

Cervicocerebral artery dissections

Hairul A Ahmad, Richard P Gerraty, Stephen M Davis, Peter A Cameron

Abstract

Objective—To determine the aetiology, frequency, presentation, and outcome of blunt cervicocerebral arterial dissection presentations.

Patients and methods—Cases were retrospectively identified through the stroke registers at Royal Melbourne Hospital (a tertiary teaching hospital) and Geelong Hospital (a regional referral centre). Medical notes were then reviewed.

Results—A total of 18 cases were identified, with ages ranging from 28 to 53 years. Fifty five per cent of the injuries sustained were to the internal carotid artery and 45% to the vertebral artery. The majority of the injuries were either spontaneous or associated with trivial forces. Other causes included motor vehicle accidents, falls, and cervical manipulations. Fifty five per cent of patients complained of significant neck pain before presentation. Most patients had delayed presentations, with only 39% presenting on the day of the incident. Seventy eight per cent presented with a neurological deficit. Initial computed tomography was normal in 71% of patients. The majority of patients were managed with anticoagulation, and had minimal functional deficit on discharge. Other treatment modalities included surgery (one patient) and thrombolysis (two patients). One patient was managed conservatively.

Conclusions—The incidence of blunt cervicocerebral arterial dissection is unknown; however it is an uncommon diagnosis. The most common presentation is that of a delayed neurological event. Initial brain computed tomography is usually normal. Minimal adverse outcomes at discharge were noted in patients treated with anticoagulation only.

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Blunt vascular injuries are challenging problems with regard to diagnosis and management. They appear to be rare, with blunt carotid injuries estimated to occur in fewer than one in 1000 blunt injuries.¹ The majority of these injuries are manifest as arterial dissections. The diagnosis is difficult because of the lack of external signs and minimal evidence of internal injuries. The management of these cases is controversial.

This retrospective study examined cases of blunt cervicocerebral arterial dissection. The

diagnostic approach and management are discussed.

Patients and methods

The stroke registers for the years 1987 to 1996 in the Royal Melbourne Hospital and Geelong Hospital were reviewed. The Royal Melbourne Hospital is a 500 bed teaching hospital serving a population of one million. It treats approximately 500 stroke patients per year. Geelong Hospital is a 450 bed regional hospital serving a population of 250 000. Therefore the total population served by the two hospitals is 1.25 million. Patients suspected of having had strokes were documented in the stroke registers. All cases in which a diagnosis of carotid or vertebral artery dissection was made on discharge were reviewed with access to full hospital medical records.

The stroke registers would have identified nearly all the patients presenting with neurological deficit as the major presentation. Patients with major multiple injuries would not necessarily be included in the stroke registers as they would be admitted under surgical units. To assess the prevalence in this group the angiography registers of the previous six years were then reviewed. A further three cases were identified. All diagnoses of cervicocerebral vascular injuries were made on radiological findings.

The functional independence measure (FIM) used in the Major Trauma Outcome Study was used to assess outcome.² The section scores were then totalled (maximum of 12). A total score of 9-12 was described as having minor impairment, 5-8 as moderate impairment, and 3-4 as severe impairment.

Results

A total of 18 patients were identified (seven female and 11 male), with ages ranging from 28 to 53 years. Two of the patients had more than one artery affected. The majority (59%) of cases involved the internal carotid arteries (ICA) with the vertebrobasilar arteries (VBA) involved in 41%. There were no injuries to the common carotid artery documented.

In the majority of patients, the arterial dissection was either spontaneous or associated with a trivial force such as sporting activities and lifting heavy weights (table 1). The other major cause was trauma related to a motor vehicle accident (MVA). There were two cases associated with therapeutic manipulations performed by physiotherapists (injuries to ICA and VBA). One patient sustained dissections to both ICA after falling while snow skiing.

Significant medical history in these patients included migraine (20%), hypertension (5%), and smoking (30%). Ten of the 18 patients

**Emergency
Department, Royal
Melbourne Hospital**
H A Ahmad
P A Cameron

**Neurology
Department, Royal
Melbourne Hospital**
R P Gerraty
S M Davis

Correspondence to:
Associate Professor Peter
Cameron, Emergency
Department, Royal
Melbourne Hospital, Grattan
Street, Parkville, Victoria
3050, Australia.

