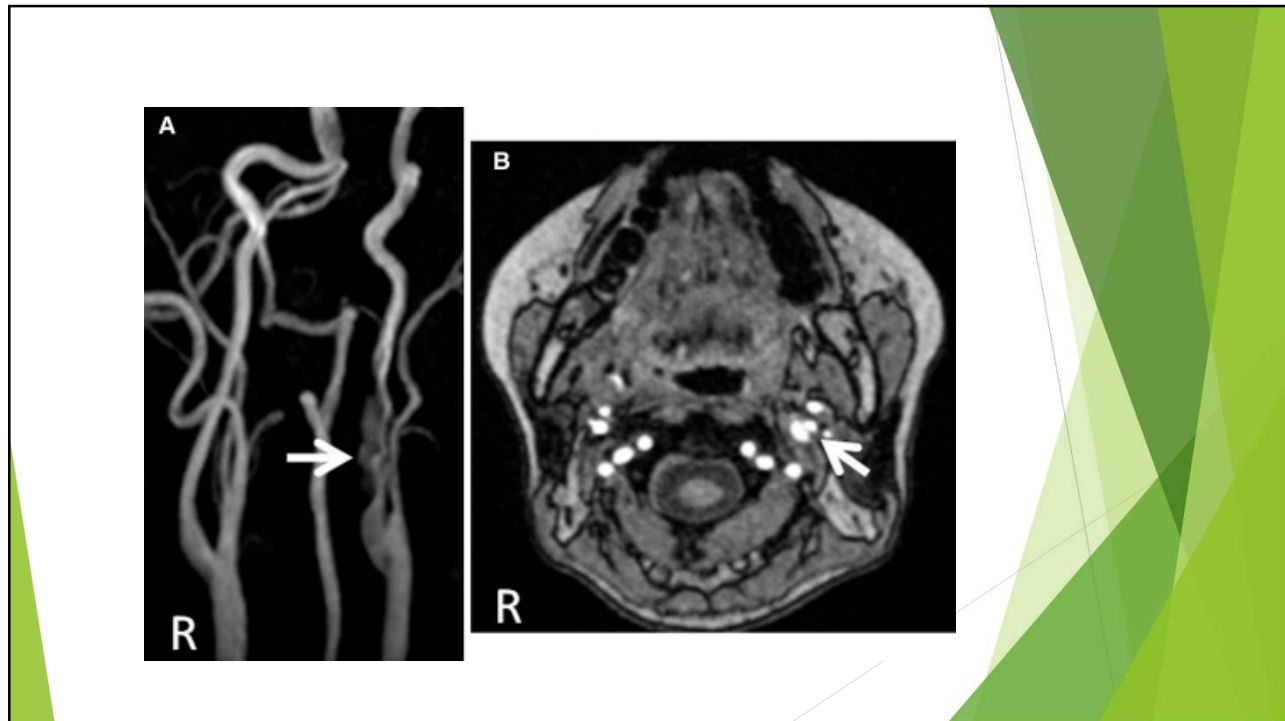
- ▶ The clinical manifestations of dissections depend on the artery involved. Carotid artery dissections typically begin with ipsilateral neck pain or headache and a partial Horner's followed by retinal or cerebral ischemia. The presence of any two of the three elements in the triad strongly suggests the diagnosis of carotid dissection²⁰. The headache caused by dissection has no specific features and can resemble migraine or cluster headaches.¹⁹ Common headache characteristics include sudden onset, unilateral, constant, and throbbing. Other headache characteristics including a thunderclap headache can also be seen. The Horner is usually partial (miosis and ptosis without anhidrosis) due to the fact that sudomotor fibers of the face travel along the external carotid artery. Focal neurological symptoms as a result of cerebral or retinal ischemia may be transient or persistent and are variable.
- ▶ Conversely, vertebral artery dissections typically present with occipito-cervical pain, which may be followed by a variety of posterior circulation ischemic symptoms including vertigo, dysarthria, visual field deficit, ataxia, and diplopia. Although strokes from vertebral artery dissection most frequently involve the lateral medulla and cerebellum, spinal cord infarction may occur when extracranial branches are affected.
- ▶ When intracranial carotid or vertebral dissections are complicated by pseudoaneurysm formation, local symptoms due to compression of adjacent structures can occur. For example, cranial nerves XII, IX and X are closest in proximity to the carotid artery and are those most commonly involved in carotid artery dissection. Furthermore, rupture of a pseudoaneurysm can cause subarachnoid hemorrhage presenting with an acute thunderclap headache and carrying a relatively high mortality and morbidity⁴¹.

