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Traumatically Induced Vertebral Artery Occlusion Associated with Cervical Spine Injuries: Prospective Study Using Magnetic Resonance Angiography

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Study Design. A prospective study using magnetic resonance angiography (MRA) on a consecutive cohort of patients with cervical spine injuries.

Objective. To investigate clinical and radiographic features of vertebral artery injury/occlusion associated with nonpenetrating cervical spine trauma.

Summary of Background Data. With the popularization of MRA, vertebral artery injury has been a common complication of cervical spine trauma. However, detailed clinical features such as restoration of blood flow in occluded vessels and collateral circulation have not been fully evaluated.

Methods. During a 2-year period, 64 consecutive patients with cervical spine fractures and/or dislocations were prospectively evaluated for patency of the vertebral artery and collateral circulation. Extracranial and intracranial MRAs were conducted at initial injury and follow-up.

Results. Vertebral artery occlusion occurred in 11 patients, including 10 with unilateral and 1 with bilateral. Only the patient with bilateral occlusion was symptomatic but had no permanent neurologic deficit as a result of brain ischemia. He had complete circle of Willis, which provides sufficient collateral blood supply from anterior circulation. Follow-up MRAs revealed restoration of blood flow in occluded vessel(s) in 3 patients. All of them had compressive injuries.

Conclusions. The incidence of traumatically induced vertebral artery occlusion was 17.2%. The potential for blood flow restoration was higher in compressive injuries than in distractive injuries. The mechanism of occlusion in compressive injuries is likely to be vasospasm or minor artery dissection, which may cause reversible occlusion because vessels are subjected to relatively minor stretching in compressive injuries. Vertebral artery occlusion was rarely symptomatic because of sufficient collateral blood supply through not only contralateral vertebral artery but also the circle of Willis.

Key words: vertebral artery occlusion/injury, cervical spine injury, magnetic resonance angiography, collateral circulation, vertebrobasilar ischemia. **Spine 2005;30: 1955–1962**

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Although vertebral artery injuries caused by penetrating injuries to the neck, chiropractic manipulation, yoga, and sustained physiologic movement have been well described, those associated with nonpenetrating cervical spine injuries were thought to be infrequent because most of them were asymptomatic.[1–5] During the last decade, with the popularization of magnetic resonance angiography (MRA), several prospective studies using MRA showed that the vertebral artery injury secondary to blunt cervical spine trauma was not uncommon as previously considered.[4,6–8] However, follow-up observation of the injured vertebral arteries and evaluation of collateral pathways to the vertebrobasilar territory in the setting of unilateral or bilateral vertebral occlusion has not been sufficiently conducted. We performed a prospective study using MRA to investigate not only the incidence and pathomechanism of vertebral artery injuries following blunt cervical spine trauma but also detailed clinical features, including long-term neurologic outcomes, restoration of blood flow in the occluded arteries, and collateral circulation to the vertebrobasilar territory in the brain.

Methods

During the 2-year period from January 2000 to December 2001, all patients admitted or transferred to the authors' center for acute cervical spine injuries were considered for MRA evaluation as part of a prospective study to investigate the incidence, pathomechanism, and clinical features of traumatically induced vertebral artery injuries. Inclusion criteria for this prospective analysis were: (1) admission or transfer to our institute within 48 hours of injury; (2) injury to the cervical spine with radiographically evident fractures and/or dislocations; and (3) spinal injury occurring between the level of C2 vertebra and C6/7 spinal segment, which is consistent with the second segment of the vertebral arteries (Figure 1). A total of 64 consecutive patients met eligibility requirements during the time frame of the study. There were 50 male and 14 female patients, with an average age of 50.2 years (range 17–89). All MRAs were performed on a 1.5-Tesla superconducting magnetic resonance imager (MAGNETOM, Siemens, Erlangen, Germany) at admission.

The MRA technique consisted of a 2-dimensional time-of-flight pulse sequence (repetition time 27 milliseconds, echo time 9 milliseconds, flip angle 20°, field-of-view 23 cm). A

