

Are your MRI contrast agents cost-effective?

Learn more about generic Gadolinium-Based Contrast Agents.



**FRESENIUS
KABI**

caring for life

AJNR

Unusual dissection of the proximal vertebral artery: description of three cases.

D P Friedman and A E Flanders

AJNR Am J Neuroradiol 1992, 13 (1) 283-286

<http://www.ajnr.org/content/13/1/283>

This information is current as
of April 19, 2024.

Unusual Dissection of the Proximal Vertebral Artery: Description of Three Cases

David P. Friedman¹ and Adam E. Flanders

Summary: We report three cases that reveal an array of etiologic and radiologic findings associated with dissection of the proximal segment of the vertebral arteries. Regardless of etiology, the proximal segment may be the principal site of dissection in these vessels.

Index terms: Arteries, vertebral; Arteries, dissection

Dissection of the extracranial vertebral arteries is infrequently recognized as a cause of vertebrobasilar stroke. Injury to these vessels has been associated with multiple causes, including fracture-dislocation of the cervical spine, chiropractic manipulation, chronic subluxation of the atlantoaxial joint, yoga, and other forms of minor trauma (1–5). Multiple cases have no apparent etiology and are termed “spontaneous” (2). Connective tissue disease, fibromuscular dysplasia, migraine headache, hypertension, and oral contraceptive use are believed to be predisposing factors (1–5). Neck pain followed by unilateral vertebrobasilar ischemia is the most frequent initial symptom. The third segment of the vertebral artery, which extends from its exit from C2 to the dura, is most often affected. Involvement of the first segment, which extends from its origin from the subclavian artery to its entry into the foramen transversarium of a cervical vertebra (usually C6), is very uncommon (1). In this article we report three cases of proximal dissections having a spectrum of etiologic and radiographic findings.

Case Reports

Case 1

A 47-year-old man with no history of antecedent trauma presented with neck pain, dizziness, and vomiting. Clinical examination revealed a right Wallenberg syndrome. A magnetic resonance (MR) scan was obtained on a 1.5 T superconducting magnet (General Electric, Milwaukee, WI). T2-

weighted (2000/80/1) (TR/TE/excitations) axial and coronal images showed an infarction in the right posterior inferior cerebellar artery (PICA) distribution (Figs. 1A and 1B). A right subclavian angiogram (Philips Maximus C1250; Einhoven, The Netherlands) showed dissection of the right vertebral artery origin (Fig. 1C). The left vertebral artery was normal, and there was no evidence of atherosclerotic disease involving the great vessels or carotid bifurcations. The patient was treated with anticoagulants, and there was moderate improvement in his neurologic deficits.

Case 2

A 58-year-old man sustained a C5–C6 fracture-dislocation (Fig. 2A) after a motor vehicle accident. He developed dizziness, nausea, and vomiting 48 hr post injury. Clinical examination revealed a left Wallenberg syndrome. A T2-weighted MR scan showed hyperintensity in the left medulla and left cerebellar hemisphere, typical of a left PICA territory infarction. Bilateral subclavian angiography demonstrated dissection with occlusion of both proximal vertebral arteries (Figs. 2B and 2C). These dissections were well below the level of the fracture-dislocation. Although the patient received anticoagulants, he eventually succumbed to multiple medical complications related to his injury.

Case 3

A 35-year-old man presented with signs of a left cerebellar lesion 2 hr after chiropractic manipulation of his neck. A T2-weighted MR scan showed hyperintensity and swelling of the left cerebellar hemisphere, with secondary tonsillar herniation. These findings were most consistent with an early cerebellar infarction. Bilateral subclavian angiography revealed dissections of both proximal vertebral arteries (Figs. 3A and 3B). There was moderate improvement in his neurologic deficit after subsequent anticoagulant therapy.

Discussion

The mechanism of insult to the vertebral artery in closed neck trauma is through stretching and

Received May 20, 1991; revision requested June 11; revision received on July 3 and accepted on July 9.