Cervical Artery Dissection

- Dissection may be spontaneous or the result of trauma and generally occurs in younger patients (median age, 40 years).
- **Internal Carotid Artery Dissection** may be suspected in a patient with **Unilateral Anterior Neck Pain or Headache**, usually around the eye or frontal area.
- Most patients present with or eventually develop **Neurologic Signs** e.g : TIA, stroke, Horner syndrome, transient monocular blindness, or cranial nerve palsies.
- **Vertebral Artery Dissection** typically presents with marked **Occipital or Posterior Neck Pain** associated with signs of a **brainstem TIA or Stroke**.

▶ Diagnosis

- ▶ Cervical artery dissection is suspected clinically and confirmed by neuroimaging techniques including magnetic resonance imaging (MRI), computerized tomographic angiography (CT), and conventional angiography. The sensitivity and specificity of each test varies based on the location and extent of the dissection. The preferred method of diagnosis is magnetic resonance angiography (MRA) along with a T1 axial cervical MRI with fat saturation technique due its lack of radiation, high sensitivity and specificity, and ability to visualize an intramural hematoma ([Figure 1](#)).²² CTA also has high sensitivity and specificity and may also be used but is associated with radiation exposure and potential technical challenges.^{23, 24} In patients with CAD, a CTA can show the double lumen sign (true and false lumen) ([Figure 1](#)) or a flame-like taper of the lumen. Carotid ultrasound with color doppler is another screening test that can be used.²⁵ Although it is non-invasive, this technique is operator dependent and is of poor diagnostic value in patients with intracranial carotid dissection and those with vertebral dissections. Although ultrasound has been shown to have high sensitivity in patients with extracranial carotid dissection and ischemic symptoms²⁵, in patients with headache and isolated Horner this technique is poor.²⁶ A possible explanation is that in patients with local symptoms only, the dissection rarely causes luminal narrowing and thus may be missed on color doppler.²⁷ Catheter angiogram should not be routinely used to diagnose dissection for several reasons, including inability to visualize intramural hematomas and being an invasive test that may potentially cause an iatrogenic dissection, especially in a patient population with underlying vessel wall weakness.



- ▶ **Pathophysiology of stroke due to CAD**
- ▶ Although the exact mechanism of stroke in patients with CAD is unclear, the most likely mechanism is artery-to-artery embolism of a thrombus or fragments of a thrombus that form in the false lumen. This has been demonstrated by brain imaging showing embolic appearing infarcts in most patients with dissection who suffered from strokes³⁴. Other possible mechanisms include hypo-perfusion causing watershed infarctions if there is severe vessel narrowing, occlusion of the dissected vessel, or, less commonly the intimal flap occluding the ostium of a branch of the dissected vessel. In one study that included 172 patients with CAD out of which 58% had evidence of an acute stroke on diffusion weighted MRI, the mechanism of stroke was found to be thromboembolic in 85% of cases, secondary to hemodynamic failure in 12%, and due to a mixed mechanism in 3% of cases³⁵. In another study, the presence of luminal narrowing was not associated with stroke occurrence; however patients with occlusive dissection had larger infarcts than those with non-occlusive dissections³⁶.

► **Natural history of CAD**

- Patients with CAD tend to have a good prognosis. The main predictor of poor outcome is cerebral infarction, which occurs in about 70% of patients with CAD², however this is based on data available from stroke centers and thus is subject to referral bias. Strokes related to CAD typically occur in the first 2 weeks after the dissection^{37, 38} and the risk of stroke falls dramatically beyond that time point, resembling what is seen with symptomatic carotid stenosis. In population-based cohorts, the risk of recurrent stroke from CAD is less than 3%^{37, 38}. In general, patients with CAD have resolution and healing of the blood vessel on follow-up imaging 6 months after diagnosis ([Figure 3](#))³⁹. Although the dissected vessel usually has complete recanalization, residual stenosis or occlusions may persist². Even in those who do not recanalize, the risk of stroke recurrence remains very low. One of the complications of CAD is the development of a pseudo-aneurysm that tends to persist on repeat imaging⁴⁰. The risk of rupture is around 1%^{3, 4} and typically occurs in intracranial vessels lacking an external elastic lamina, causing subarachnoid hemorrhage with a relatively high mortality rate. Although CAD tends to occur only once, about 7% of patients have recurrent CAD seven years from the diagnosis⁴¹. Patients with dissection who have a stroke tend to have a good long-term outcome with 75% being functionally independent at 3 months¹⁶.

