# Risk factors, radiological features, and infarct topography of craniocervical arterial dissection

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***Background*** **Craniocervical arterial dissection is a common cause of ischemic stroke in the young to middle-aged population. There have been a number of previous studies where radiological features have been described but few with detailed mapping of infarct topography and none where these features have been related to the reported risk factors.**
***Aims*** **The aims of this study were to describe the radiological characteristics of dissection patients ≤55 years and relate these to reported risk factors.**
***Methods*** **Craniocervical arterial dissection cases ≤55 years, and age- and gender-matched controls were identified from a medical records database between 1998 and 2009. Control cases had stroke from another cause than dissection. Records and radiology were reviewed.**
***Results*** **Thirty-six radiologically confirmed dissection cases [20 (56%) vertebral artery, 16 (44%) internal carotid], and 43 controls were identified. Dissections were extracranial with intracranial extension in 10 (28%) cases. Infarction was demonstrated in 22 (61%) dissection cases. The most common wall deficit identified was an intimal flap. Twenty-three (64%) dissection cases had a recent history of neck trauma ($P > 0{\cdot}000$) and 13 (36%) had vascular variants ($P = 0{\cdot}013$).**
***Conclusion*** **Craniocervical arterial dissection cases, particularly vertebral artery, were more likely to have a history of neck trauma. Dissections were most commonly extracranial, in the upper cervical region, with intracranial extension in 28%. Dissection cases with trauma more commonly had a dissection flap and evidence of infarction in the lateral medulla, anterior or posterior inferior cerebellar artery territory. Close inspection of the V3 segment of the vertebral or skull base for internal carotid artery may be warranted with a history of neck trauma.**

Key words: cerebral infarction, ischemic stroke, MRI, radiology, risk factors, stenosis

## Introduction

Craniocervical arterial dissection (CAD), vertebral artery dissection (VAD) or internal carotid artery dissection (ICAD), is one of the most common causes of ischemic stroke in the young to middle-aged population (1,2), accounting for 10–25% of all ischemic stroke in this age group. It is considered to often occur spontaneously, that is without obvious cause, and has an annual incidence of around 2·5–3:100 000 (3,4).

The diagnosis of CAD is generally based on suggestive history, clinical findings, and radiological imaging (5), usually computed tomography (CT) or magnetic resonance (MR). There is a need for better identification of this condition in young people as recognition is sometimes difficult. It has been proposed that certain risk factors may be related to CAD, in particular minor neck trauma, including neck manipulation, recent infection, vascular anomaly, or connective tissue disease such as fibromuscular dysplasia (2,3,6).

Previous research has generally described case series of patients with CAD from hospitals. There have been a number of recent studies of the radiological features and risk factors for CAD (7); however, these are often limited by the lack of comparison with a control group and the inclusion of older patients in whom cardiovascular risk factors for stroke were more likely to be present (8–11). There have been a number of studies detailing methods for mapping infarct patterns for internal carotid and middle cerebral artery stroke (12–16), but few studies for posterior circulation stroke where detailed description or mapping of resultant infarct topography have been performed (17,18). There are also relatively few studies mapping infarct topography specifically in CAD (12,14,17,19). There have been none where arterial wal l findings have been related to reported risk factors. Studies have often focused more on comparison of MR, CT, and conventional angiography (5) in the natural history of CAD.

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## Aims

The aims of the current study were to identify patients ≤55 years with radiologically confirmed CAD presenting to hospital in the Hunter and Manning regions of New South Wales, Australia, to describe the site and nature of the dissection, topography of any associated infarction, and to identify any association between previously reported risk factors and the site and radiological characteristics of the dissection. The specific research questions were:

- What are the radiological features resulting from CAD?
- What is the infarct topography resulting from CAD?
- What are the relationships between arterial wall deficits and risk factors for CAD?
- What is the relationship between infarct topography and risk factors for CAD?

## Methods

### Design

The study was a retrospective case control study. The methods are outlined in a flow chart (see Fig. 1). Cases of VAD or ICAD were identified via the medical record coding system of the John Hunter Hospital between 1998 and 2009. This hospital is a large tertiary referral hospital of 550 beds servicing the Hunter and Manning regions with a population of approximately 617 000 (20). The medical records database was searched using the international diagnostic coding system international classification of diseases (ICD-10) (21) for patients with radiologically confirmed or suspected VAD or ICAD.

The following ICD-10 categories, used:

- 1670 dissection of cerebral arteries, were nonruptured
- 1630–1639 cerebral infarctions, unspecified
- 1640 stroke not specified as hemorrhage or infection
- 1660–1669 occlusion and stenosis of unspecified cerebral artery and
- 1650–1659 occlusion and stenosis of carotid artery.

Although some of these codes do not directly identify VAD or ICAD, in case of possible miscoding, it was expected that dissection cases would at least be coded under one of these categories. An additional search was also made of the hospital electronic discharge referral system using keywords of vertebral or internal carotid artery dissection.