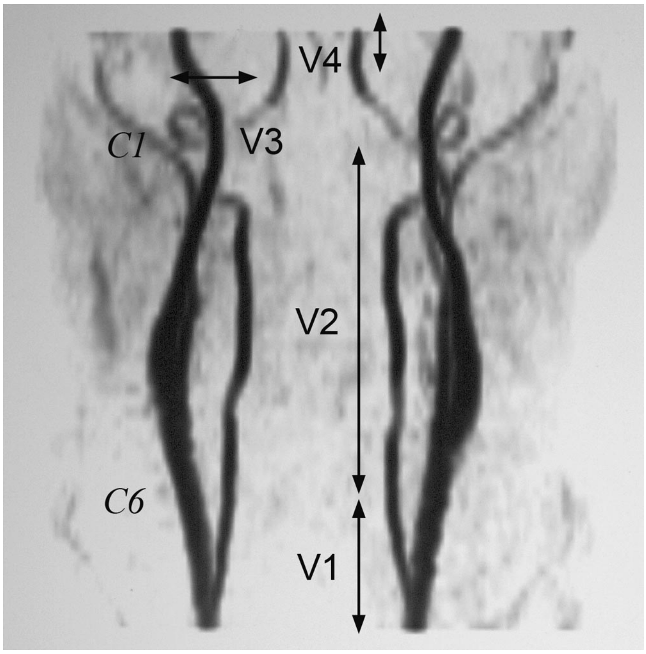

Figure 1. MRA of nonoccluded extracranial vertebral artery obtained from patient with C4 dislocation. MRA shows 4 segments of the vertebral artery: first (V1), subclavian to entrance to the foramen transversarium (C6 or C7); second (V2), C6–C1, in which the vertebral arteries pass through the foramen transversarium; third (V3), C1 to entrance into the dura; and fourth (V4), entrance to its termination in the basilar artery.

saturation pulse was applied superiorly to suppress venous flow. All images were reconstructed at 10° intervals using a maximum intensity projection algorithm. Only nonvisualization of the first through second segment of the vertebral artery in the expected location, which indicates occlusion, was determined by extracranial MRA.[4,6,7] Other findings, such as luminal narrowing or increase in external diameter of the vertebral arteries, were not assessed because of inherent spatial limitations on the MRA technique. In the patients with vertebral artery occlusion detected by initial MRA, an additional intracranial MRA was performed to examine the configuration of the circle of Willis as collateral pathways to vertebrobasilar territory from the anterior circulation of the brain. Furthermore, all patients in whom vertebral artery occlusion had been detected at initial MRA underwent follow-up MRA study 1–17 months after injury to investigate restoration of blood flow in the occluded vertebral artery. Magnetic resonance imaging (MRI) of the whole brain was performed to assess brain infarction at the same time of the initial and follow-up MRAs on all patients with vertebral artery occlusion.

Regarding neurologic examination, not only specific findings for spinal cord injury but those for vertebrobasilar ischemia (*e.g.*, dizziness, vertigo, dysarthrias, dysphasia, visual field defect, blurry vision, drowsiness, and altered consciousness) were carefully evaluated and prospectively recorded in all patients. Injury types according to Allen *et al*[9] classification were distractive flexion in 21, compressive extension in 20, compression flexion in 11, vertical compression in 5, distractive extension in 3, and lateral flexion in 1 patient. This cohort included 3 other patients who had a hangman fracture. All patients underwent early reduction and surgical stabilization with titanium instrumentation when clinical and radiographic evaluations had been completed. Anterior and/or posterior stabilization was properly selected in each case according to the type of spinal injuries. For statistical analysis, the Fisher exact test was used. The results were considered statistically significant if the P value was <0.05.

Results

Occlusion of the vertebral artery was shown on MRA in 11 of the 64 patients (17.2%) (Table 1). There were 9 male and 2 female patients, with an average age of 54.3 years (range 33–79). All 11 patients were followed up for a minimum of 2 years, with an average of 32.8 months (range 24–46). Unilateral occlusion of the right vertebral artery occurred in 6 patients and of the left in 4. Only 1 patient had bilateral vertebral artery occlusions. Injury types of the cervical spine were distractive flexion in 6 patients, compressive flexion in 3, and compressive extension in 2. The incidence of vertebral artery occlusion by injury types was 28.6% in distractive flexion, 27.3% in compressive flexion, and 10% in compressive extension. Levels of the spine fractures and/or dislocations existed in C3/4 segment in 1 patient, C4/5 in 3, C5/6 in 3, C4/5/6 in 2, C5/6/7 in 1, and C6/7/T1 in 1. Of 11 patients with vertebral artery occlusion, 6 had facet dislocation, including 4 unilateral and 2 bilateral, whereas of 53 patients without vertebral artery compromise, 15 had a facet dislocation, including 4 unilateral and 11 bilateral. There was a statistically significant association between unilateral facet dislocation and vertebral artery occlusion ($P = 0.024$).

Of 11 patients with vertebral artery occlusion, 5 had a foramen transversarium fracture ipsilateral to the involved side. On the other hand, 13 of 53 patients without vertebral artery compromise had a foramen transversarium fracture. Although transverse foramen fractures were more frequently observed in the patients with vertebral artery occlusion than in those without, there was no significant statistical difference ($P = 0.267$) between patients with and without vertebral artery occlusion regarding foramen transversarium fractures.