Carotid Dissection

- [Can J Neurol Sci.](#) 2008 May;35(2):146-52.

- Extracranial carotid and vertebral artery dissection: a review.

- [Redekop GJ](#)†.

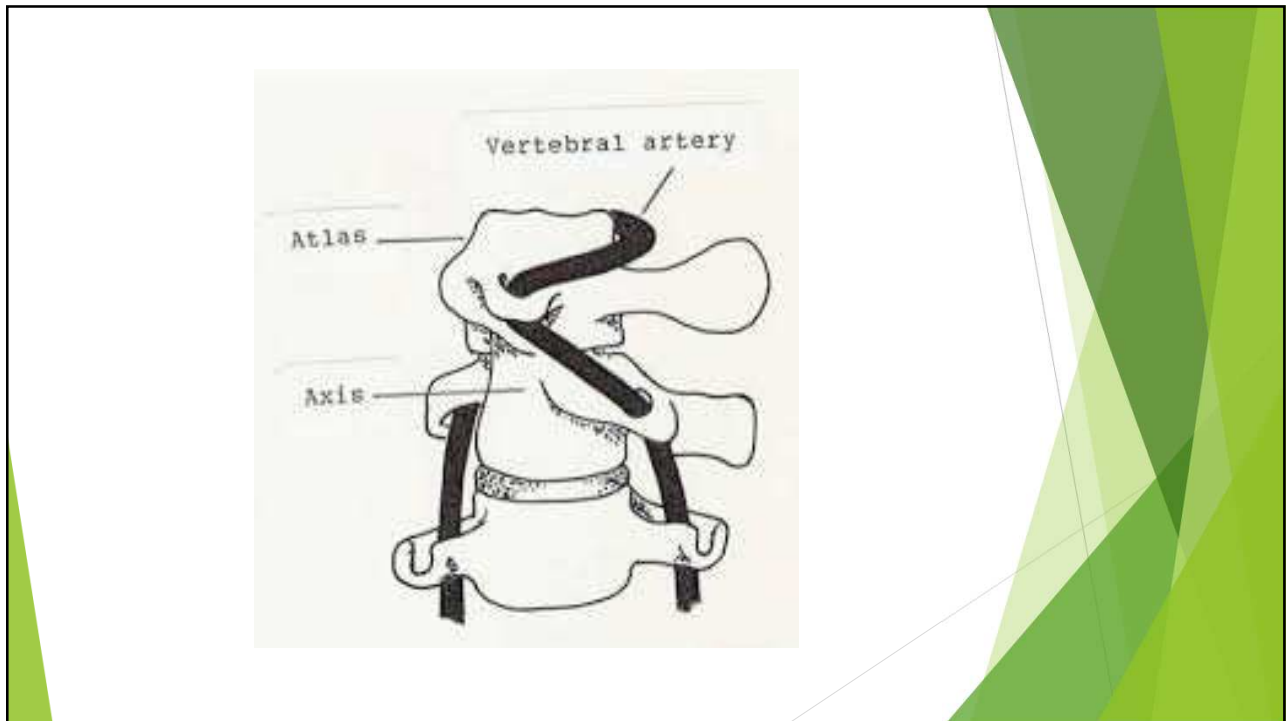
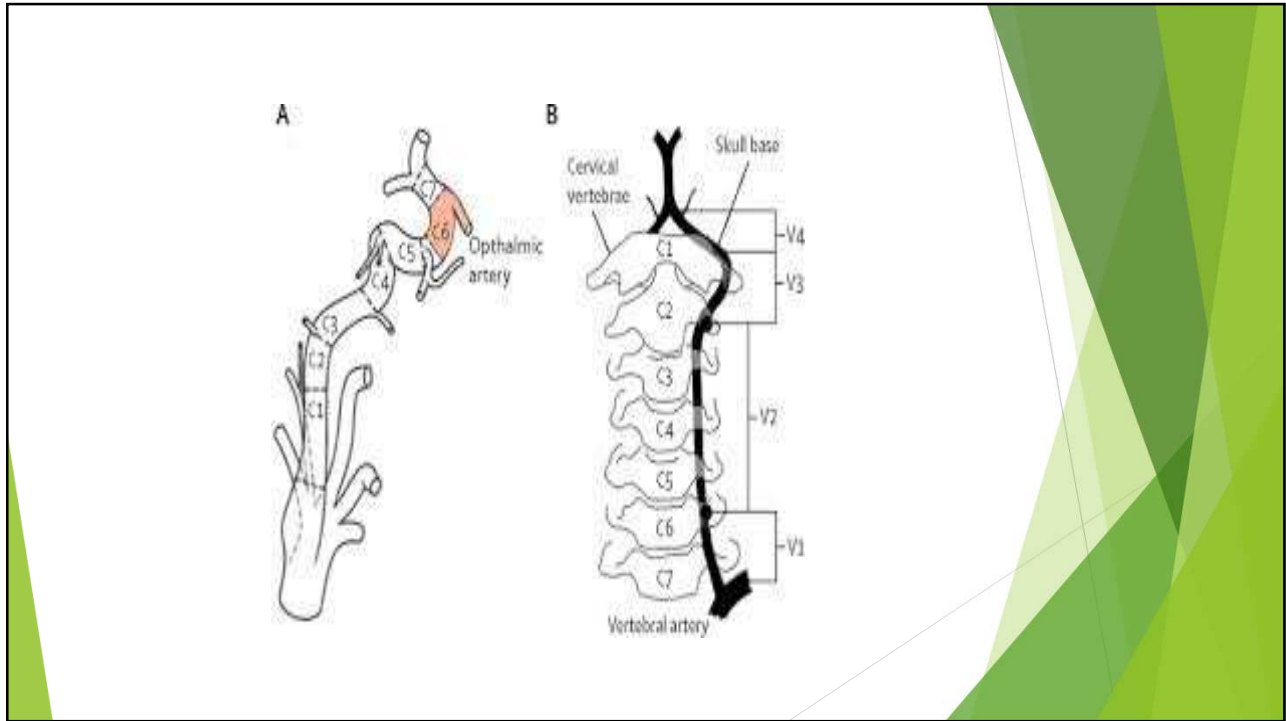
- [Author information](#)