### Participants

Inclusion criteria for cases were patients ≤55 years with a discharge diagnosis of radiologically confirmed VAD or ICAD. The upper age was chosen as CAD occurs most commonly in the fourth and fifth decades before more traditional cardiovascular risk factors are likely to have developed.

Patients were excluded if they had sub-arachnoid hemorrhage (SAH) or dissection from iatrogenic causes. Controls were age- and gender-matched patients randomly selected from the hospital database with a clinico-radiological diagnosis of ischemic stroke without dissection.

### Measurement of risk factors

Medical records for cases and controls were retrieved (see Fig. 1), and data were extracted using a standardized data extraction proforma. Preexisting medical status was determined by detailed review of records to identify risk factors including any history of preceding events, in particular minor mechanical trauma to the head or neck, or recent infection or febrile illness. Mechanical trauma was defined based on the work of Dittrich *et al.* (2) as report of any heavy lifting, direct or indirect trauma to the head or neck, jerky or abrupt movements of the head, sporting activities, or manual treatment of the neck in the preceding four-weeks. Recent infection was defined as a positive history of upper respiratory tract infection, or urinary tract infection diagnosed by a doctor or requiring antibiotic medication. Imaging was inspected for any evidence of vascular variant or anomaly of the posterior or anterior cerebral circulation such as aplasty of a vessel or a vertebral artery ending in posterior inferior cerebellar artery, or evidence of fibromuscular dysplasia.

### Measurement of radiological features of CAD

In accordance with the local stroke management protocol, all cases with clinically suspected CAD underwent both extracranial and intracranial vascular imaging with either computed tomography angiography (CTA) or magnetic resonance angiography (MRA). All cases with suspected stroke related to CAD underwent brain magnetic resonance imaging (MRI). Imaging was performed on a 1·5 T MRI system (Magnetom Vision; Siemens, Erlangen, Germany). Brain and vascular imaging was reviewed using a standardized proforma. Imaging was evaluated online by two experienced stroke neurologists (CL and MP) blinded to the case or control status. Radiological features and infarct topography were recorded on a standardized proforma. The imaging diagnosis of confirmed CAD was made by consensus. The location of dissection was identified, and the following radiologic criteria were applied based on recent reviews (5,22,23):

- the presence of a crescent sign, a cresentic rim of hyperintense signal seen on CT or T1 or T2 weighted MR images;
- an increase in the external diameter of the vertebral artery (VA) or ICA due to a thickening of the wall or narrowing of the lumen;
- a long tapering stenosis shown by a 'string sign' characterized by a long segment of narrowing of the artery;
- a 'pearl and string sign' where the narrowing included one or more areas of dilation;