Three of 11 patients with vertebral artery occlusion had complete configuration in the posterior part of the circle of Willis in which the bilateral posterior communicating artery or fetal-type posterior cerebral artery was present (Figure 2D).[10,11] The circle of Willis with complete posterior configuration can provide collateral blood flow to the vertebrobasilar territory *via* the posterior communicating artery from anterior circulation of the brain. The configuration of the circle of Willis in the other 8 patients was incomplete (Figures 3D, available for viewing online through ArticlePlus only; 4C, available for viewing online through ArticlePlus only). The patient who had bilateral vertebral artery occlusion had complete posterior configuration of the circle of Willis.

All 11 patients with vertebral artery occlusion had spinal cord injury. Neurologic status evaluated by the American Spinal Injury Association (ASIA) impairment scale (AIS)[7] was grade A in 8 patients, B in 1, and C in 2.

Table 1. Data for Patients with Vertebral Artery Occlusion

Patient No.	Age (ys)/ Gender	Cause of Injury	AIS Grade/ Level		Cervical Spine Injury	Injury Class/ Stage (Allen *et al* [9])	Side of VA Occlusion	VA Symptoms		Posterior Configuration of circle of Willis	Timing of F/U MRA (mos)	Restoration of Blood Flow	Length of F/U (mos)
			Initial	F/U				Initial	F/U				
1	33/F	MVA	C/C5	D/C5	C4–C5 bilateral facet dislocation	DF/3	Left	None	None	Complete	6	No	39
2	79/M	Fall	A/C6	A/C6	C5–C6 bilateral facet dislocation C6 right transverse foramen fracture	DF/3	Right	None	None	Incomplete	4	No	26
3	38/F	MVA	B/C6	C/C8	C5–C6 unilateral facet dislocation C6 left transverse foramen fracture	DF/2	Left	None	None	Incomplete	3	No	29
4	59/M	Fall	A/C5	A/C5	C4–C5 unilateral facet dislocation C5 bilateral transverse foramen fracture	DF/2	Right	None	None	Incomplete	17	No	41
5	70/M	Fall	A/C5	A/C6	C4–C5 unilateral facet dislocation C5 left transverse foramen fracture	DF/2	Right	None	None	Incomplete	3	No	32
6	55/M	Fall	A/C5	A/C5	C5–C6 unilateral facet dislocation C6 left transverse foramen fracture C5 vertebral body fracture	DF/2	Left	None	None	Incomplete	10	No	24
7	32/M	MVA	A/C5	A/C6	C6 teardrop fracture C6 posterior subluxation	CF/5	Left	None	None	Complete	1	No	37
8	49/M	MVA	A/C4	A/C4	C5 teardrop fracture C5 posterior subluxation C4 right lateral mass fracture	CF/5	Right	None	None	Incomplete	11	yes	46
9	34/M	MVA	A/C5	A/C5	C5 burst fracture	CF/4	Bilateral	Blurry vision	None	Complete	1	Yes	33
10	74/M	MVA	A/C7	A/T1	C7 right lateral mass fracture	CE/1	Right	None	None	Incomplete	3	No	28
11	74/M	Fall	C/C6	D/C8	C3 inferior facet fracture C3 anterior subluxation C4 right transverse foramen fracture	CE/1	Right	None	None	Incomplete	3	Yes	26

AIS = ASIA impairment scale; CE = compressive extension; CF = compressive flexion; DF = distractive flexion; F/U = follow-up; MVA = motor vehicle accident; VA = vertebral artery.

While, in the other 53 patients without vertebral artery occlusion, there were 24 with AIS A, 11 with B, 10 with C, 4 with D, and 4 with E. Although complete cord injuries (AIS A) were more frequently observed in the patients with vertebral artery occlusion than in those without, there was no significant correlation between the severity of spinal cord injury and vertebral artery occlusion ($P = 0.184$). None of 11 patients with vertebral artery occlusion had radiographically detected brain trauma at injury. Only 2 of 53 patients without vertebral artery occlusion had major brain injury (*e.g.*, traumatic subarachnoid hemorrhage). There was no significant association between vertebral artery occlusion and brain trauma ($P = 0.513$). Regarding clinical manifestations related to vertebrobasilar ischemia, 10 patients with unilateral vertebral artery occlusion had no symptoms, whereas 1 with bilateral occlusion had transient blurry vision. This transient visual symptom rapidly subsided within a few hours after injury. No patient underwent systemic anticoagulation therapy because of a lack of persistent symptoms related to vertebrobasilar ischemia, and its potential adverse effect to the injured spinal cord with intramedullary hemorrhage and the other hemorrhagic traumatic lesions. No neurologic impairment in terms of vertebrobasilar ischemia occurred during follow-up.

