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Sheppard , R., Kennedy , GEM., Nelson , A., Abdel Meguid, E., & Darwish , N. (2020). Vertebral artery injury in cervical spine fractures: a cohort study and review of the literature. *Ulster Medical Journal*, 89(2), 89-94. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7576392/>

Published in:
Ulster Medical Journal

Document Version:
Publisher's PDF, also known as Version of record

Queen's University Belfast - Research Portal:
[Link to publication record in Queen's University Belfast Research Portal](#)

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Clinical Paper

Vertebral Artery Injury in Cervical Spine Fractures: A Cohort Study and Review of the Literature

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Accepted: 17th February 2020

Provenance: externally peer-reviewed

Key words: cervical spine fracture; vertebral artery injury

ABSTRACT

BACKGROUND: The risk of vertebral artery injury (VAI) secondary to cervical spine fracture is increasingly recognised in the literature. The aim of this study was to determine the incidence of VAI amongst patients presenting to the Royal Victoria Hospital (Northern Ireland's regional trauma centre with emergency surgical spinal services) with acute cervical spine fractures, and to identify fracture patterns associated with the highest risk of VAI.

METHODS: A retrospective review of 1,894 computed tomography (CT) reports of patients who underwent imaging of their cervical spine and/or vertebral arteries over a 12-month period, from June 2018 to June 2019, was conducted.

RESULTS: Sixty-eight patients (3.59%) with a confirmed cervical spine fracture were identified. These patients had an age range of 18-97 years and included 39 males (57.4%) and 29 females (42.6%). The fractures were then classified according to the AOSpine Cervical Spine Fracture Classification. Of the 68 patients with a confirmed cervical spine fracture, five (7.35%) were diagnosed with VAI, all involving fractures of their upper cervical spine. Two involved fractures extending into the transverse foramen, two involved subluxation of the vertebrae and one involved both. In all five cases, these fractures resulted from high-energy injuries. Regarding management, the patients with VAI in this study were either monitored and given no specific treatment or treated medically with antiplatelet therapy. None underwent surgical intervention.

CONCLUSIONS: Fracture patterns associated with increased risk of VAI are fractures involving the upper cervical spine, fractures with associated subluxation, and fractures of the transverse process extending into the transverse foramen - urgent CT-angiography in these cases is recommended. Further work should develop a targeted set of criteria for screening for VAI in cervical spine fractures, with consideration of high-risk fracture patterns.

INTRODUCTION

Vertebral artery injury (VAI) is a potentially serious complication of cervical spine fractures. The artery is at high

risk due to its passage through the transverse foramina of the cervical vertebrae. The incidence of VAI in patients with blunt cervical spine trauma ranges from 0.53% to 39% in the literature.^{1,2,3} This wide variation in incidence is most likely due to differences in sample size and the imaging modality used, as well as patient selection bias. Recently, there has been a higher incidence of VAI reported, most likely due to advances in imaging technology.⁴

The types of cervical spine fractures most associated with vertebral artery damage are fractures of the transverse process extending into the foramen transversarium, upper cervical spine fractures involving C1-C3 and facet dislocations/subluxations.⁵ The mechanisms of injury involve direct impingement of the artery in the foramen, or stretching of the vertebral artery between adjacent vertebrae.⁶

Clinical symptoms of VAI may include dizziness, vomiting and vertigo due to ischaemia of the cerebellum, which is responsible for balance and coordination. Ischaemia of the primary visual cortex may result in visual disturbance and damage to the brainstem may result in focal weakness.⁷ Vertebral artery injury can have devastating consequences for patients, causing neurological deficits, stroke and death, although the majority of patients are initially asymptomatic.⁸ De Souza et al reported that 70% of cases showed neurological symptoms within the first 24 hours. Biffi et al reported a period of 18 hours between time of injury and neurological symptoms in 44% of cases.^{9,10} Explanations for this delay in symptoms include thrombus progression and progression of the vascular injury to a higher grade, such as a pseudoaneurysm or dissection.¹ The overall mortality of VAI is reported as 4% to 8%.⁹ Therefore, it is important to consider the possibility of VAI in patients presenting with cervical spine trauma. Early diagnosis will allow prompt management

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and improved patient outcomes.