¹ Both authors: Department of Radiology, Jefferson Medical College and Thomas Jefferson University Hospital, 10th and Sansom Streets, Philadelphia, PA 19107. Address reprint requests to D. P. Friedman.

AJNR 13:283–286, January/February 1992 0195-6108/92/1301-0283 © American Society of Neuroradiology

Fig. 1. Case 1: 47-year-old man with spontaneous onset of Wallenberg syndrome.

A and B, T2-weighted (2400/80) axial and coronal MR images show hyperintensity in the right medulla (arrow) and right cerebellar hemisphere. The distribution is typical of a right PICA territory infarction.

C, Frontal subtraction view, right subclavian angiogram. The origin of the right vertebral artery is severely narrowed (arrows), most consistent with dissection. There is incomplete opacification of the vessel distal to the dissection, suggesting intramural and/or intraluminal thrombus.

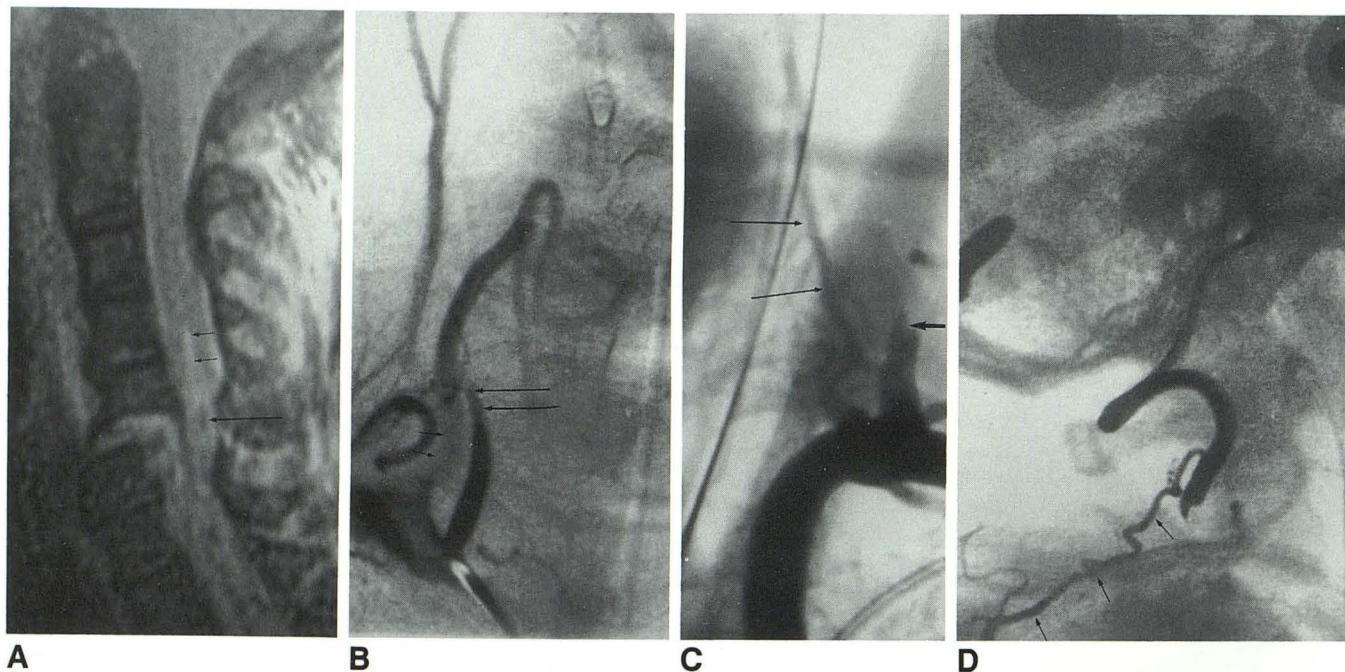
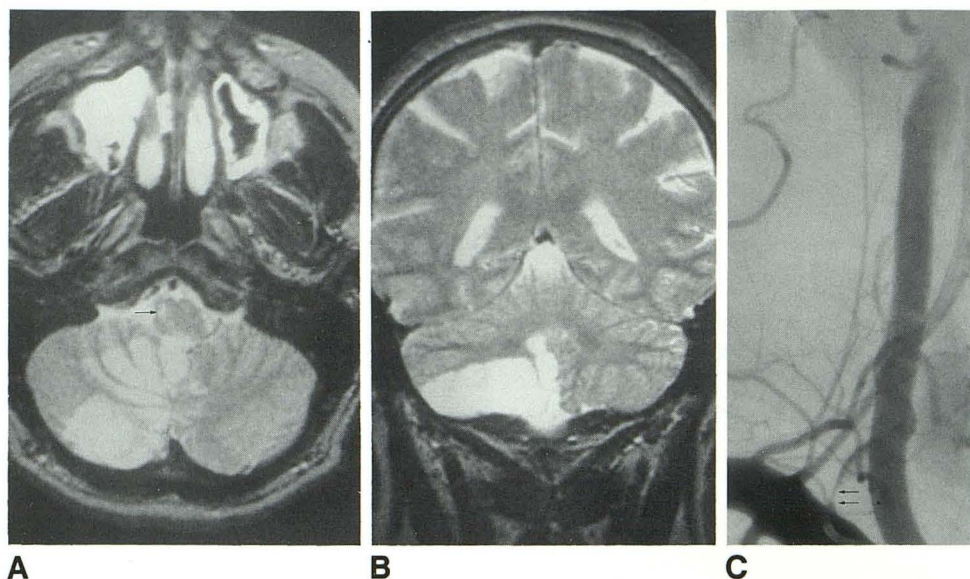