Follow-up MRA was obtained at 5.6 months after injury (range 1–17) on the average. Although metal artifacts of used titanium implants influenced follow-up MRAs, the absence or presence of signal flow along the trail of the vertebral artery could be diagnosed by MRA because the metal artifacts existed only around the implants (Figures 2E, 3F). Postoperative computerized axial tomography showed no implant-related violation to the foramen transversarium, which can result in interference of arterial flow in all patients (Figure 3G, available for viewing online through ArticlePlus only). Restoration of blood flow

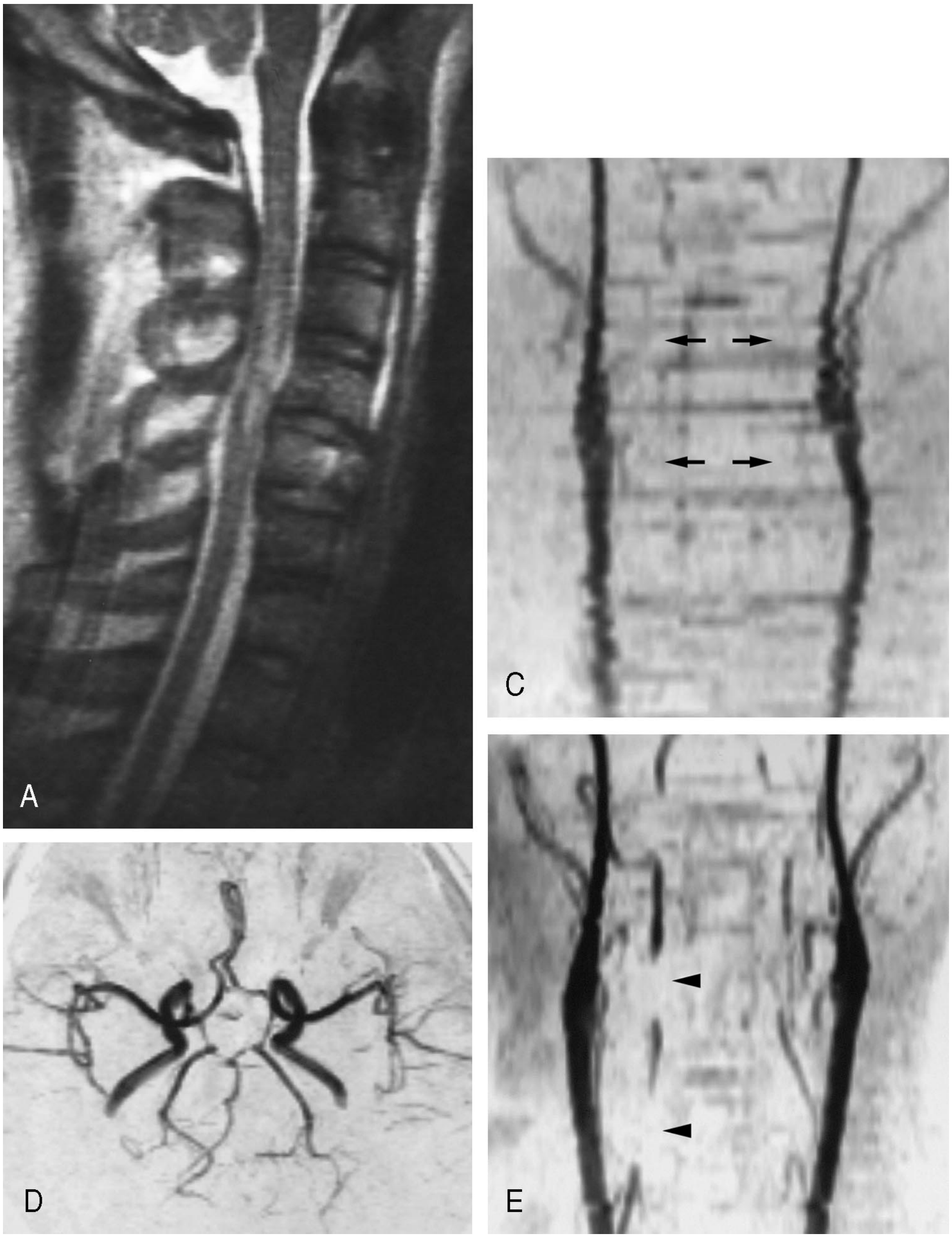

Figure 2. **A**, Sagittal T2-weighted MRI of a 34-year-old man (patient No. 9) obtained after burst fracture of C5 caused by compressive flexion mechanism shows severe intramedullary lesion. **C**, Initial MRA shows complete occlusion of the bilateral vertebral artery (arrows). **D**, Intracranial MRA shows complete configuration of the circle of Willis. **E**, MRA obtained 1 month after surgical stabilization with instrumentation shows recanalization of the bilateral vertebral artery. Signal of the vertebral artery is partially interrupted by the metal artifact of the implants (arrowheads).

