Original Article

Vertebral artery injury after cervical spine trauma: A prospective study using computed tomographic angiography

Jae-Won Jang1, Jung-Kil Lee1,2, Hyuk Hur1, Bo-Ra Seo1, Jae-Hyun Lee1, Soo-Han Kim1

1Department of Neurosurgery, Chonnam National University Medical School and Research Institute of Medical Sciences, 2The Brain Korea 21 Project, Center for Biomedical Human Resources at Chonnam National University, Gwangju, Republic of Korea

E-mail: Jae-W on Jang - genius60@hanmail.net; *Jung Kil Lee - jkl@chonnam.ac.kr; Hyuk Hur - mrc38@hanmail.net; Bo-Ra Seo - cd1379@lycos.co.kr; Jae-Hyun Lee - mute94@hanmail.net; Soo-Han Kim - soohan@chonnam.ac.kr

*Corresponding author

Abstract

Background: Although the vertebral artery injuries (VAI) associated with cervical spine trauma are usually clinically occult, they may cause fatal ischemic damage to the brain stem and cerebellum.

Methods: We performed a prospective study using computed tomographic angiography (CTA) to determine the frequency of VAI associated with cervical spine injuries and investigate the clinical and radiological characteristics. Between January 2005 and August 2007, 99 consecutive patients with cervical spine fractures and/or dislocations were prospectively evaluated for patency of the VA, using the CTA, at the time of injury.

Results: Complete disruption of blood flow through the VA was demonstrated in seven patients with unilateral occlusion (7.1%). There were four men and three women with a mean age of 43 (range, 33-55 years). Unilateral occlusion of the right vertebral artery occurred in four patients and of the left in three. Regarding the cervical injury type, two cases were cervical burst fractures (C6 and C7), two had C4-5 fracture/dislocations, two had a unilateral transverse foraminal fracture, and one had dens type III fracture. All patients presented with good patency of the contralateral VA. None of the patients developed secondary neurological deterioration due to vertebrobasilar ischemia during the follow-up period with a mean duration of 23 months.

Conclusions: VAI should be suspected in patients with cervical trauma that have cervical spine fractures and/or dislocations or transverse foramen fractures. CTA was useful as a rapid diagnostic method for ruling out VAI after cervical spine trauma.

Key Words: Cervical spine, computed tomographic angiography, injury, vertebral artery
INTRODUCTION

Vertebral artery injuries (VAI) associated with cervical spine trauma most frequently involve the second segment that runs through the transverse foramen of C6 to C1. The fixation of the VA within the transverse foramen makes the second segment more vulnerable to injury caused by cervical spine fractures and/or dislocations or transverse foramen fractures.\[10,27\] The unilateral occlusion of the VA, especially the nondominant VA, may not cause ischemic symptoms. Therefore, the injury may be underestimated in asymptomatic patients.\[21,26\] Many clinical studies have reported that VAI, after cervical spine trauma, occurs more frequently than previously thought.\[3,6,10,15,18,24,25\] The outcome following VA occlusion is dependent on adequate perfusion of the contralateral vertebral artery. If the collateral flow is inadequate, an occlusion may cause vertebrobasilar ischemia. Approximately 15% of patients have hypoplasia of one VA; in such cases there may not be sufficient collateral arterial supply if a patient develops a unilateral occlusion.\[14\] Only 12-20% of patients with unilateral VAI initially have presented with symptoms and signs of vertebrobasilar ischemia.\[3,11\] VAI, although often initially asymptomatic, may have devastating consequences.\[19\] Therefore, the clinician must be aware of the potential morbidity and mortality following VAI.

Although angiography remains the gold-standard, its role as a screening study has been questioned due to its invasiveness. A reliable less invasive and inexpensive screening modality would be preferred. Currently, computed tomographic angiography (CTA) has been increasingly used for screening for VAI.\[12,16\] We performed a prospective study using CTA to determine the frequency of VAI following cervical spine injuries, and to investigate the clinical features, including long-term neurologic outcomes of the affected patients.