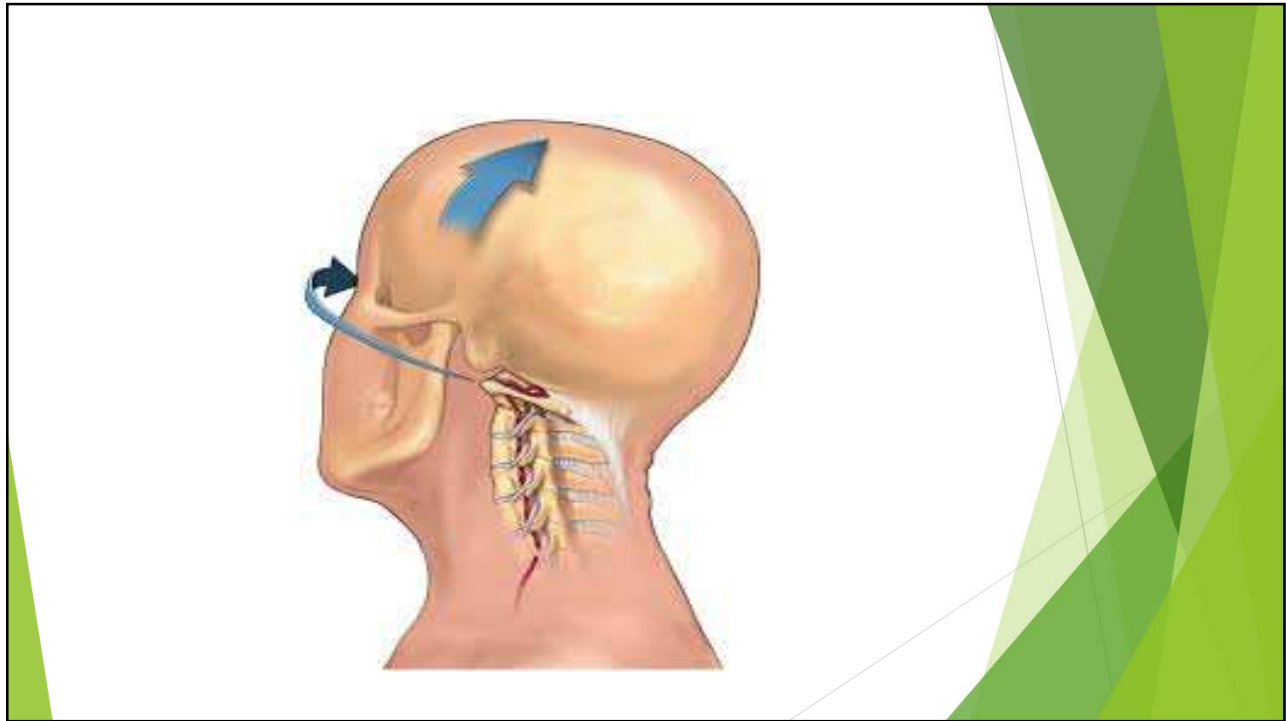
- [Abstract](#)