in the occluded vertebral artery could be confirmed in 3 of the 11 patients, with occlusion at an average of 5.0 months after injury (range 1–11) (Figures 2, 3; Figures 2B, 3D, 3E, 3G, and 3H are available for viewing online through ArticlePlus only). In the other 8 patients, no signal flow could be recognized along the vertebral artery in the expected location on follow-up MRAs (Figure 4; Figures 4C to 4E are available for viewing online through ArticlePlus only). Injury types of the 3 patients in whom blood flow was restored were compressive flexion in 2 and compressive extension in 1. On the other hand, in all 6 patients who had both vertebral artery occlusion and distractive flexion injury, occlusion remained unchanged on the follow-up MRAs. No adverse consequence associated with restoration of blood flow, such as distal embolization, occurred during the follow-up. MRI of the brain, which was obtained in the patients with vertebral artery occlusion did not show any abnormal findings related to vertebrobasilar ischemia.

Discussion

The incidence of traumatically induced vertebral artery occlusion associated with nonpenetrating cervical spine fractures and/or dislocations was 17.2% in the current prospective study. In the past, this lesion was thought to be infrequent because it was rarely symptomatic, and conventional angiographic screening has rarely been performed because of its inherent risks. Thereafter, with the popularization of noninvasive MRA, several prospective studies using MRA were conducted to determine the incidence of this lesion.[4,6–8,12] The incidence derived from these clinical series was within a narrow range between 18.9% and 25.5%,[4,6,7,12] which is comparable to that

Figure 3. **A**, Sagittal T2-weighted MRI of 49-year-old man (patient No. 8) obtained after compressive flexion injury of C4–C6 segments shows severe compression of the spinal cord. **B**, Sagittal reformatted computerized tomography shows teardrop fracture of C5 and posterior translation of the fractured vertebral body. **C**, Initial MRA shows complete occlusion of the right vertebral artery (arrows). **F**, MRA obtained 11 months after surgery shows recanalization of the right vertebral artery. Signal of the right vertebral artery is partially interrupted by metal artifacts of screws (arrowheads).

found in our results. Vertebral artery occlusion can be considered a common complication in approximately 20% of cervical spine fractures and/or dislocations.

Anatomically, the vertebral artery is divided into 4 segments: first, subclavian to entrance to the foramen transversarium (C6 or C7); second, C6–C1, in which the vertebral arteries pass through the foramen transversarium; third, C1 to entrance into the dura; and fourth, entrance to its termination in the basilar artery (Figure 1).[3] The most common site of vertebral artery injury associated with cervical spine trauma was the second segment.[13] Vertebral artery injury generally occurs either through excessive distraction and stretching of the vessels between 2 adjacent foramina transversia (as observed in facet dislocation)[2,3,5,7,8] or through direct trauma to the vessel wall (as observed in fractures involving the articulating facet or the foramen transversarium).[5,14] Previously, hyperextension injuries had been accepted as the most common mechanism of vertebral artery injury.[15,16] However, recent reports have identified distractive flexion injury as the main cause of this lesion.[4,5,7,8,17]

The reported incidence of vertebral artery occlusion in distractive flexion injury was 75% by Louw,[17] 28% by Giacobetti[7] *et al*, and 29% by the current authors. Facet dislocation, especially the unilateral one, was closely related to this lesion.[4,7,8,17] In the present study, all pa-