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Table 1 Patients' profiles

No	Age	Sex	Mechanism of injury	Presentation		Computed tomography	Arteriography	Treatment	FIM
				Duration	Reason				
1	42	F	MBA, fracture of right tibia, facial laceration	<3 days	Internuclear, ophthalmoplegia	Normal	Dissection VBA (C3 level), FMD	Heparin→warfarin	11
2	47	F	MVA	>7 days	Left hemiplegia	Normal	Right ICA dissection (distal cervical segment), bilateral FMD	Heparin→warfarin	12
3	19	F	Turning neck while playing golf	>7 days	Right hemiparesis	Infarct left internal capsule, caudate nucleus	Left ICA dissection (bifurcation→ophthalmic artery)	Warfarin	12
4	41	M	Bending over	<1 day	Ataxia	Normal	Left VBA dissection (terminal portion)	Nil	10
5	34	M	MVA	>7 days	Ataxia, vomiting	Normal	VBA dissection	Heparin→warfarin	12
6	42	M	Lifting heavy object	<3 days	Headache	Normal	Right ICA dissection (proximal portion)	Heparin→warfarin	12
7	47	F	Post-hysterectomy	<1 day	Ataxia	Vermal and cerebellar infarct	Left VBA dissection and thrombosis	Heparin→warfarin	12
8	51	M	MVA	<1 day	MVA	Right frontoparietal infarct	Right ICA dissection (C2→foramen pseudoaneurysm)	Radiological occlusion (failed)→surgery	11
9	28	F	Neck manipulation	<7 days	Altered conscious state	Normal	Bilateral VBA dissection (C5→C6; C3)	Urokinase into basilar artery	Death day 5
10	41	F	Turning neck (skiing)	<3 days	Right hemiplegia	Basilar artery thrombosis	Distal (left) VBA dissection	Urokinase →heparin→warfarin	11
11	43	M	Fall during skiing	<3 days	Right hemiplegia	Left MCA thrombosis	Left ICA dissection, right ICA dissection	Heparin→warfarin	11
12	50	M	Neck manipulation	<1 day	Dysphasia	Left temporoparietal infarct	Left ICA dissection (above bifurcation)	Warfarin	11
13	50	M	Sudden neck rotation	<1 day	Right Horner's syndrome, headache	Normal	Right ICA dissection (distal portion)	Heparin→warfarin	12
14	38	M	Playing basketball	<1 day	Right facial anaesthesia	Normal	Right VBA dissection (distal portion)	Heparin→warfarin	10
15	44	M	Rotation of neck	>7 days	Visual disturbance	Normal	Right ICA dissection (base of skull)	Warfarin	12
16	45	M	Turning neck	<1 day	Right hemiplegia	Normal	Left ICA dissection (proximal portion)	Heparin→warfarin	10
17	53	F	Playing tennis	<7 days	Left hemiplegia	Normal	Right ICA dissection (distal portion)	Heparin→warfarin	12
18	40	M	Fall while walking	>7 days	Left hemianaesthesia	Normal	Right VBA dissection (distal portion)	Heparin→warfarin	12

FMD: fibromuscular dysplasia; ICA: internal carotid artery; MBA: motor bike accident; MVA: motor vehicle accident; VBA: vertebral artery.

(55%) noted pain to the affected side of the neck before presentation.

The majority of patients had a delay in presentation of more than one day (61%) (table 1). Fourteen out of the 18 patients presented with a neurological deficit. One case was identified as part of a multiple trauma assessment (patient 8, table 1).

Of the four patients who were involved in a MVA, arterial dissection was suspected on the day of incident in only one case. Out of these four patients only one sustained other injuries (orthopaedic and facial laceration).

All 18 patients had computed tomography of the head at initial presentation as part of the investigation; however only six were noted to be abnormal.

The majority of the patients were treated with anticoagulation using a combination of heparin and warfarin (table 1). One patient had radiological occlusion of an aneurysm extending from the C2 level to the foramen magnum. Two patients were treated with thrombolysis using urokinase infused directly into the affected arteries.

All the patients treated with anticoagulation or conservatively were discharged with minimal functional disabilities (FIM 9–12). The patient who was treated surgically had residual disability (FIM 11). Of the two patients who had thrombolysis, one died, while the other had a FIM score of 9–12. The immediate cause of death was pneumonia on day 5 of admission. This 28 year old woman suffered a dissection of her bilateral cervical VBA as a result of neck manipulation. She was treated with urokinase infusion delivered directly into the affected system.