- Dissection of the extracranial carotid and vertebral arteries is increasingly recognized as a cause of transient ischemic attacks and stroke. The annual incidence of spontaneous carotid artery dissection is 2.5 to 3 per 100,000, while the annual incidence of spontaneous vertebral artery dissection is 1 to 1.5 per 100,000. Traumatic dissection occurs in approximately 1% of all patients with blunt injury mechanisms, and is frequently initially unrecognized. Overall, dissections are estimated to account for only 2% of all ischemic strokes, but they are an important factor in the young, and account for approximately 20% of strokes in patients less than 45 years of age. Arterial dissection can cause ischemic stroke either by thromboemboli forming at the site of injury or as a result of hemodynamic insufficiency due to severe stenosis or occlusion. Available evidence strongly favors embolism as the most common cause. Both anticoagulation and antiplatelet agents have been advocated as treatment methods, but there is limited evidence on which to base these recommendations. A Cochrane review on the topic of antithrombotic drugs for carotid dissection did not identify any randomized trials, and did not find that anticoagulants were superior to antiplatelet agents for the primary outcomes of death and disability. Healing of arterial dissections occurs within three to six months, with resolution of stenosis seen in 90%, and recanalization of occlusions in as many as 50%. Dissecting aneurysms resolve on follow-up imaging in 5-40%, decrease in size in 15-30%, and remain unchanged in 50-65%. Resolution is more common in vertebral dissections than in carotid dissections. Aneurysm enlargement occurs rarely. The uncommon patient presenting with acute hemodynamic insufficiency should be managed with measures to increase cerebral blood flow, and in this setting emergency stent placement to restore cerebral perfusion may be considered, provided that irreversible infarction has not already occurred.