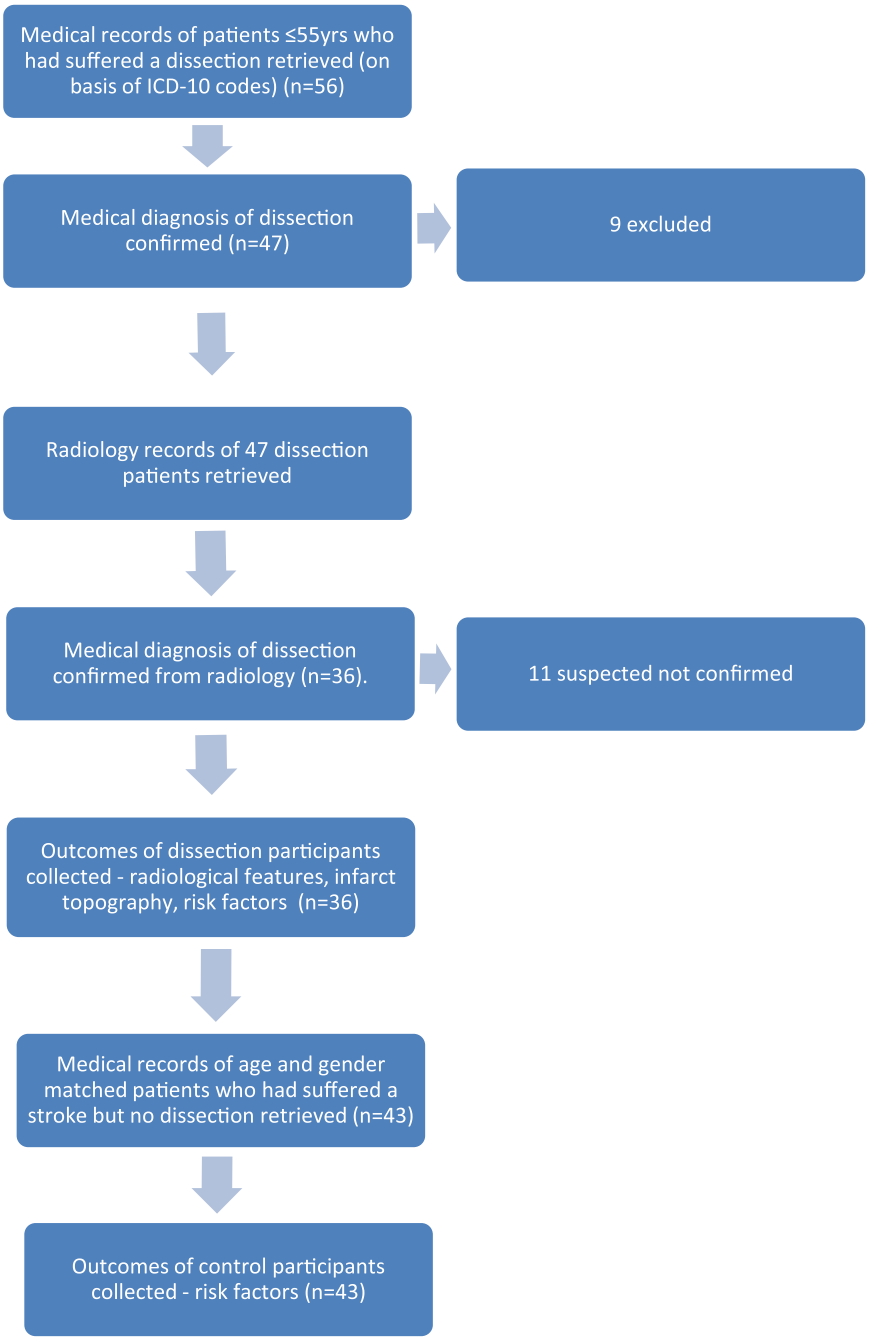


**Fig. 1** Flow chart of study methods for identifying dissection cases.

- an intimal flap or double lumen as demonstrated by a crescent sign;
- an intramural thrombus as evidenced by a thickening of the arterial wall; and
- a pseudoaneurysm or dissecting aneurysm, where the dissection had extended into the adventitial layer.

Stenosis was defined as a reduction in the lumen of the artery and graded as <30%, 30–70%, >70%, or occlusion >2 cm.

## Measurement of infarct topography

Infarction was defined in the acute phase of stroke as high signal on diffusion weighted imaging ($B = 1000$). In the subacute phase, it was defined as high-signal intensity on fluid attenuated inversion recovery (FLAIR) T2-weighted imaging. Location of the infarct was defined according to anatomical location and vascular territory involved, according to

**Table 1** Characteristics of dissection and control groups

| Case characteristics | Dissection (CAD) $n = 36$ | Control (ischemic stroke) $n = 43$ | *P*-value |
|---|---|---|---|
| Vertebral artery territory | 20 (56%) | 5 (12%) | <0·0001 |
| Internal carotid artery territory | 16 (44%) | 38 (88%) | <0·0001 |
| Age (year), mean (SD) | 36·9 (10) | 42·6 (7·3) | 0·42 |
| Males | 20 (56%) | 22 (51%) | 0·7 |

CAD, craniocervical arterial dissection; SD, standard deviation.

**Table 2** Risk factors identified in dissection and control cases

| Risk factor | VAD $n = 20$ (%) | ICAD $n = 16$ (%) | Total CAD $n = 36$ (%) | Control $n = 43$ (%) | Odds ratio (95% CI) | Adjusted odds ratio* (95% CI) | *P*-value |
|---|---|---|---|---|---|---|---|
| Recent head or neck trauma | 13 (65) | 10 (63) | 23 (64) | 3 (7) | 26·67 (6·83,104·17) | 25·29 (6·04, 105·82) | <0·000 |
| Recent manual therapy | 6 (30) | 1 (6) | 7 (19) | 1 (2) | 12·0 (1·42, 101·30) | 12·67 (1·43, 112·0) | 0·022 |
| Recent infection | 4 (20) | 4 (25) | 8 (22) | 4 (9) | 1·94 (0·62, 6·08) | 1·90 (0·58, 6·22) | 0·287 |
| Vascular variant/anomaly† | 9 (45) | 4 (25) | 13 (36) | 7 (16)(allVA) | 3·27 (1·14, 9·36) | 4·46 (1·37, 14·6) | 0·013 |
| Fibromuscular dysplasia | 1 (5) | 1 (6) | 2 (6) | 0 | N/A | N/A | N/A |

*Multiple regression analysis with odds ratios adjusted for age and gender.
†Anomalies involving the posterior circulation such as ectatic or hypoplastic VA or VA ending in posterior inferior cerebellar artery.
VAD, vertebral artery dissection; ICAD, internal carotid artery dissection; CAD, craniocervical arterial dissection; CI, confidence interval; VA, vertebral artery.

established vascular maps (24). Topography of the infarct was mapped onto a schematic representation of the axial slices of the brain to show the frequency of infarct in the different locations.

### Statistical analysis

Descriptive statistics were used to summarize demographic data, radiological features, and risk factors. Simple logistic regression was performed for the major risk factors identified. All factors with a *P*-value of ≤0·2 were included in a multiple logistic regression model; outcomes were expressed as odds ratios with 95% confidence intervals (CIs). Statistical analysis was performed using STATA statistical/data analysis software (version 11, Statacorp, College Station, TX, USA).

