Acute craniocervical artery dissection: What the on call radiologist needs to know

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Learning objectives

To review the epidemiology and typical clinical presentation of carotid and vertebral artery dissection

Present imaging findings in acute craniocervical artery dissection on plain CT and CT angiography.

Discuss treatment and pitfalls in diagnosis
Background

Epidemiology

The incidence of carotid artery dissection is 2.5 - 3 per 100000, and incidence of vertebral artery dissection is 1 - 1.5 per 100000. They account for about 2% of all ischaemic strokes but have a much higher prevalence in young and middle-aged patients accounting for between 10 and 25% of strokes. They occur all ages but appear to peak in the fifth decade.

Pathophysiology

Vertebral and carotid arteries are medium-sized arteries containing 3 layers. The intima (inner), media (middle) and adventitia (outer) layers. Arterial dissection arises as a tear in the intimal layer allowing blood under arterial pressure into the media, forming intramural haematoma. These haematomas widen the arterial wall and enlarge the overall vessel diameter. Haematoma pointing externally to the adventitia form a dissecting aneurysm, those pointing internally to the intima cause vessel stenosis or occlusion.

Clinical presentation

The classic triad in carotid artery dissection is, unilateral pain (in the neck, face and head), partial Horner's syndrome (meiosis and ptosis) followed hours or days later by cerebral or retinal ischaemia. The triad is however found in less than a third of the patient and presence of 2 of the 3 symptoms should suggest the diagnosis. Pain, usually fronto-parietal in two thirds of patients, occurs a median of 4 days before other symptoms. The lower cranial nerves, particularly hypoglossal nerve (taste) are affected in 10% of patients. Pulsatile tinnitus has been reported in 25% of patients.

Vertebral artery dissection presents more insidiously with pain at the back of the head or the neck almost always involving the occipital area. This can initially be mistaken for musculoskeletal pain or migraine. It is followed in 90% of patients by signs suggesting ischemia of the posterior circulation involving the brainstem (particularly lateral medulla) thalamus and cerebellum hemispheres.

Traumatic dissection following penetrating or blunt trauma (automobile accident, stabbing, accidental hanging) is rare. Spontaneous dissection is more common although on detailed enquiry, history of minor precipitating trauma can usually be elucidated. The event usually involves hyperextension or rotation of the neck and varies from
practising yoga or painting a ceiling to coughing and sneezing. Chiropractic manipulation is associated with carotid and in particular vertebral artery dissection. It is estimated that 1 in 20,000 spinal manipulations results in a stroke. Recent respiratory tract infection is also associated with spontaneous dissection although the cause is uncertain.

There is a genetic component where patient may have an underlying structural defect in the arterial wall. Ehlers-Danlos, Marfan's, autosomal dominant polycystic kidney disease and osteogenesis imperfecta are associated with increased risk. Fibromuscular dysplasia (FMD) and cystic medial necrosis, non-specific conditions associated with many systemic disorders are also associated with increased risk. Angiographic findings of FMD are found in about 15% of patients with spontaneous dissection.

**Treatment**

90% of cerebral infarcts due to dissection are caused by embolic phenomena rather than insufficient blood flow. To this end, the aim of treatment is to prevent thromboembolic complications. Anticoagulation with heparin followed by warfarin is recommended for between 3 and 6 month. Intracranial dissections and the dissecting aneurysms are harder to treat as there is a risk of subarachnoid haemorrhage. Although there may be a role for anticoagulation where there is cerebral ischaemia but no subarachnoid haemorrhage, no consensus has been reached.

Most dissections will heal spontaneously, usually recanalising within the first 3 months. Imaging performed at this time is useful to reassess the vessel. Patients with persistent symptoms of cerebral ischaemia despite adequate anticoagulation may need surgical treatment. This is complex with high morbidity and mortality involving ligation and bypass of the affected artery. Endovascular treatment with balloon angioplasty or metallic stents has a lower risk although long-term results are awaited.
Imaging findings OR Procedure details

The extracranial segments of carotid and vertebral arteries are more mobile and more prone to damage from surrounding bony structures and as a result are more prone to the dissection. Up to 60% of internal carotid dissections are located at the C1 -- 2 level (figure 1), starting around 2 cm above the bifurcation and do not extend into the petrous portion which is protected by bone. Around two thirds of vertebral artery dissections are also located at the C1 -- 2 levels (figure 1). Of these, around half occur at the V2 level and half at the atlas loop (V3). Multiple vessel dissection is found in upto 28% of patients.