Discussion

This retrospective review demonstrates the rarity of identified blunt cervicocerebral arterial dissections. Various studies have estimated that these injuries occur in fewer than 3% of all carotid artery injuries.³⁻⁵ This study documented an annual incidence of 1.4 per million patients per year. However the true incidence is not known as it is almost impossible to quantify asymptomatic cases. Moreover, not all ischaemic strokes are investigated with angiography. The possible underdiagnosis of this disorder carries significant implications for the patient's functional outcome.

The reported causes of blunt cervicocerebral arterial dissection include MVA, falls, chiropractic manipulation, strangulation, and diagnostic carotid compression.⁶⁻⁸ The pathogenesis of the mechanism involves hyperextension, hyperflexion, rotation, or stretching of the arteries resulting in intimal tear, dissection, pseudoaneurysm, and arteriovenous fistula. In this study similar mechanisms were identified. The VBA are more protected than carotid arteries from blunt injuries due to the protection afforded by the cervical vertebrae.

This study identified a significant percentage of patients who suffered arterial dissection with trivial or no obvious trauma. The activities that were reported to be associated included sporting activities, turning the neck, and lifting heavy objects. Other studies have identified a similar group of patients.⁹⁻¹⁰ Most authors considered spontaneous dissection and those associated with trivial injuries to be the same entity.¹¹⁻¹³ Various pathological processes have been proposed to explain the increased susceptibility to spontaneous dissection. These in-

cluded fibromuscular dysplasia, atheromatous plaques, Ehlers-Danlos disease, or Marfan's syndrome.^{9 14-16}

One of the difficulties in the diagnosis of blunt vascular injuries is delayed presentation. In this study, more than 50% of the patients presented more than one day after the incident. This is also true for those that sustained the injuries as a result of a MVA. Neck pain as a preceding symptom was noted in 55% of patients. Previous studies demonstrated that neck pain often precedes the onset of other symptoms.¹⁷

The majority of these patients presented with a neurological deficit. Computed tomography is rarely useful in the diagnosis of arterial dissection, although dynamic computed tomography has been used.¹⁸ Only six of the 18 cases had abnormal computed tomography. Therefore we recommend that arteriography be performed to make or exclude the diagnosis of dissection if there is a high degree of suspicion. Patients in this high risk group includes young patients, those with hyperflexion, hyperextension or rotational forces to the neck, those complaining of pain to the affected side of the neck, and those with neurological symptoms not explained by other mechanisms. Similar risk groups was identified by Davis and Cogbill.^{1 19}

The management of non-penetrating injuries of cervicocerebral arteries remains controversial. A rational management strategy is difficult to devise due to the rarity of the problem with reported series consisting of small numbers. This is further complicated by the lack of consistent stratification of injuries based on site, type, and extent of injuries and also severity of neurological deficit and other associated injuries. Previous studies have suggested that these rare injuries are associated with mortality rates of 20%–40% and permanent neurological impairment of 40%–80%.^{3 20 21} There appeared to be a significantly better outcome in this study.

In other studies, most cases have been treated with anticoagulation (heparin with or without warfarin). Due to the reported high incidence of severe neurological impairment in the non-surgical group compared with that in the surgical group, aggressive surgical treatments had been advocated for these lesions.^{22 23} However more recent publications have advocated conservative management.^{1 20 24}

The majority of cases treated in this study suffered from dissection without evidence of thrombus. Prevention of arterial thrombosis and embolisation is the rationale behind anticoagulation in these patients. All of these patients were discharged with no or minimal functional impairment (FIM >9). Whether the same result could have been obtained in those patients with no intervention at all cannot be predicted from this study.

Conclusion

This study demonstrated that non-penetrating vascular injuries with neurological symptoms

or signs were uncommon. Injuries occurred in the ICA and VBA. Most patients present with delayed neurological deficit. High risk groups (young patients, those with hyperflexion, hyperextension, or rotational forces to the neck, those complaining of pain to the affected side of the neck, and those with unexplained neurological symptoms) should undergo arteriography. The majority of arterial dissections can be managed with anticoagulation with no or minimal functional impairment.

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