- ▶ [Lancet Neurol](#). 2015 Apr;14(4):361-7. doi: 10.1016/S1474-4422(15)70018-9. Epub 2015 Feb 12.
- ▶ **Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): a randomised trial.**
- ▶ **CADISS trial investigators, Markus HS, Hayter E, Levi C, Feldman A, Venables G, Norris J.**
- ▶ [Collaborators \(78\)](#)
- ▶ [Erratum in](#)
- ▶ [Corrections](#). [Lancet Neurol. 2015]
- ▶ **Abstract**
- ▶ **BACKGROUND:**
- ▶ Extracranial carotid and vertebral artery dissection is an important cause of stroke, especially in young people. In some observational studies it has been associated with a high risk of recurrent stroke. Both antiplatelet drugs and anticoagulant drugs are used to reduce risk of stroke but whether one treatment strategy is more effective than the other is unknown. We compared their efficacy in the Cervical Artery Dissection in Stroke Study (CADISS), with the additional aim of establishing the true risk of recurrent stroke.
- ▶ **METHODS:**
- ▶ We did this randomised trial at hospitals with specialised stroke or neurology services (39 in the UK and seven in Australia). We included patients with extracranial carotid and vertebral dissection with onset of symptoms within the past 7 days. Patients were randomly assigned (1:1) by an automated telephone randomisation service to receive antiplatelet drugs or anticoagulant drugs (specific treatment decided by the local clinician) for 3 months. Patients and clinicians were not masked to allocation, but investigators assessing endpoints were. The primary endpoint was ipsilateral stroke or death in the intention-to-treat population. The trial was registered with EudraCT (2006-002827-18) and ISRN (CT144652527).
- ▶ **FINDINGS:**
- ▶ We enrolled 250 participants (118 carotid, 132 vertebral). Mean time to randomisation was 3·65 days (SD 1·91). The major presenting symptoms were stroke or transient ischaemic attack (n=224) and local symptoms (headache, neck pain, or Horner's syndrome; n=26). 126 participants were assigned to antiplatelet treatment versus 124 to anticoagulant treatment. Overall, four (2%) of 250 patients had stroke recurrence (all ipsilateral). Stroke or death occurred in three (2%) of 126 patients versus one (1%) of 124 (odds ratio [OR] 0·335, 95% CI 0·006-4·233; p=0·63). There were no deaths, but one major bleeding (subarachnoid haemorrhage) in the anticoagulant group. Central review of imaging failed to confirm dissection in 52 patients. Preplanned per-protocol analysis excluding these patients showed stroke or death in three (3%) of 101 patients in the antiplatelet group versus one (1%) of 96 patients in the anticoagulant group (OR 0·346, 95% CI 0·006-4·390; p=0·46).
- ▶ **INTERPRETATION:**
- ▶ We found no difference in efficacy of antiplatelet and anticoagulant drugs at preventing stroke and death in patients with symptomatic carotid and vertebral artery dissection but stroke was rare in both groups, and much rarer than reported in some observational studies. Diagnosis of dissection was not confirmed after review in many cases, suggesting that radiographic criteria are not always correctly applied in routine clinical practice.

- ▶ [J Vasc Surg](#). 2011 Aug;54(2):370-4; discussion 375. doi: 10.1016/j.jvs.2011.02.059. Epub 2011 May 28.
- ▶ **Long-term outcomes of internal carotid artery dissection.**
- ▶ **Rao AS¹, Makaroun MS, Marone LK, Cho JS, Rhee R, Chaer RA.**
- ▶ **OBJECTIVE:**
- ▶ The natural history of acute carotid artery dissection is poorly characterized. The purpose of this study is to report on single institutional long-term outcomes.
- ▶ **METHODS:**
- ▶ A retrospective review of patients treated for acute spontaneous or posttraumatic carotid artery dissection over a 20-year period from August 1989 to July 2009 was performed.
- ▶ **RESULTS:**
- ▶ Twenty-nine patients with a mean age of 47 ± 19.6 years were identified with acute carotid dissection. Six (25%) were related to trauma, while 23 (79%) were spontaneous. Neurologic symptoms included contralateral limb weakness (55%), facial pain (35%), and Horner's syndrome (21%). Eight patients (28%) presented with an acute hemispheric stroke. Diagnostic imaging modalities used included computed tomography angiography (52%), magnetic resonance angiography (41%), and conventional angiography (48%). Twenty percent of patients had complete carotid occlusion and 25% had near occlusion. Most dissections (65%) had intracranial extension, and 35% were limited to the extracranial cervical internal carotid. The majority (96%) of patients were treated conservatively with anticoagulation or antiplatelet therapy or both. One patient underwent stenting for persistent symptoms resulting in complete recovery. There were two deaths, one from unrelated traumatic injuries and the other from unknown causes. Long-term follow-up was available for 20 patients: 14 had complete symptom resolution (70%) and five (25%) had partial clinical symptom resolution. Two patients had initial resolution of symptoms, with subsequent recurrence that was successfully managed conservatively. Follow-up imaging revealed luminal patency in 79% of patients with minimal residual stenosis. Two patients developed a small asymptomatic internal carotid aneurysm that did not require treatment. Mean follow-up was 1133.2 days.
- ▶ **CONCLUSIONS:**
- ▶ Most cervical carotid dissections can safely be conservatively managed, with the majority achieving anatomic and symptomatic resolution, with low rates of recurrence over long-term follow-up.