Imaging findings on plain CT

The classic target sign was initially described on post-contrast CT scan where a narrow contrast filled lumen is surrounded by low attenuation intramural haematoma and thin annular external enhancement (thought to be enhancement in the vaso-varum). This is rarely seen now as post contrast heads has been replaced with CT angiogram as the diagnostic scan of choice. It is however seen incidentally and on post contrast MRI scans (figure 2) and should still be recognised.

Direct signs on plain CT head (figure 3, 4, 5) include enlarged vessel diameter wall and hyper attenuating crescent shaped clot in the wall representing intramural haematoma.

Indirect signs on plain CT (figure 2, 3, 6, 7, 8) head are the sequelae of dissection namely, ischaemic phenomena in the distribution of the affected vessel. These present as embolic infarcts, large vessel infarcts and water-shed ischaemia (probably secondary to hypovolaemia rather than emboli). In the vertebral artery dissection involving the intradural segment (V4) subarachnoid haemorrhage may be seen.

On a plain CT head, indirect signs are easier to pick up. When found the imaged craniocervical arteries need to be scrutinised with a high index of suspicion. In the right clinical setting, there should be a low threshold for performing CT angiogram.

Imaging findings on the CT angiogram (CTA)

The vessels and the walls must be carefully examined using multiplanar reformatted images. On axial angiogram, an enlarged vessel diameter with a narrowed, extrinsic lumen is seen (figure 3, 4, 5, 8, 9, 10, 11). On reformatted images, tapering of the vessel lumen to thin string of contrast (string sign) is seen (figures 5, 6, 8, 9, 10, 11). Complete occlusion (figure 6) must be sought. Arterial wall thickening can be seen more clearly
using soft tissue windows (width = 400HU, level 40HU), keeping in mind that it cannot be used to differentiate atherosclerotic thrombus from intramural haematoma (figure 8, 10, 11). Other signs to pick up on CTA are an intimal flap (figure 7, 11, 12) or a dissecting aneurysm (figure 7, 12).

**Pitfalls**

Pitfalls in diagnosis include bone or dental artefacts which can mimic intimal flap. Complete atherosclerotic occlusion (figure 13, 14) can mimic dissection but they usually have a more abrupt cut-off, the affected vessel has a normal calibre, and they occur in older patients. They also occur at different portions of the vessel, namely at the bulb (rather than distal it) in the carotid artery and at the osteum or V1 and V2 segments in the vertebral artery. Surrounding atherosclerotic calcification and evidence of atherosclerotic disease in the other vessels should also be sought. Dysgenesis of the ICA can mimic occlusion or stenosis however hypoplasia or absence of the carotid canal suggests the diagnosis.
Fig. 0: Figure 1: (a) Sagittal MIP CTA of normal left carotid artery. Internal carotid dissections are usually confined to the region above the carotid bulb (arrow) and below the petrous bone (arrowhead). (b) 3D volume rendered CTA of normal left vertebral. Vertebral artery segments are: V1 (from origin o entry into transverse foramen), V2 (within transverse foramen from C6 to C2), V3 (atlas loop region) and V4 (intracranial segment, not shown).

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**Fig. 0:** Figure 2: 51 year old presenting with neck pain, left sided clumsiness and history of head injury 2 days previously. (a) initial Plain CT showed subtle left cerebella low attenuation. Patient deteriorated and MRI was performed the next day. (b) T1 MRI shows the classic target sign of dissection in both vertebral arteries (arrows). Initially described in post contrast CT but also seen in post contrast MRI, it shows a narrow eccentric contrast filled lumen surrounded by mural thickening and thin annular enhancement.

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**Fig. 0:** Figure 3: 48 year old patient presenting acutely with signs of brainstem infarct. No previous trauma. (a) Plain CT scan showing low attenuation and swelling in the pons in keeping with brain stem infarct. (b) Axial T2 shows brain stem infarct more clearly. (c, d, e) showing vertebral artery dissection in right V3 segment. (c) Plain scan shows high attenuation intramural haematoma in an enlarged right vertebral artery (V3 segment). Compare with low attenuation in the normal left V3 segment (arrowhead). (d) CTA shows high grade stenosis with contrast flowing through a tiny eccentric lumen. (e) Axial T2 images showing high signal clot intracranial (V4) segment of right vertebral (arrow) and normal flow void in the right vertebral (arrowhead).

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**Fig. 0:** Figure 4: 36 year old with spontaneous left sided Horner's 2 days earlier. (a) Plain CT shows an enlarged distal left ICA just inferior to the petrous bone with a suggestion of high attenuation haematoma in the wall. (b) Coronal CTA shows no luminal narrowing but shows swollen distal left ICA (arrow) with wall thickening. Compare with normal right ICA.