The study was approved by the local ethics committee.

## Results

### Characteristics of participants

Thirty-six CAD participants and 43 controls entered the study. Eleven suspected dissections were excluded because of lack of radiological confirmation of their diagnosis. The demographic characteristics are reported in Table 1. In the CAD group, the mean age was 36·9 years [standard deviation (SD) 10], and 20 (56%) were male. In the control group, mean age was 42·6 years (SD 7·3), and 22 (51%) were male. The clinical features of the participants have been described in detail elsewhere (6). VAD participants most commonly presented with headache, neck pain, and unsteadiness or ataxia, whereas ICAD participants typically presented with headache, facial palsy and ptosis, or limb weakness. Control participants were more likely to present with limb weakness and paresthesia, dysphasia or dysarthria, and facial palsy. The majority (72%) of all participants had a favorable outcome, i.e. modified Rankin Scale (mRS) ≤2. An mRS of 2 means a patient is able to walk and manage their own affairs (25).

### Risk factor analysis

The risk factors of the CAD and control cases are shown in Table 2.

#### Mechanical trauma

Thirteen (65%) of the cases with VAD and 10 (63%) with ICAD had a history of minor mechanical trauma within the preceding three-weeks; of these, seven (30%) cases (six VAD, one ICAD) reported recent manual treatment of their neck by a chiropractor, physiotherapist, or masseur. In contrast, only three (7%) control cases had a history of recent mechanical trauma to the head or neck, and only one of these involved manual treatment of the cervical spine. This equated to an odds ratio of 26·67 (95% CI 6·83–104·17), which remained significant in the adjusted model (adjusted for age and

gender). Eleven (85%) VAD and five (50%) ICAD cases with a history of trauma also had a cerebral infarct and all were extracranial.

### Recent infection

Eight (22%) of the dissection cases (four VAD, four ICAD) had a recorded history of recent infection or viral illness prior to their admission to hospital, compared with just four (9%) of the control cases, adjusted odds ratio 1·94 (95% CI 0·62–6·01).

### Vascular variants and anomalies

Thirteen (36%) of the dissection cases had radiological evidence of vessel anomalies mainly involving the posterior circulation, such as an ectatic or hypoplastic vertebral artery, or a vertebral artery ending in the posterior inferior cerebellar artery. One ICAD case had an anterior circulation variant with an absent anterior cerebral artery. On the other hand, only seven (16%) control cases had a report of vessel abnormalities; these were all posterior circulation. The adjusted odds ratio was 4·46 (95% CI 1·37, 14·6, $P = 0·13$). Two CAD cases (6%) had evidence of fibromuscular dysplasia. Ten (43%) of the 23 dissection cases with a history of trauma also had vascular anomaly.

## Radiological characteristics

Of the 36 CAD cases, dissection was identified in the extracranial vertebral artery (VAD) in 20 (56%) cases and in the extracranial internal carotid artery (ICAD) in 16 (44%) cases. Of the VADs, 12 were of the left vertebral artery and eight of the right artery, and in six cases, the dissection extended intracranially. Of the ICADs, three were left sided, 12 were right sided, and one was bilateral. In four cases, the dissection extended intracranially. In the control group, 38 (88%) cases had sustained a stroke in the anterior cerebral circulation (i.e. carotid territory) and five (12%) cases had suffered a posterior circulation stroke (i.e. vertebrobasilar territory).

The majority 29 (62%) of CAD cases had both CT and MR imaging. Twenty VAD and 15 ICAD cases were confirmed on MRI/MRA, with one ICAD confirmed on CTA. Interestingly, five VADs had normal findings on CT/CTA but positive signs for dissection on MR.

### Location of dissections

In the VA, the majority of dissections (14, 70%) occurred at the atlas loop or skull base (V3 segment) (see Table 3). Six (30%) VADs extended intracranially (V4 segment) to involve the basilar artery. One case had two sites of dissection (C2 and C5). The majority of ICADs (nine, 56%) occurred at the skull base or C2 level (cervical segment). Four (25%) of these extended intracranially within the skull base, one leading to SAH. Interestingly, of the 13 VAD cases with a history of trauma, 10 (77%) were at the atlas loop, only two (15%) extended intracranially.

**Table 3** Location of dissection in dissection cases

| Location of dissection | VAD *n* = 20 (%) | ICAD *n* = 16 (%) | Total CAD *n* = 36 (%) |
|---|---|---|---|
| Extracranial | 20 (100) | 16 (100) | 36 (100) |
| Atlas loop/C2 | 12 (60) | 4 (25) | 16 (44) |
| Skull base | 2 (10) | 5 (31) | 7 (19) |
| C3 | 2 (10) | 3 (19) | 5 (14) |
| Origin/C5 | 1 (5) | 0 | 1 (3) |
| Unspecified* | 3 (15) | 3 (19) | 6 (17) |
| Intracranial extension | 6 (30) | 4 (25) | 10 (28) |

Some vessels had >one dissection site.
*Unspecified VAD were extracranial.
VAD, vertebral artery dissection; ICAD, internal carotid artery dissection; CAD, craniocervical arterial dissection.