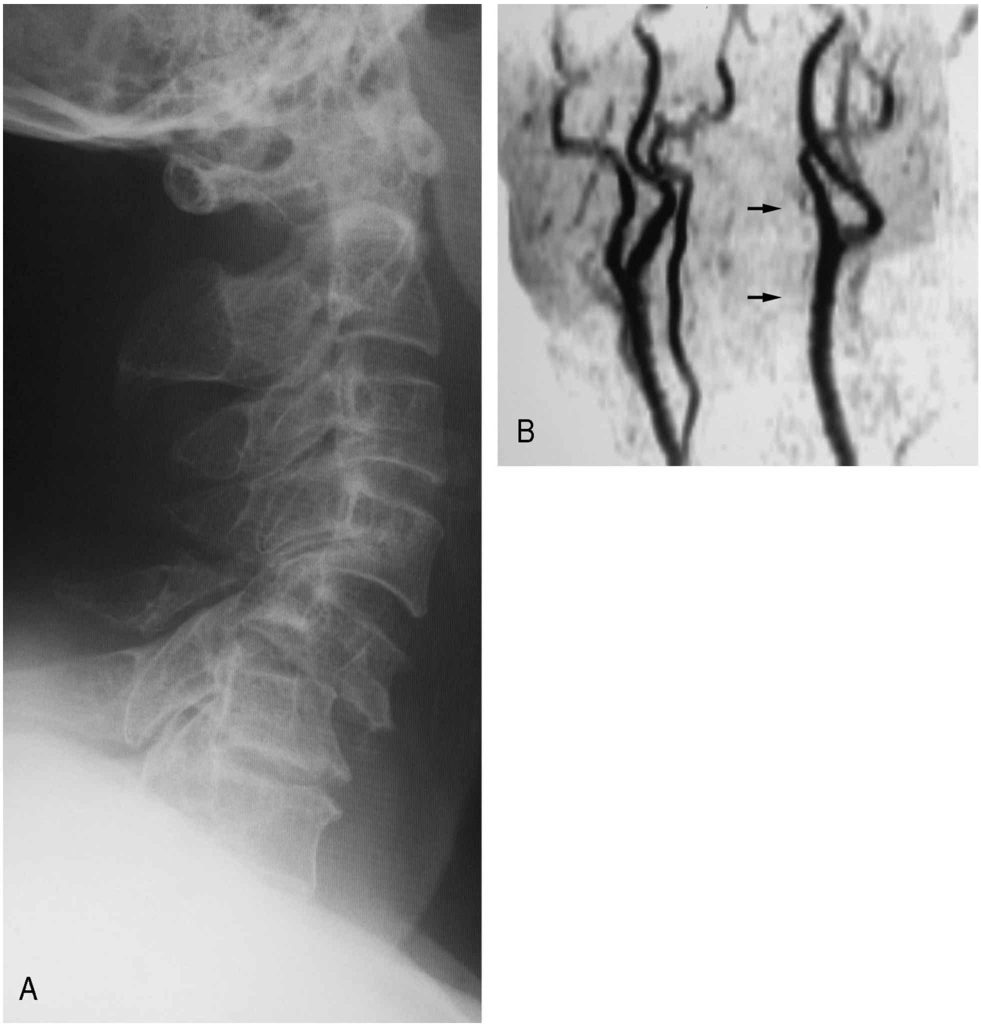

Figure 4. **A**, Lateral radiograph of a 55-year-old man (patient No. 6) who had destructive flexion injury of C5–C6 with unilateral facet dislocation. **B**, MRA obtained 6 hours after injury shows complete occlusion of the left vertebral artery (arrows).

tients with vertebral artery occlusion had traumatic spinal cord lesion. However, vertebral artery occlusion without spinal cord injury was reported in the literature.[4,7,8] There was no significant correlation between vertebral artery occlusion and severity of spinal cord injury in this study.

Vertebral artery injury secondary to cervical spine trauma includes a wide spectrum of vessel damages, such as intimal dissection, aneurysm, intramural or subintimal hematomas, and occlusion. The most common lesion shown in previous studies using varying imaging methods was occlusion.[5,6] A pathomechanism of traumatic vertebral artery occlusion is: intimal disruption initially occurs, then secondary events such as thrombus formation may lead to clot occlusion of vessel lumen.[4,5]

The current study was the first report in which all the patients with vertebral artery occlusion could be prospectively followed up. Restoration of blood flow was observed in 3 of the 11 patients with occlusion, and all 3 had compressive injuries. On the other hand, no patients with distractive flexion injury had recanalization. The mechanism of occlusion in a compressive injury is likely to be vasospasm or minor artery dissection, which may cause reversible occlusion,[18,19] because, comparatively, vessels are subjected to less severe stretching in compressive injuries than in distractive injuries. Flow disturbance caused by vasospasm can be restored within a short period.[4] Furthermore, occlusion secondary to vertebral artery dissection can be recanalized up to 85% within 3 months by spontaneous mechanism, which could have depended on the intrinsic condition of the vessels.[18] Therefore, the potential for restoration of blood flow may be higher in compression injuries than in distraction injuries. Vaccaro *et al*[4] reported that reconstitution of flow occurred in 1 of 6 patients with vertebral artery occlusion and that this patient also had a burst fracture caused by compressive flexion mechanism. In the current study, the restoration rate of blood flow may be underestimated because follow-ups of MRA were not long enough (*e.g.*, 1 month) in some patients.