Diagnostic techniques for VAI include Digital Subtraction Angiography (DSA), Computed Tomographic Angiography (CTA), Doppler Ultrasonography (USS) or Magnetic Resonance Angiography (MRA).¹¹

Regarding VAI management, treatment options range from observation to medical or surgical intervention. The choice of treatment depends on both the grade and site of the injury⁹, as well as associated injuries and bleeding risk. Medical management, involving the use of antiplatelets or anticoagulants, may decrease the risk of thromboembolic mechanisms, resulting in ischaemic events.¹² Endovascular intervention, including stenting, artery occlusion and embolization, may be indicated if medical therapy is contraindicated or has failed.¹³ Surgical intervention on the vertebral arteries is technically challenging and tends to be reserved for patients unsuitable for anticoagulation, who have failed with endovascular options or who have uncontrollable haemorrhage.^{9,11}

The aim of this retrospective cohort study was to determine the incidence, clinical features and management of VAI amongst patients with cervical spine fractures presenting to Northern Ireland's regional trauma centre, and to identify fracture patterns most commonly associated with VAI.

MATERIALS AND METHODS

Study population

A retrospective review was carried out involving patients who presented to the Royal Victoria Hospital (RVH) Emergency Department (ED) and underwent subsequent CT imaging of their cervical spine and/or CTA of their vertebral arteries, arranged by the ED. The study involved patients who presented over a 12-month time period, from June 2018 to June 2019. The CT reports of 1,894 patients who underwent a CT scan of their cervical spine or a CTA of their vertebral arteries in this time period were obtained. The reports were then reviewed to determine the number of patients who had a confirmed cervical spine fracture. Of these 1,894 patients, 68 were found to have an acute cervical spine fracture. Patients without a confirmed fracture were then excluded from further study.

Information was collected regarding patient sex, age at injury, mechanism of injury, type of fracture and whether subsequent CTA was undertaken.

The Northern Ireland Online Electronic Care Records System (NIECR)¹⁴ was used to assess patient outcome and follow up. For those patients with VAI, further data on associated injuries, neurological status and treatment were collected. The purpose of gathering this extra information was to compare the VAI patients in this study with those in previous studies.

Radiography

The imaging of patients with a cervical spine fracture was reviewed and classified according to the AOSpine Cervical

Spine Fracture classification system (Types I-III).¹⁵ In cases where CT angiography was undertaken, the results were reviewed to determine if the patient had VAI. This was classified by the segment of the vertebral artery involved and also by the Blunt Carotid and Vertebral artery Injury (BCVI) grading system.¹⁰

Statistical Analysis

Due to small patient numbers, meaningful statistical analysis was not possible, thus descriptive analysis of the data using Microsoft® Office Excel® 2007 was performed.

RESULTS

Overall, 1,894 patients presenting to the RVH ED from June 2018 to June 2019 underwent CT imaging of their cervical spine and/or vertebral arteries. Sixty-eight patients were found to have a cervical spine fracture (3.59%). There were 39 males (57.4%) and 29 females (42.6%), mean age 60.4 years (range 18-97 years, standard deviation \pm 22.8 years).

Mechanism of Injury

The most common mechanism of injury was a fall, which accounted for 41 (60.3%) of the 68 cases (**Table 1**). This included both falls from standing and falls from height. Road traffic collisions (RTC) caused 23 cases (33.8%). Two patients sustained a cervical spine fracture following assault, one due to a rugby tackle and one in a go-karting accident.

Table 1.

The mechanisms of injury for the 68 patients with acute cervical spine fracture are outlined.

Mechanism	Number of patients
Fall	41 (60.3%)
Road traffic accident	23 (33.8%)
Assault	2 (2.9%)
Go-karting accident	1 (1.5)
Rugby tackle	1 (1.5)

Fracture Pattern

Of the 68 patients with cervical spine fractures, 37 (54.4%) had a fracture of their upper cervical spine and 31 (45.6%) had a fracture affecting the subaxial cervical spine. When classified by injury morphology as per the AOSpine Fracture Classification System¹⁵, 41 patients (60.3%) had Type A injuries (i.e. bony injury only), nine (13.2%) had Type B injuries (i.e. tension band injuries) and 18 (26.5%) had Type C injuries (i.e. translation injuries).