**Table 4** Arterial wall deficits of dissection cases

| Arterial wall deficit | VAD *n* = 20 (%) | ICAD *n* = 16 (%) | Total CAD *n* = 36 (%) |
|---|---|---|---|
| Dissection/intimal flap | 12 (60) | 7 (44) | 19 (56) |
| Double lumen | 5 (25) | 6 (38) | 11 (31) |
| Pseudoaneurysm | 1 (5) | 0 | 1 (3) |
| Dissecting aneurysm | 2 (10) | 3 (19) | 5 (14) |
| Intraluminal thrombus | 1 (5) | 3 (19) | 4 (11) |
| Mural hematoma | 4 (20) | 3 (19) | 7 (19) |
| Long tapering stenosis | 4 (20) | 7 (44) | 11 (31) |
| Stenosis | 15 (75) | 10 (63) | 23 (64) |
| Occlusion | 5 (25) | 6 (38) | 10 (28) |
| Stenosis >70% | 2 (10) | 2 (13) | 4 (11) |
| Stenosis 30–70% | 4 (20) | 2 (13) | 6 (17) |
| Stenosis <30% | 2 (10) | 0 | 2 (6) |

VAD, vertebral artery dissection; ICAD, internal carotid artery dissection; CAD, craniocervical arterial dissection.

### Arterial wall deficits

Arterial wall deficits were evident in both VADs and ICADs, with characteristics described in Table 4. Most commonly seen in VAD cases was a dissection or intimal flap, (12·60%), with five (25%) also showing a double lumen. The finding of an intimal flap was slightly less common in ICA [seven (44%)] cases, six (38%) having evidence of a double lumen. All VAD cases and four with ICADs showing an intimal flap had a history of trauma. Dissecting aneurysm (two VAD, 10%; three ICAD, 19%) and pseudoaneurysm (one VAD, 5%) were uncommon pathologies.

Stenosis was present in 15 (75%) VADs and 10 (63%) ICADs, with a long, tapering stenosis identified in four VADs

**Table 5** Infarct topography of dissection cases

| Location | VAD *n* = 20 (%) | ICAD *n* = 16 (%) |
|---|---|---|
| Infarct on MRI | 14 (70) | 9 (56) |
| Cerebellum | 8 (57) | 0 |
| Midbrain | 2 (14) | 0 |
| Medulla | 7 (50) | 0 |
| Occipital lobe | 2 (14) | 0 |
| Pons | 1 (7) | 0 |
| Middle cerebral artery | 0 | 8 (89) |
| Brainstem | 0 | 1 (11) |

VAD, vertebral artery dissection; ICAD, internal carotid artery dissection; MRI, magnetic resonance imaging.

and seven ICADs. Most commonly in both types of dissection there was total occlusion of the vessel.

Intraluminal hematoma (one VA, three ICA) and mural hematoma (four VAD, three ICAD) were much less commonly identified. Two ICADs had a SAH secondary to dissection. Seven (70%) ICADs with stenosis <70% or occlusion also had infarction. Ten (75%) of the VADs also had infarcts, but only 50% were <70% or occlusion.

### Infarct topography

Infarction on MRI was demonstrated in 22 (61%) cases (see Table 5). Of the VADs, 13 (65%) were in the anterior or posterior inferior cerebellum or lateral medulla. Four (31%) of cases had two or more sites of infarction. Nine (56%) of the ICADs showed infarction, mainly in the middle cerebral artery (MCA) territory with two (22%) cases having two sites of infarction. Figure 2 shows the topography and frequency of infarction in the different topographies of the posterior cerebral circulation, and Fig. 3 shows that for the anterior circulation. An increase in color density implies an increased frequency of infarction in a specific region. Interestingly, six (43%) VADs with infarction also had vascular anomalies. Thirty (70%) of control cases had infarcts, the majority of which were in the MCA territory.

## Discussion

This study investigated a group of young Australian patients with CAD and reported in detail the radiological features and brain topography of the infarct pattern associated with CAD (5,11,26) and related these to the presence of particular risk factors. Understanding of the particular radiological features of this condition and the relative importance of associated risk factors is important, as its recognition can sometimes be difficult particularly in primary care or emergency settings (27). Better knowledge of imaging characteristics of CAD in a young population is also important given the current interest in therapies for dissection, such as antithrombotic or endovascular treatment. Notably, 28% of patients had intracranial extension of their dissection, raising issues of SAH risk with the use of an anticoagulant. Our case series used multimodal imaging (CT and MR) and review by two assessors with consensus and provides the first pooled topographical mapping of infarction patterns in CAD patients.