- ▶ BioMed Research International
- ▶ Volume 2017 (2017), Article ID 3072098, 6 pages

- ▶ Review Article:
- ▶ Treatment of Cervical Artery Dissection: Antithrombotics, Thrombolysis, and Endovascular Therapy
- ▶ Jing Peng,¹ Zunjing Liu,² Chunxia Luo,¹ Lin Chen,¹ Xianhua Hou,¹ Li Xiao,¹ and Zhenhua Zhou¹

- ▶ ¹Department of Neurology of Southwest Hospital, Third Military Medical University, No. 30 Gaotanyan Road, Chongqing 400038, China
- ▶ ²Department of Neurology, China-Japan Friendship Hospital, 2 Yinghua Dongjie, Hepingli, Beijing 100029, China

'lysis in CAD

- ▶ from randomized controlled studies. In a recent meta-analysis on patients receiving intravenous thrombolysis and arterial therapies in the Safe Implementation of Thrombolysis in Stroke International Stroke Thrombolysis Register (SITS-ISTR) as of March 2010, 180 cases of CAD patients with acute ischemic stroke (with an average NIHSS score of 16) were investigated, of whom 67% received intravenous thrombolysis therapy and 33% received arterial thrombolysis therapy; the outcome was that the overall incidence of intracranial hemorrhage, the overall mortality rate, and the proportion of patients with a good prognosis were 3.1%, 8.1%, and 41%, respectively. Compared with stroke cases caused by other etiologies in the SITS-ISTR, the CAD patients receiving thrombolysis therapies showed no significant differences in terms of safety and prognosis [27]. Thus, we believe that the treatment of CAD-induced acute ischemic stroke using intravenous rtPA within 4.5 h of onset is safe. However, we should strive to develop new therapeutic strategies to lower the mortality and disability rates of CAD patients after thrombolytic therapy [25].

- ▶ Endovascular treatment has been widely used to treat cardiovascular and cerebrovascular diseases [28]. However, randomized controlled studies on the application of endovascular treatment or surgeries for CAD patients have not been reported to date [29, 30], and the efficacy and safety of endovascular therapy or surgical treatment have not been evaluated in CAD patients. Endovascular treatment has been primarily used in CAD patients with failed antithrombotic treatment with contraindications for anticoagulation and a pseudoaneurysm and when stent implantation is the main vascular interventional procedure. Due to the special pathological physiology of cervical artery dissection, the method of endovascular treatment is cervical artery stenting. Endovascular treatment/surgical treatment for CAD should be limited because CAD patients have a lower risk of recurrent ischemic stroke, there is no significant correlation with CAD-induced vascular stenosis and pseudoaneurysm, and endovascular/surgical treatments are traumatic. With the development of vascular interventional procedures, the application of endovascular treatment in CAD patients may be underestimated; furthermore, it was previously believed that the dissection leads to clinical events mainly through thromboembolism rather than hypoperfusion; thus, antithrombotic therapy has been the preferred treatment for CAD [31]. However, endovascular treatment can also be viewed as the preferred option for the treatment of CAD patients, especially when the patient has both an embolism and obvious hypoperfusion [32]. In this case, endovascular treatment can effectively relieve stenosis, increase blood flow, and improve low perfusion. In a retrospective study, 140 cases of CAD patients received stenting, and angiographic follow-up was conducted for an average of 12.8 months. The results showed that dissection-induced vascular stenosis was significantly improved and that secondary stroke events accounted for only 1.4% of cases. Thus, endovascular therapy could effectively improve CAD-induced vascular stenosis and reduce the incidence of ischemic stroke [33]. Multiple overlapping stents could also effectively reduce the blood flow velocity in pseudoaneurysms and promote thrombosis, thereby shrinking the pseudoaneurysm or causing it to disappear. Previous studies showed that dissection stenosis of CAD patients undergoing stenting therapy could be largely eased, from 71% to complete remission [29]. In terms of the progression of CAD and the structural damage to the vessel wall, patients in the acute stage and Borgess type IB and II patients would significantly benefit from the use of stenting as the preferred treatment [23].