Fig. 2. Case 2: 58-year-old man with Wallenberg syndrome after a C5-6 fracture-dislocation.

A, T2-weighted (2000/80) sagittal view of the cervical spine demonstrates a C5-6 fracture-dislocation; intramedullary hematoma (long arrow) and edema (short arrows) are identified. The patient became quadriplegic as a result of the injury.

B, Frontal subtraction view, right subclavian digital subtraction angiography (DSA). There is a tapered occlusion of the right vertebral artery (long arrows) well below the level of C5-6. Short arrows identify the thyrocervical trunk.

C, Frontal subtraction view, left subclavian DSA. There is a more abrupt occlusion of the left vertebral artery (short arrow). The left ascending cervical artery is prominent (long arrows).

D, Lateral subtraction view, left subclavian DSA. The ascending cervical artery reconstitutes the distal left vertebral artery through an enlarged muscular collateral (arrows).

tearing of the intimal and medial tissues of the vessel. This may lead to vasospasm, intramural hematoma, pseudoaneurysm formation, or thrombosis (4). Although many dissections are subclinical, others may result in devastating vertebrobasilar strokes. As demonstrated in this se-

ries and confirmed in the literature, Wallenberg syndrome and cerebellar infarction are especially common clinical presentations (1-5). There was excellent correlation between the cranial MR examinations and the clinical findings in these three patients. The infarctions may be due to throm-

basis of the vertebral artery, propagation of intramural thrombus, or more distal branch occlusion from intraluminal thromboembolism. The time of onset of the neurologic syndrome in relation to the dissection is variable, and may not be seen for weeks or months following injury. In the past, only a very high index of suspicion and early angiography could result in the diagnosis and treatment of dissection *before* the clinical manifestations became evident.