MRA is quite accurate in the detection of near or total occlusion of the extracranial neck vessels.[6,20,21] On the other hand, slow blood flow in the small vessels on MRA can be confused with occlusion as a result of insufficient resolution.[5,22] Although conventional angiography is a much more invasive modality, it is superior to MRA in the detection of nonoccluded intimal disruption, which occasionally causes distal embolization. However, because occlusion is the most common vertebral artery injury, the majority can be successfully detected by noninvasive MRA.[5,6]

Symptoms of vertebrobasilar ischemia include headaches, dizziness, vertigo, tinnitus, unsteady gait, dysarthrias, diplopia, visual field defect, blurry vision, ptosis, drowsiness, syncope, altered consciousness, nystagmus, and dysphagia.[4–8,17] A low frequency of vertebrobasilar ischemia in patients with cervical spine trauma has been shown in many published reports.[4–8,12,14,17,22] In our series, all the patients were also asymptomatic, except one who had transient blurry vision. However, it is also apparent that vertebrobasilar ischemia can have devastating consequences (mortality rate 75% to 86%);[23] this highlights the value of a noninvasive screening test.[2,4,23–27] Unilateral occlusion of the vertebral artery rarely results in a neurologic deficit because of sufficient collateral blood supply through the contralateral vertebral artery.[4–6,13,17] Whereas, approximately 15% of patients have hypoplasia of one vertebral artery, which emphasizes the fact that there may not always be sufficient collateral arterial supply in a patient with unilateral occlusion.[14,17]

There are other potential sources of collateral circulation to the vertebrobasilar territory, such as the posterior communicating arteries, which are the important elements of the circle of Willis,[28] the posterior inferior cerebellar arteries,[29] distal branches of the thyrocervical and costocervical trunks, the occipital artery, interspinal branches, and muscular branches.[13,30,31] The circle of Willis plays an important role in the collateral pathway between the anterior and posterior circulation of the brain.[28] In patients with a complete circle of Willis, sufficient collateral circulation is present even when bilateral vertebral artery occlusion occurs.[10,28] In the present study, the patient with bilateral occlusion had no severe or persistent symptoms of vertebrobasilar ischemia. A possible explanation for this phenomenon is the existence of sufficient collateral blood flow from anterior circulation *via* the posterior communicating artery and/or early restoration of blood flow in the occluded artery.

■ Conclusions

The incidence of traumatically induced vertebral artery occlusion associated with nonpenetrating cervical spine injuries was 17.2%. The potential for restoration of blood flow in occluded vessel(s) was higher in compressive injuries than in distractive injuries. The mechanism of occlusion in compressive injuries is likely to be vasospasm or minor artery dissection, which may cause reversible occlusion because vessels are subjected to relatively minor stretching in this type of injury. Vertebral artery occlusion was rarely symptomatic because of sufficient collateral blood supply through not only the contralateral vertebral artery but also the circle of Willis.

■ Key Points

- The incidence of vertebral artery occlusion secondary to cervical spine fractures and dislocations determined by MRA was 17.2%.
- Restoration of blood flow in occluded vessel(s) occurred in 3 of 11 patients with vertebral artery occlusion, and all of them had compressive injuries.
- The potential for blood flow restoration was higher in compressive injuries than in distractive injuries. The mechanism of occlusion in a compressive injury is likely to be vasospasm or minor artery dissection, which may cause reversible occlusion because vessels are subjected to relatively minor stretching.
- Vertebral artery occlusion was rarely symptomatic because of sufficient collateral blood supply through not only contralateral vertebral artery but also the circle of Willis.
- One patient who had bilateral vertebral artery occlusion and sufficient collateral blood supply from anterior circulation of the brain through the patent posterior communicating arteries had no permanent neurologic deficit.