MATERIALS AND METHODS

All patients admitted or transferred to our department due to suspicion of cervical spine injuries between January 2005 and August 2007 were considered for CTA evaluation as part of a prospective study. Inclusion criteria for this prospective analysis were: (1) injury to the cervical spine with radiographically evident fractures and/or dislocations; (2) spinal injury occurring between the level of C2 vertebra and the C 6/7 spinal segment, consistent with the second segment of the vertebral arteries. A total of 99 consecutive patients met the eligibility requirements during the time frame of the study. Plain anteroposterior and lateral radiographs were assessed in all patients. In cases of suspected fractures and equivocal findings, CT scans were obtained, followed by immediate investigation of the VA with a helical CTA. All scans from January 2005 to December 2005 were obtained on a General Electric Lightspeed Four scanner with four slices per rotation. Starting in January 2006, a 64-detector row CTA was used. Both the axial cuts and the sagittal reconstructions were read by a radiologist. If a CTA study was read as negative, no further studies were ordered. If a study was suspicious or strongly suggestive of VAI, angiography was performed. An occlusion was strictly defined as a complete disruption in the blood flow of the second portion of the VA. Regarding the neurological examination, not only specific findings for spinal cord injury but signs or symptoms of vertebrobasilar ischemia were carefully followed in all patients. Early reduction and surgical stabilization were performed when clinical and radiographic evaluations were completed. In cases with VAI, for reducing of the damage to the uninjured VA by lateral mass screws or pedicle screws, if possible, we preferred anterior cervical fusion as a primary surgical plan for unstable cervical injury.

RESULTS

Occlusion of the VA was demonstrated by CTA in 7 out of the 99 patients (7.1%). There were four men and three women with a mean age of 43 (range, 33-55 years). The average follow-up was 23 months (range, 9-36 months). The causes of injury were motor vehicle accidents in three patients, falls in three patients and an unknown etiology in one patient (found in the street with abnormal mental status). Table 1 summarizes the findings of the seven patients with VA occlusion.

The CTA showed unilateral VAI in the seven cases. Unilateral occlusion of the right vertebral artery occurred in four patients and of the left in three. All patients presented with good patency of contralateral VA. None of the patients had an associated bilateral VA and carotid injury. Angiography was performed in six patients with the exclusion of case 4. Neurological examination, using the American Spinal Injury Association (ASIA) impairment scale, was grade A in three patients, D in one, and E in three [Table 2]. Regarding to the injury type to the cervical spine, there were two cases with a cervical burst fracture (C6 and C7), 2 with C4-5 fracture/dislocation, two with unilateral transverse foraminal fracture, and one with dens type III fracture. The level of the spine injuries were: the C2 segment in one patient, C4-5 in two, C5 in one, C6 in two, and C7 in one. Case 3 was illustrated on Figures [1-6]. Routine brain CT scanning was performed on admission and revealed an epidural hematoma with fracture of the skull in one out of the seven patients; this patient was found in the street with an altered mental status (case 4). Except for case 4, conventional angiography was performed on the basis of the CTA findings and
Table 1: Summary of patients with vertebral artery injuries

| Case No. | Age (yr)/Gender | Cause of injury | Cervical spine injury | Level of injury | Injury classification | Primary force vector | Side of VA injury | VA Sx. Initial | VA Sx. F/U |
|----------|-----------------|-----------------|-----------------------|-----------------|-----------------------|---------------------|-----------------|---------------|-----------|
| 1        | 42/F            | MVA             | Fracture/dislocation  | C4-5            | Flexion/Distraction   | Flexion             | Right           | None          | None       |
| 2        | 49/F            | Fall            | Burst fracture        | C7              | Vertical Compression  | Compression         | Right           | None          | None       |
| 3        | 40/F            | Fall            | Fracture/dislocation  | C4-5            | Flexion/Distraction   | Flexion             | Left            | None          | None       |
| 4        | 36/M            | Fall            | Burst fracture        | C6              | Flexion/Compression   | Flexion             | Right           | None          | None       |
| 5        | 33/M            | Fall            | Fracture/dislocation  | C6              | Lateral Flexion       | Flexion             | Left            | None          | None       |
| 6        | 46/M            | Fall            | Type III Dens fracture| C2              | Flexion/Compression   | Flexion             | Right           | None          | None       |
| 7        | 55/M            | MVA             | Foramen fracture      | C5              | Lateral Flexion       | Flexion             | Left            | None          | None       |