For purposes of anatomic localization, the vertebral artery may be divided into four segments: first segment (V1), from its origin to its entry into the foramen transversarium of a cervical vertebra, usually C6; second segment (V2), from the foramen of C6 to that of C2; third segment (V3), from its exit from C2 to the dura; and fourth segment (V4), the intracranial vertebral artery (1, 6) (Fig. 4). After arising from the subclavian artery, the first segment is held tightly by fascia as it traverses the neck to enter the transverse foramen of C6. At this point the vertebral artery passes between the tendons of the anterior scalene, middle scalene, and longus colli muscles. It then ascends in its second segment in a relatively straight course through the transverse foramina of C6 to C2. The third segment exits from the transverse foramen of C2 and runs posteriorly and medially around the lateral mass of C1. The fourth segment penetrates the oblique ligament of the atlas, pierces the dura and passes upward through the foramen magnum to unite with the opposite vertebral artery to form the basilar artery (6, 7). The first and third portions of the vertebral artery are more movable than the second and fourth, the latter two segments being relatively fixed by bony attachments. Motion of the V1 segment against the fascial bands and skeletal muscles that traverse it can result in damage to the vessel (3). There is extensive *in vivo* and *in vitro* evidence that the vertebral arteries may be compromised at the atlantoaxial articulation by head motion, particularly rotation and extension (4, 7).

In all larger series of vertebral artery dissections, the V3 segment was the most frequently affected level (1-5); involvement of the V1 segment was relatively rare (1). Review of the recent literature yielded 56 patients with a total of 77 vertebral artery dissections (1-5, 8-10). Etiologic factors ranged from "spontaneous" to major trauma. In only seven patients (9%) did the dissection affect the V1 segment; moreover, there were no cases with bilateral proximal involve-

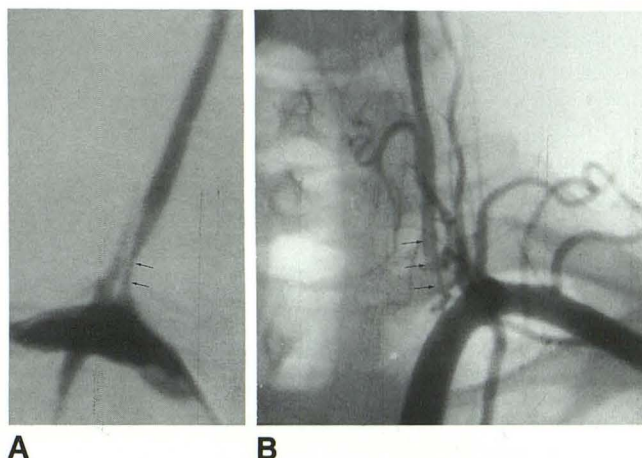


Fig. 3. Case 3: 35-year-old man who developed ataxia and dizziness 2 hr after chiropractic manipulation.

A, Frontal subtraction view, right subclavian DSA. There is marked narrowing of the proximal right vertebral artery (arrows).

B, Frontal subtraction view, left subclavian angiogram. Dissection of the proximal left vertebral artery is clearly identified (arrows). The appearance is more irregular than the right side, consistent with the patient's left-sided symptoms.

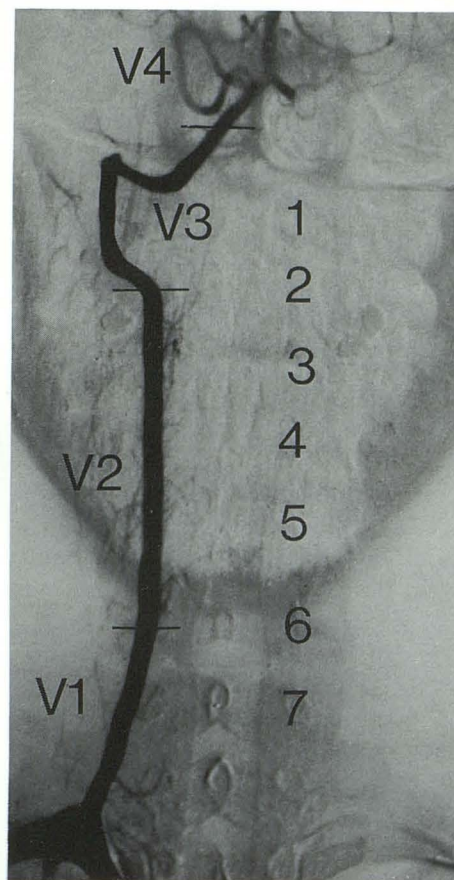


Fig. 4. Frontal subtraction view, right subclavian angiogram. The segmental anatomy of the right vertebral artery is demonstrated. The numbers on the left indicate each cervical level.