References

1. Deen HG, McGirr SJ. Vertebral artery injury associated with cervical spine fracture. *Spine* 1992;17:230–4.
2. Miyachi S, Okamura K, Watanabe M, et al. Cerebellar stroke due to vertebral artery occlusion after cervical spine trauma. Two case reports. *Spine* 1994;19:83–8.
3. Parent AD, Harkey HL, Touchstone DA, et al. Lateral cervical spine dislocation and vertebral artery injury. *Neurosurgery* 1992;31:501–9.
4. Vaccaro AR, Klein GR, Flanders AE, et al. Long-term evaluation of vertebral artery injuries following cervical spine trauma using magnetic resonance angiography. *Spine* 1998;23:789–94.
5. Willis BK, Greiner F, Orrison WW, et al. The incidence of vertebral artery injury after midcervical spine fracture or subluxation. *Neurosurgery* 1994; 34:435–42.
6. Friedman D, Flanders A, Thomas C, et al. Vertebral artery injury after acute cervical spine trauma: Rate of occurrence as detected by MR angiography and assessment of clinical consequences. *AJR Am J Roentgenol* 1995;164: 443–7.
7. Giacobetti FB, Vaccaro AR, Bos-Giacobetti MA, et al. Vertebral artery occlusion associated with cervical spine trauma. A prospective analysis. *Spine* 1997;22:188–92.
8. Veras LM, Pedraza-Gutierrez S, Castellanos J, et al. Vertebral artery occlusion after acute cervical spine trauma. *Spine* 2000;25:1171–7.
9. Allen BL, Ferguson RL, Lehmann TR. A mechanistic classification of closed, indirect fractures and dislocations of the lower cervical spine. *Spine* 1982;7: 1–27.
10. Hartkamp MJ, Grond JVD, Everdingen KJV, et al. Circle of Willis collateral flow investigated by magnetic resonance angiography. *Stroke* 1999;30: 2671–8.
11. Hoksbergen AWJ, Legemate DA, Csiba L, et al. Absent collateral function of the Circle of Willis as risk factor for ischemic stroke. *Cerebrovasc Dis* 2003; 16:191–8.
12. Parbhoo AH, Govender S, Corr P. Vertebral artery injury in cervical spine trauma. *Injury* 2001;32:565–8.
13. Prabhu V, Kizer J, Patil A, et al. Vertebrobasilar thrombosis associated with nonpenetrating cervical spine trauma. *J Trauma* 1996;40:130–7.
14. Weller SJ, Rossitch E, Malek AM. Detection of vertebral artery injury after cervical spine trauma using magnetic resonance angiography. *J Trauma* 1999;46:660–6.

15. Simeone FA, Goldberg HI. Thrombosis of the vertebral artery from hyperextension injury to the neck. *J Neurosurg* 1968;29:540–4.
16. Six EG, Stringer WL, Cowley AR, et al. Posttraumatic bilateral vertebral artery occlusion: Case report. *J Neurosurg* 1981;54:814–7.
17. Louw JA, Mafoyane NA, Small B, et al. Occlusion of the vertebral artery in cervical spine dislocation. *J Bone Joint Surg* 1990;72-B:679–81.
18. Caso V, Paciaroni M, Corea F, et al. Recanalization of cervical artery dissection: Influencing factors and role in neurological outcome. *Cerebrovasc Dis* 2004;17:93–7.
19. Pelkonen O, Tikkakoski T, Leinonen S, et al. Extracranial internal carotid and vertebral artery dissections: Angiographic spectrum, course and prognosis. *Neuroradiology* 2003;45:71–7.
20. Bowen BC, Quencer RM, Margosian P, et al. MR angiography of occlusive disease of the arteries in the head and neck: Current concepts. *AJR Am J Roentgenol* 1994;162:9–18.
21. Heiserman JE, Drayer BP, Fram EK, et al. Carotid artery stenosis: Clinical efficacy of two-dimensional time-of-flight MR angiography. *Radiology* 1992;182:761–8.
22. Rodriguez M, Tyberghien A, Matge G. Asymptomatic vertebral artery injury after acute cervical spine trauma. *Acta Neurochir (Wien)* 2001;143:939–45.
23. Becker KJ, Monsein LH, Ulatowski J, et al. Intraarterial thrombolysis in vertebrobasilar occlusion. *AJNR Am J Neuroradiol* 1996;17:255–62.
24. Brandt T, Kummer RV, Mueller-Kueppers M, et al. Thrombolytic therapy of acute basilar artery occlusion. *Stroke* 1996;27:875–81.
25. Cross III DT, Moran CJ, Akins PT, et al. Relationship between clot location and outcome after basilar artery thrombolysis. *AJNR Am J Neuroradiol* 1997;18:1221–8.
26. Schellinger PD, Schwab S, Krieger D, et al. Masking vertebral artery dissection by severe trauma of the cervical spine. *Spine* 2001;26:314–9.
27. Wirbel R, Pistorius G, Braun C, et al. Bilateral vertebral artery lesion after dislocating cervical spine trauma. A case report. *Spine* 1996;21:1375–80.
28. Wentz KU, Roether J, Schwartz A, et al. Intracranial vertebrobasilar system: MR angiography. *Radiology* 1994;190:105–10.
29. Golueke P, Sclafani S, Phillips T. Vertebral artery injury–Diagnosis and management. *J Trauma* 1987;27:856–65.
30. Faillace WJ, Okawara SH. Pontomedullary infarction from sustained cervical spine hyperflexion. *Surg Neurol* 1986;26:282–6.
31. Jabre A. Subintimal dissection of the vertebral artery in subluxation of the cervical spine. *Neurosurgery* 1991;29:912–5.