F/U: Follow-up, MVA: Motor vehicle accident, VA: Vertebral artery

Table 2: Summary of treatments and outcomes

| Case No. | Treatment                          | Neurologic exam. | Frankel grade | Antiplatelet | Last F/U (months) |
|----------|------------------------------------|------------------|---------------|--------------|------------------|
|          |                                    | Initial/F/U      | Initial/F/U   |              |                  |
| 1        | C4-5 post. wiring & lat. mass SF   | Quadriplegia/FU   | A/A           | Aspirin      | 24               |
| 2        | C7 corpectomy & IF                 | Intact/FU        | E/E           | Aspirin      | 36               |
| 3        | C4-5 discectomy & IF               | Quadriplegia/FD   | A/D           | Aspirin      | 30               |
| 4        | C6 corpectomy & IF                 | Quadriplegia/FU   | A/A (-)       | Aspirin      | 9                |
| 5        | Conservative treatment             | Monoparesis/FU    | D/E           | Aspirin      | 26               |
| 6        | Unilateral C1-2 post. TASF & wiring| Intact/FU        | E/E           | Aspirin      | 21               |
| 7        | Conservative treatment             | Intact/FU        | E/E           | Aspirin      | 17               |

F/U: Follow-up, IF: Interbody fusion, TASF: Transarticular Screw fixation, post.: posterior

demonstrated the same diagnostic results as the CTA. Four patients presented with neurological deficits due to cervical cord injury. No patient had clinical signs of vertebrobasilar ischemia. Five patients underwent surgery, within 3 days after the trauma for early reduction and fusion, and two patients had conservative treatment with a soft collar brace. There was no operative mortality and no permanent sequelae from any perioperative complication. For protection of the vertebrobasilar circulation, intra-venous heparin was not administered. All but one patient (in whom traumatic intracranial hemorrhage was combined) were treated with an oral...
in one patient with C2 dens fracture. In this case, C1-2 transarticular screw was inserted unilaterally in site of VAI to avoid the additional damage of uninjured VA.

DISCUSSION

The frequency of VAI among all blunt trauma admissions has been reported to range from 0.20% to 0.77%.\cite{1,6,15} However, the incidence of VAI is much higher in selected subpopulations, such as patients with blunt cervical spine trauma. Miller et al.\cite{15} and Biffl et al.\cite{3} screened all patients with cervical spine fractures prospectively with four vessel angiography, and reported that VAI occurred in 33% and 39% of cases, respectively. Three types of cervical fractures are cited in the literature as significant risk factors for VAI: (1) fractures involving a transverse foramen, (2) subluxation, and (3) fractures involving antiplatelet agent (acetylsalicylic acid) for 3 months. In case 4, the patient underwent two cranial surgeries for bilateral epidural hematomas, one surgical stabilization of the cervical spine, and then tracheostomy due to recurrent atelectasis. No side effects were attributed to the acetylsalicylic acid. None of the seven patients had VAI-related complications and all survived.

Anterior cervical fusion was performed in three of five patients treated with surgical intervention. In one patient who failed with preoperative closed reduction, surgical intervention via posterior approach was planned. Firstly, the open reduction via a posterior cervical approach was firstly performed. Preoperative CTA showed the enough space of the lateral mass for the screw insertion; therefore, posterior lateral mass screw fixation and fusion were performed in this case. C1-2 fusion was required...
the upper cervical spine (C1-C3). Gothen et al. reported a 37% frequency of VAI in patients with these three fractures, and suggested that a screening protocol using these three fracture patterns would detect 95% of VAI. The frequency of VAI derived from other clinical series ranged between 13.2% and 26%. We found a lower rate, 7.1%, of VAI in 99 consecutive patients with blunt cervical spinal injury assessed by CTA. CTA tends to underestimate stenosis, and the ability of the spiral CTA to show stenosis has been reported to be limited, less than 30%. Bill et al. suggested a VAI grading scale according to angiographic findings, which had been firstly developed for blunt carotid artery injury: grade I, dissection with less than 25% luminal narrowing; grade II, dissection with more than 25% luminal narrowing; grade III, pseudoaneurysm; grade IV, occlusion; and grade V, transaction. Thus, grade I injuries could have been missed in our series. One case with right side VAI was associated with left dominant C7 burst fracture and lamina fracture. In this case, we thought that main injury mechanism was the vertical compression of left side of the C7, and contralateral VAI with dissection type might be injured due to retraction force although VA is located extraforaminal space in C7 level.