The majority (89%) of dissections occurred extracranially, though 10 dissections (28%) extended intracranially. ICAD mainly occurred at the skull base or C2 level (56%), whereas VAD occurred mainly at the atlanto-axial (V3) segment of the artery (70%). This is in agreement with other authors describing CAD locations (3,28). Dissections where there was a history of trauma were more likely to be extracranial, 12 (92%) VADs and five (50%) ICADs. Seventy percent of VADs were at the V3 segment. Knowledge about common sites of dissection should prompt radiologists to explore these areas in particular when a dissection is suspected from the patient history. The high proportion of intracranial extension was an interesting finding that may be due, at least in part, to presentation severity and ascertainment bias given the hospital-based sample. However, none of these cases suffered SAH and did not appear to have a less favorable outcome with the majority (72%) having an mRS of 2 or less at follow-up. It would be interesting to follow this finding up in a larger prospective study to ascertain if the high proportion in the current study was due to chance.

Cerebral infarction occurred in 61% of dissection cases, similar to findings in the literature (14,17,22,26). For the posterior circulation, infarcts were most commonly located in the lateral medulla and also relatively commonly located in the anterior and posterior inferior cerebellar artery territories. This is similar to the findings of other authors (17) and may suggest not only an embolic mechanism but also *in situ* thrombosis of, or involvement at, the origin of the posterior inferior cerebellar artery. Infarcts were less common in the superior cerebellar artery territory and rare in the pons, midbrain, and thalamus. Two cases also had infarction in the occipital lobe indicating posterior cerebral artery involvement. In contrast with the findings of Koch *et al.* (17), multiple infarct sites were found in only six (27%) cases. Anterior circulation infarct topographies suggested a mix of embolic and potential hemodynamic mechanisms. Infarcts involving the middle cerebral artery territory, considered likely to be of embolic cause (16,26), were predominant. Watershed infarctions involving the internal and external watershed, considered likely to be hemodynamic (14,16,26), were less frequently observed but did occur in larger lesions similar to those identified by Lanczik *et al.* (14). Lanczik *et al.* suggest this type of lesion might be caused by sudden hemodynamic compromise, in contrast to the slow decrease in perfusion over time in atherosclerotic disease where collateral circulation might have time to develop. It is possible that trauma to the head or neck might provide such a mechanism for acute vessel obstruction.