ment. Mas et al (1) reviewed 38 additional cases; isolated V1 lesions were seen in only 6% of patients. The fact that there were bilateral dissections in two of our three cases is probably a chance occurrence. Bilateral dissections at more distal levels have already been well described in the literature (29%–61% of cases) (1, 2). In addition, multiple segments may be affected.

These cases reveal the spectrum of etiologies (major trauma, minor trauma, and "spontaneous") that can be seen with proximal vertebral artery dissection. Patients 2 and 3 are remarkable for bilateral lesions; patient 2 is also unusual in that the vertebral artery occlusions were well below the level of the cervical spine trauma. The V2 segment is most often affected in cases of fracture-dislocation (1), with the dissection beginning at the level of injury. Fracture extending through the foramen transversarium need not be present.

Angiographic criteria of dissection include intimal flap, irregularity and/or tapering of the vessel, pseudoaneurysm formation, and thrombosis (1–4). Although there is no pathologic proof in our cases, the radiographic findings are quite typical of dissection. Moreover, all were previously healthy, without specific risk factors for atherosclerotic disease or cerebral embolism. These patients underscore the importance of studying (in cases of suspected vertebral dissection) the origins of both vessels via subclavian injections prior to selective catheterization. In addition, our cases demonstrate that if MR in the future is to be used as a screening and/or defini-

tive diagnostic tool for dissection, the *entire* course of the extracranial vertebral arteries must be imaged.

In summary, review of the literature indicates that dissection involving the V1 segment of the vertebral arteries occurs in less than 10% of cases. However, our series demonstrates that, irrespective of etiology, the proximal segment may be the principal site of dissection of these vessels.

References

1. Mas JL, Bousser MG, Hasboun D, Laplane D. Extracranial vertebral artery dissections: a review of 13 cases. *Stroke* 1987;18:1037–1047
2. Mokri B, Houser OW, Sandok BA, Piepgras DG. Spontaneous dissections of the vertebral arteries. *Neurology* 1988;38:880–885
3. Caplan LR, Zarins CK, Hemmati M. Spontaneous dissection of the extracranial vertebral arteries. *Stroke* 1985;16:1030–1038
4. Katirji MB, Reinmuth OM, Latchaw RE. Stroke due to vertebral artery injury. *Arch Neurol* 1985;42:242–248
5. Youl BD, Coutellier A, Dubois B, Leger JM, Bousser MG. Three cases of spontaneous extracranial vertebral artery dissection. *Stroke* 1990;21:618–625
6. Newton TH, Mani RL. The vertebral artery. In: Newton TH, Potts DG, eds. *Radiology of the skull and brain: angiography*. St. Louis: C.V. Mosby, 1974:1659–1673
7. Davis JM, Zimmerman RA. Injury of the carotid and vertebral arteries. *Neuroradiology* 1983;25:55–69
8. Fakhry SM, Jaques PF, Proctor HJ. Cervical vessel injury after blunt trauma. *J Vasc Surg* 1988;8:501–508
9. Quint DJ, Spickler EM. Magnetic resonance demonstration of vertebral artery dissection. *J Neurosurg* 1990;72:964–967
10. Rae-Grant AD, Lin F, Yaeger BA, et al. Post-traumatic extracranial vertebral artery dissection with locked-in syndrome: a case with MRI documentation and unusually favorable outcome. *J Neurol Neurosurg*