Most cases of VAI usually remain asymptomatic and therefore are easily overlooked. In particular, non-dominant unilateral VA occlusion rarely results in neurological deficits because of adequate collateral circulation to the basilar system through the contralateral VA and the posterior inferior cerebellar arteries. In addition, when head injury is combined, these symptoms can be missed or misinterpreted. Clinical symptoms related to vertebrobasilar ischemia include vertigo and nystagmus, dysphagia and dysarthria, diplopia or blurred vision, and altered consciousness. The symptoms usually manifest within the first 24 h after trauma. Occasionally the lucid interval between injury and onset of neurological symptoms may be delayed further, up to 3 months. Bilateral or dominant VA occlusion have devastating consequences, and these include an altered mental status, pinpoint pupils, and sudden respiratory arrest, resulting in a high morbidity and mortality rate. Overall, the mortality in patients with VAI, those without neurological event, had 7% mortality, whereas those with a neurological event had 18% mortality. The primary therapeutic goal of treatment of VAI should not concentrate on vessel recanalization, but on reconstruction of the unstable spine segment to maintain flow of the opposite VA and minimize thrombus propagation in the injured vessel. Early surgical stabilization to release tension on the VA wall and prevent future ischemic events may be sufficient for stable and asymptomatic patients. Heparin or antiplatelet agents have been used for the treatment of patients with asymptomatic VAI; aggressive and early treatment has been reported to result in a significant reduction of the development of vertebrobasilar strokes later. Heparin was initially the treatment of choice for blunt VAI. In patients with contraindications to heparin, the initiation of antiplatelet agents has been successfully used. Although the optimal regimen remains unknown, both therapies appear to be effective. Many trauma patients with VAI have other multiple organ system injuries, and anticoagulation using heparin increases the risk of hemorrhagic complications. Therefore, aspirin might be more useful agent than heparin for the prevention of an infarct in patients with VAI and multiple organ injuries. In this study, none of the patients with unilateral VAI had symptoms associated with vertebro-basilar ischemia. We did not use intra-venous anticoagulation, but oral antiplatelet agents were administered for protection of the vertebrobasilar circulation in six out of the seven patients. During the follow-up period, a mean duration of 23 months, vertebro-basilar insufficiency was not detected in any of our patients. The limitation in this study is that collateral circulation to the basilar system via the contralateral VA was maintained adequately in all seven cases with VAI and short follow-up duration. Therefore, to determine the efficacy of antiplatelet agents, randomized controlled study will be required with a large number of VAI cases and long-term follow-up duration.

There has been no consensus on the optimal diagnostic modality for patients with suspected of VAI. Angiography is the most accurate method for the definition of VAI as well as collateral flow. However, it is not practical as a screening method due to its invasiveness and risk of complications. Angiography is risky in critically ill patients with unstable spines. Many investigators have suggested that angiography should be reserved for patients in whom: (1) a VAI is strongly suspected due to neurological symptoms, (2) neuroradiological intervention is necessary, or (3) the results of other imaging studies are equivocal. Magnetic resonance angiography (MRA) is less sensitive for the diagnosis of VAI than angiography. However, it has been frequently used to screen and diagnose patients with VAI that have cervical spine fractures and/or dislocations as an alternative diagnostic modality. MRA is noninvasive, does not require the injection of contrast and allows imaging of all cervical and cerebral vasculature. However, slow blood flow on the MRA could be confused with occlusion, where angiography would demonstrate a high degree of stenosis. Furthermore, many ventilator and orthopedic external fixation devices are incompatible with the scanning equipment, obviating its use in patients with multisystem severe injuries.