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Fig. 0: Figure 5: 53 year old with a history of road traffic accident 1 month previously, presenting with right Horner’s syndrome and right sided neck pain. CT angiogram showed right ICA and right vertebral artery dissection. (a) Plain CT showing high attenuation intramural haematoma in the wall of the right V3 segment (arrow). Note normal V3 on the left. (b) CTA shows narrow lumen of contrast flowing in a thickened V3 segment (arrow). (c) Axial T2 weighted MRI showing small acute right cerebella tonsil infarct, not seen on CT. (d) Axial angiogram showing narrow lumen of contrast in a thick-walled (intramural haematoma) right ICA. (e) Sagittal angiogram showing long tapered stenosis (string sign).

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Fig. 0: Figure 6: 23 year old female who stopped speaking. (a) Plain CT shows low attenuation infarct in the left MCA territory (arrow). Sagittal CTA shows narrowing of left ICA (arrow) with complete occlusion distally. Contrast superiorly is in the left internal jugular (arrowhead). (c) CTA 6 weeks later shows narrow recanalisation of the left ICA.

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Fig. 0: Figure 7: 59 year old. Previous stroke and catheter angiogram while abroad with post procedure left ICA dissecting aneurysm. (a) Plain scan shows left watershed territory infarct. (b and c) Axial and sagittal CT angiogram shows dissecting left ICA aneurysm (arrows) with dissection flap (arrowheads). (d) Catheter angiogram showing dissecting left ICA aneurysm.

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Fig. 0: Figure 8: 48 year old, road traffic accident 3 weeks previously, presenting with right sided neglect and right Horner’s syndrome. (a) Plain CT shows acute infarct in the right frontal opperculum. (b) CT angiogram viewed on soft tissue windows (400HU width, 40HU level) more clearly shows intramural haematoma (arrow) in the wall of the distal common carotid. (c) Saggital CTA shows extension of dissection into the bulb of the right ICA (arrow).

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Fig. 0: Figure 9: 32 year old with dissection post catheter angiogram. (a) Direct catheter angiogram was performed showing mild dilatation of the left ICA. (b and c) CT angiogram done immediately after catheter angiogram. (b) Axial and (b) saggital images showing
dissection with crescent of high attenuation contrast from catheter angiogram in the wall of the left ICA. The lumen of the ICA is patent.

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**Fig. 0:** Figure 10: 53 year old involved in minor road accident 1 month prior to presenting with right sided neck pain and Horner's syndrome. (a) Axial CTA on soft tissue windows (W 400HU, L 40HU) showing enlargement and wall thickening (intramural haematoma) of right ICA (arrow) just inferior to the petrous bone. (b) Sagittal CTA shows gradual narrowing (arrow) of the distal right ICA (string sign). Wall thickening is again seen.

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Fig. 0: Figure 11: 58 year old with 10 day history of headache and right Horner's syndrome. (a) Axial CTA showing Right ICA swelling with luminal narrowing in keeping with dissection. (b) Axial CTA, soft tissue windows (w 400HU, L 40HU) shows the right ICA thickening more clearly. Note the thin, compressed left internal jugular (arrowhead). (c) Sagittal CTA showing irregular narrowing of the right ICA extending from the carotid bulb to the petrous bone. MRI showed normal petrous and cavernous ICA. Dissection flap is seen (arrow).

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Fig. 0: Figure 12: 31 year old. Tiny right ICA dissecting aneurysm post catheter angiogram for known ophthalmic artery aneurysm. (a and b) sagittal and axial CTA showing the tiny dissection (arrow). (c and d) Sagittal and axial TOF MRI showing the dissection with intimal flap shown of axial image (d).

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Fig. 0: Figure 13: (a) Sagittal CTA showing abrupt cut-off of an atherosclerotic left ICA with speck of calcification (arrow). (b) axial CTA on soft tissue windows (W 400HU, L 40HU) showing speck of calcification (arrow) in completely occluded but relatively normal calibre left ICA (arrowhead).

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Fig. 0: Figure 14: Atherosclerotic occlusion in the distal left brachiocephalic artery (arrow) Note the relatively abrupt cut off at site of occlusion (arrow head) and atherosclerotic calcification (arrow) at the origin of the vessel differentiating it from dissection.

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Conclusion

While the diagnosis of craniocervical artery dissection can be challenging, it is an important diagnosis to make as it affects young to middle-aged patient. Plain CT and CT angiography is usually initial imaging modality. Radiologist need to be aware of findings on plain scan which suggest dissection and have a low threshold for performing CT angiography.
References