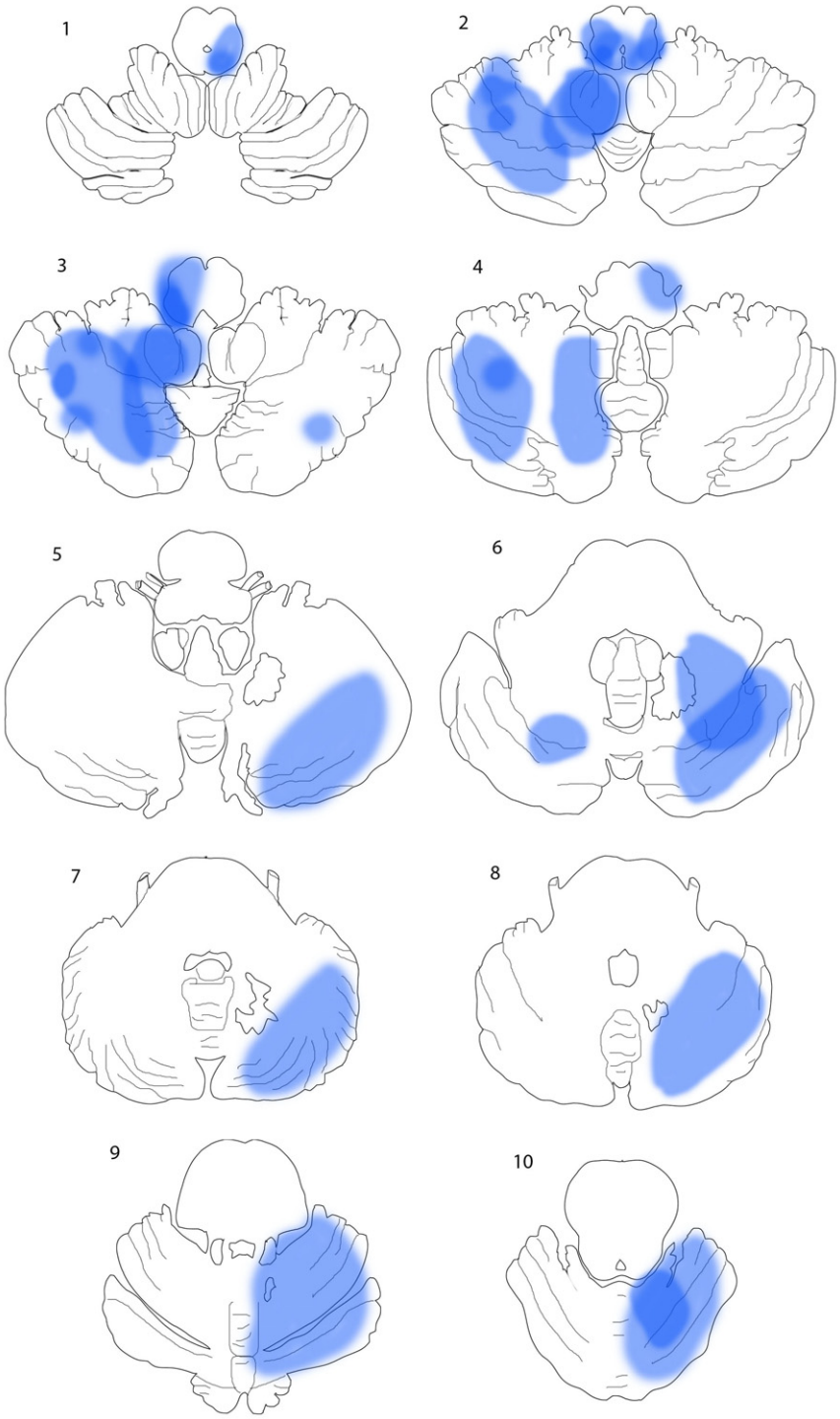


**Fig. 2** Schematic representation of brain showing pooled topography of brainstem and hemispheric infarction of posterior circulation dissections (VADs). Pooled mapping of infarct topography performed by layering of individual topography using Adobe Photoshop CS5 (Adobe Systems Incorporated, San Jose, CA, USA). VADs, vertebral artery dissections.

Infarction was more frequent in patients with VAD (70%) than ICAD (56%). Interestingly, six (43%) VAD cases with infarction also had vascular anomalies of the posterior circulation, which could perhaps have contributed to a lack of potential for collateral circulation to develop. Clinicians may consider there is a greater possibility of infarction in the presence of a vascular variant. Only two ICAD cases with infarction also had a vascular anomaly. Notably, cerebral infarction