The CTA may provide the screening tool of choice although its diagnostic value remains to be determined. With the CTA, the VA within the transverse foramen may
be difficult to image accurately because of bony artifacts. However, the CTA is noninvasive, examination times are relatively short and the imaging can be manipulated in three dimensions. In addition, the use of CT scanning for patients with cervical spine injuries has become so routine that it takes little additional effort to add CTA, and this minimizes transport needs. The multidetector CTA provides markedly improved vascular images for the identification of VAI in a timely manner. CTA can also provide the information for the relations between the VA and adjacent bony structure. Therefore, it is the most valuable for preoperative surgical planning, for example, how the dislocation of the cervical spine or the displaced VA should be managed without additional injuries. Although dilated or tortuous VA and anomalous formation of transverse foramen were not observed in our series, through the result of the preoperative CTA, we planned for the degree of lateral exposure of neural foramen during anterior cervical fusion for avoiding the damage of uninjured VA. If possible, we avoid posterior cervical screw techniques; however if needed, optimal insertion site and direction of posterior screws were preoperatively planned according to the result of CTA. As the results, spine surgeon can avoid or reduce unexpected damage of uninjured VA, consequently, the morbidity and mortality of patients with VAI will be reduced.

Those patients with cervical spine injuries that are at high risk, such as those with transverse foramen fractures, dislocation, or high cervical injuries should undergo CTA promptly as screening examination. CTA enables rapid selection of patients for angiography, so that prophylactic therapy can be promptly started to minimize ischemic neurological events. The possible complications of dominant or bilateral vertebral artery occlusion, resulting from cervical spine trauma, have to be excluded as early as possible by angiography, in patients with dubious or vague neurological symptoms. The awareness of a VAI in patients with unstable fractures is very useful at the time of surgery. Embolic events can occur, with various extension of the vertebro-basilar infarction, based on the collateral circulation, during surgical manipulation and reduction of a vertebral dislocation. Obviously, excessive distraction must be avoided during an operation. Early and late emboli are the major risk factors for neurological morbidity and outcome. All patients with VAI should be informed of the associated risks and followed for several months to screen for signs of late ischemic complications that can occur several weeks after the initial trauma.

In conclusion, VAI after blunt cervical spine fractures is more frequent than once thought and should be kept in mind while evaluating trauma patients. Although the diagnostic accuracy of CTA remains to be confirmed, the results of this study showed that CTA was rapid and easy to perform for the screening of at risk patients to rule out VAI. Aggressive screening has been shown to identify injuries in asymptomatic patients, allowing prompt antithrombotic treatment to prevent ischemic neurological events. By facilitating the early diagnosis and treatment of VAI, screening with CTA might improve the clinical outcomes of affected patients. Clear treatment guidelines remain to be determined; however, prophylactic antiplatelet therapy might be necessary for asymptomatic VAI. Further multicenter trials are essential to determine the safest and most effective treatment guidelines not only in symptomatic patients but also in asymptomatic patients with VAI.

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REFERENCES

1. Berne JD, Norwood SH, McAuley CE, Villareal DH. Helical computed tomographic angiography: An excellent screening test for blunt cerebrovascular injury. J Trauma 2004;57:11-7.
2. Biffi WL, Eggin T, Benedetto B, Gibbs F, Cioffi WG. Sixteen-slice computed tomographic angiography is a reliable noninvasive screening test for clinically significant blunt cerebrovascular injuries. J Trauma 2006;60:745-51.
3. Biffi WL, Moore EE, Elliott JP, Ray C, Offner PJ, Franciose RJ, et al. The devastating potential of blunt vertebral arterial injuries. Ann Surg 2000;231:672-81.
4. Biffi WL, Ray CE Jr, Moore EE, Mestek M, Johnson JL, Burch JM. Noninvasive diagnosis of blunt cerebrovascular injuries: A preliminary report. J Trauma 2002;53:850-6.
5. Bose B, Northrup BE, Osterholm JL. Delayed vertebo-basilar insufficiency following cervical spine injury. Spine 1985;10:108-10.
6. Cothren CC, Moore EE, Biffi WL, Ciesla D, Ray CE Jr, Johnson JL, et al. Cervical spine fracture patterns predictive of blunt vertebral artery injury. J Trauma 2003;55:811-3.
7. Cothren CC, Moore EE, Ray CE Jr, Ciesla DJ, Moore JB, et al. Screening for blunt cerebrovascular injuries is cost-effective. Am J Surg 2005;190:845-9.
8. Cothren CC, Moore EE, Ray CE Jr, Johnson JL, Moore JB, Burch JM. Cervical spine fracture patterns mandating screening to rule out blunt cerebrovascular injury. Surgery 2007;141:76-82.
9. Eastman AL, Chason DP, Perez CL, McAnulty AL, Minei JP. Computed tomographic angiography for the diagnosis of blunt cervical vascular injury: Is it ready for primetime? J Trauma 2006;60:925-9.
10. Giacobetti FB, Vaccaro AR, Bos-Giacobetti MA, Deeley DM, Albert TJ, Farmer JC, et al. Vertebral artery occlusion associated with cervical spine trauma: A prospective analysis. Spine 1997;22:188-92.
11. Inamasu J, Guiot BH. Vertebral artery injury after blunt cervical trauma: An update. Surg Neurol 2006;53:238-45.
12. Kurokawa Y, Yonematsu Y, Kano H, Sasaki T, Inaba K, Shigemori S, et al. The use of three-dimensional computed tomographic angiography in the accurate diagnosis of internal carotid artery aneurysms: Degree for expression of posterior communicating and anterior choroidal arteries. Comput Med Imaging Graph 2000;24:231-41.
13. Link J, Grossmann J, Graber M, Mueller-Huelsbeck S, Steffens JC, Brinkmann G, et al. Spiral CT angiography and selective digital subtraction angiography of internal carotid artery stenosis. AJNR Am J Neuroimaging 1996;17:89-94.
14. Louw JA, Mafoyane NA, Small B, Nesper CP. Occlusion of the vertebral artery in cervical spine dislocations. J Bone Joint Surg Br 1990;72:679-81.
15. Miller PR, Fabian TC, Croce MA, Caggiornos C, Williams JS, Vang M, et al. Prospective screening for blunt cerebrovascular injuries: Analysis of diagnostic modalities and outcomes. Ann Surg 2002;236:386-93.
16. Nunez DB Jr, Torres-Leon M, Munera F. Vascular injuries of the neck and thoracic inlet: Helical CT-angiographic correlation. Radiographics 2004;24:1087-98.

17. Papp Z, Patel M, Ashtari M, Takahashi M, Goldstein J, Maguire W, et al. Carotid artery stenosis: Optimization of CT angiography with a combination of shaded surface display and source images. AJNR Am J Neuroradiol 1997;18:759-63.

18. Parbhoo AH, Govender S, Corr P. Vertebral artery injury in cervical spine trauma. Injury 2001;32:565-8.

19. Parent AD, Harkey HL, Touchstone DA, Smith EE, Smith RR. Lateral cervical spine dislocation and vertebral artery injury. Neurosurgery 1992;31:501-9.

20. Prabhu V, Kzier J, Paul A, Hellbusch L, Taylor C, Leibrock L. Vertebrobasilar thrombosis associated with nonpenetrating cervical spine trauma. J Trauma 1996;40:130-7.

21. Rodriguez M, Tyberghien A, Matge G. Asymptomatic vertebral artery injury after acute cervical spine trauma. Acta Neurochir (Wien) 2001;143:939-45.

22. Rogers FB, Baker EF, Osler TM, Shackford SR, Wald SL, Vieco P. Computed tomographic angiography as a screening modality for blunt cervical arterial injuries: Preliminary results. J Trauma 1999;46:380-5.

23. Sim E, Vaccaro AR, Beratanovich A, Pienaar S. The effects of staged static cervical flexion-distraction deformities on the patency of the vertebral arterial vasculature. Spine 2000;25:2180-6.

24. Taneichi H, Suda K, Kajino T, Kaneda K. Traumatically induced vertebral artery occlusion associated with cervical spine injuries: prospective study using magnetic resonance angiography. Spine 2005;30:1955-62.

25. Vaccaro AR, Klein GR, Flanders AE, Albert TJ, Balderston RA, Cotler JM. Long-term evaluation of vertebral artery injuries following cervical spine trauma using magnetic resonance angiography. Spine 1998;23:789-94.

26. Veras LM, Pedraza-Gutierrez S, Castellanos J, Capellades J, Casanitjana J, Rovira-Canellas A. Vertebral artery occlusion after acute cervical spine trauma. Spine 2000;25:1171-7.

27. Willis BK, Greiner F, Orrison WW, Benzel EC. The incidence of vertebral artery injury after midcervical spine fracture or subluxation. Neurosurgery 1994;34:435-41.