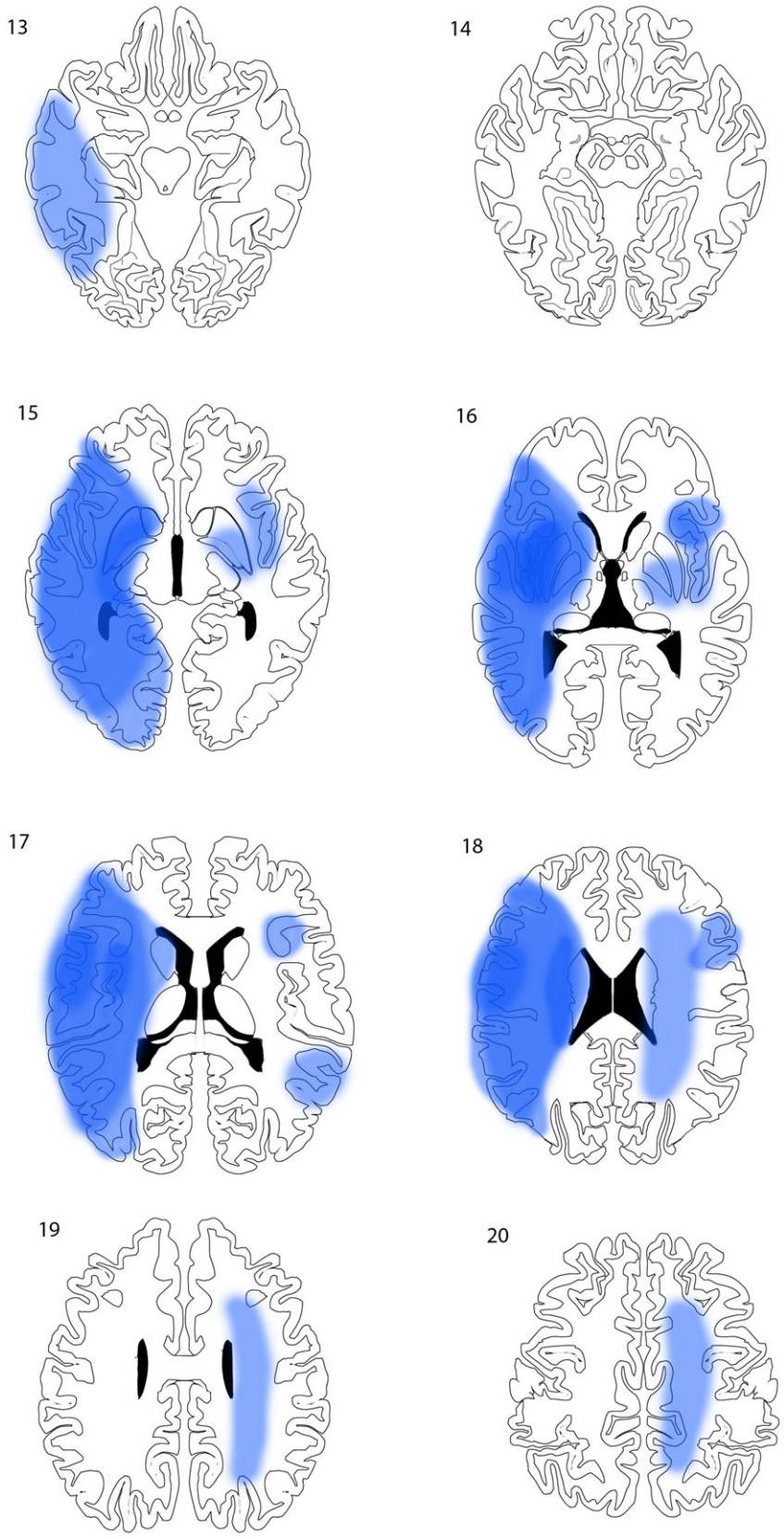


**Fig. 3** Schematic representation of brain showing pooled topography of brainstem and hemispheric infarction of anterior circulation dissections (ICADs). Pooled mapping of infarct topography performed by layering of individual topography using Adobe Photoshop CS5. ICADs, internal carotid artery dissections.

in VAD cases tended to occur with a lesser degree of stenosis than in ICAD cases, perhaps reflecting the efficiency of collateral flow in the posterior circulation.

Exposure to an environmental trigger such as mechanical trauma appeared to be associated with an increased risk of infarction. Fourteen (61%) CAD patients with infarction also had a history of minor mechanical trauma (nine VAD, five ICAD), whereas only three (7%) control cases had a history of recent trauma to the head or neck. Patients with infarction from VAD were much more likely to have a history of trauma (85%) compared with those from ICAD (50%). This may be due to the greater tortuosity of the vertebral artery and its relative fixation within the cervical spine making it more vulnerable to insult and trauma, thus potentially subjecting the arterial wall to greater stress.

The study identified 36 radiologically confirmed dissections, giving an annual incidence of approximately 1·01 cases per 100 000 population for the locality, extrapolated from recent regional stroke attack rates (29). This is considerably less than that generally reported (2·5–3 per 100 000) (3) perhaps highlighting difficulties in identification of CAD. An accurate estimate of incidence is likely to have been limited by the retrospective design, the hospital-based sampling frame, possible coding errors, and that radiological confirmation may have been limited by late imaging and resolution of the dissection signs.

Radiological confirmation of dissection was possible in 77% of patients (VAD 74%, ICAD 80%). Analysis of probable cases was not included as the purpose of the study was to describe the radiological features and risk factors of radiologically confirmed and definitive cases. Of the unconfirmed cases, imaging was often performed some time after the onset of symptoms, in some cases two- or five-months later. In other cases, dissection was strongly suspected due to the history and clinical signs, but pathognomonic signs such as an intimal flap or mural hematoma were not identifiable. It is possible that if a dissection was minor, it may have already healed by the time imaging was performed, particularly if this is as much as five-months later (22). The majority of unconfirmed cases occurred early in the study period perhaps reflecting an improvement in the ability of imaging to detect more subtle signs of dissection in recent years. There is also debate in the literature as to the reliability of MR or CT to detect signs of dissection in VAD or ICAD. Some authors advocate CTA as being more sensitive for the detection of clinical signs in VAD than MRA (26,28,30), whereas MRA has been reported as having a high sensitivity (95%) and specificity (99%) for ICAD but only low sensitivity (20%) and specificity (100%), respectively, for VAD (30). In the majority of our cases (69%), patients had both modalities performed. Digital subtraction angiography is not routinely performed in our unit for this condition.

Consistent with other studies (1,2,4), risk factors with statistically significant association were recent minor mechanical neck trauma ($P < 0{\cdot}000$), including manual therapy to the neck ($P = 0{\cdot}022$). Reporting bias may be present, however, as young people presenting with posterior circulation stroke are more likely to be questioned about neck trauma if other more obvious causes of stroke cannot be identified. In addition, VAD, involving the posterior cerebral circulation, was slightly more common (57%) than ICAD in the present study, which is in contrast with other reports (3,22). Strokes in the control group mainly involved the anterior circulation (88%). However, the sample was modest and may not reflect a true distribution of CAD. Limitations have been highlighted previously.

Radiological evidence of intimal flap or double lumen suggesting an intimal tear was the most common finding in all dissections 14 (39%) [seven (35%) VAD, seven (44%) ICAD], and all of the VAD cases had a history of trauma. Only six (17%) cases [three (15%) VAD, three (19%) ICAD] demonstrated a dissecting aneurysm or pseudoaneurysm suggesting a tear of the adventitial layer.

## Conclusion

Patients with CAD, particularly VAD, are more likely to have a recent history of trauma to the head or neck and are more likely to have extracranial pathology, in the atlas loop region in the case of VAD or at C2 or the skull base in ICAD. CAD cases with a history of trauma are more likely to have evidence of cerebral infarction. Intracranial extension was identified in about one in four cases. Vascular anomaly, particularly of the posterior circulation, is also associated with CAD and such cases are more likely to demonstrate infarction. Infarct topography of VAD typically involves the anterior and posterior inferior cerebellar artery territory and lateral medulla. Patients diagnosed with CAD, particularly VAD, who have radiological features suggestive of intimal tear, commonly have a history of trauma.

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