Of the patients with upper cervical spine fractures, four patients (10.8%) had Type I injuries (i.e. involving the occipital condyle/ occipital-cervical joint complex injuries), 11 (29.7%) had Type II injuries (i.e. C1 ring and C1/C2 joint complex injuries), and 22 (59.5%) had type III injuries (i.e. C2 and C2/C3 joint complex injuries) as per the AOSpine classification. When classified by injury morphology, 16 patients (43.2%) had Type A injuries, six (16.2%) had Type



B injuries and 15 (40.5%) had Type C injuries.

Of the patients with subaxial cervical spine fractures, the most common injury morphology was Type A as per the AOSpine classification, accounting for 80.6% of cases (**Table 2**).

Table 2.

Subaxial cervical spine fracture morphology as per AOSpine Fracture Pattern Classification.

AOSpine classification	Key features	Number of patients
A0	A fracture not significantly affecting spinal stability	22 (68.8%)
A1	Compression fracture involving a single endplate without involvement of the posterior vertebral body wall	3 (9.7%)
A2	Coronal fracture of the vertebral body involving both endplates but not the posterior wall	0
A3	Incomplete burst fracture involving a single endplate and the posterior wall	0
A4	Complete burst fracture involving both endplates and the posterior wall	0
B1	Disruption to the osseous posterior tension band	1 (3.2%)
B2	Complete disruption of the posterior capsuloligamentous or bony capsuloligamentous structures together with a vertebral body, disk, and/or facet injury	2 (6.5%)
B3	Disruption of the anterior tension band	0
C	Failure of anterior and posterior elements leading to displacement or translation of one vertebra compared to another in any axis	3 (9.7%)

Sixty-two (91.2%) of the 68 patients were neurologically intact following their injury. Five patients (7.35%) were diagnosed with an incomplete spinal cord injury and one patient (1.47%) with a complete spinal cord injury. Of note, all three of the Type C subaxial spine fractures resulted in neurological damage.

Thirty patients (44.1%) were found to have fractures of the transverse processes. Further study was undertaken as these can potentially involve the foramen transversarium, posing a risk to the vertebral arteries. The foramen transversarium were involved in ten (33.3%) cases, and three (30%) of these patients were found to have vertebral artery injury.

Vertebral Artery Injury

Fifteen patients of this cohort underwent CTA, eleven (73.3%) of whom had fractures involving the upper cervical spine and nine (60%) of whom had fractures involving the foramen transversarium. Five patients were found to have VAI (i.e. 7.35% of the overall cohort with a confirmed cervical spine fracture). All five of these patients had fractures involving the upper cervical spine resulting from high-energy injuries (**Table 3**).

Table 3.

Clinical and radiographic features corresponding to the five patients with VAI.

Sex	Age (years)	Mechanism of injury	AOSpine Classification	Significant associated injuries	Neurological status	VAI grade	Follow-up CT-angiography	Fracture management	VAI management
M	33	RTA	Type I, A, F3, fracture extends into TF	Haemorrhagic cortical contusions	Intact	II	Day 6; injury unchanged	Aspen collar	Observation
M	50	RTA	Type II, C	SAH	Intact	I	Day 10; injury not seen	Doll's collar	Observation
M	26	RTA	Type III, C	SAH, base of skull fracture	Intact	IV	Day 3; partial recanalisation	Minerva jacket	Aspirin
M	83	RTA	Type III, C, fracture extends into TF	Thoracic vertebral body fracture	Unknown	I	Day 7; injury unchanged	Posterior stabilisation C4-T4	Aspirin
F	58	Fall off horse	Type III, A, fracture extends into TF	Nil significant	Incomplete spinal cord injury	IV	Day 10; injury unchanged	Aspen collar	Aspirin

Abbreviations: M=male, F=female, RTA=road traffic accident, TF=foramina transversarium, SAH=subarachnoid haemorrhage,



All cases of VAI were found to involve the right vertebral artery in either the V2 (foraminal) or V3 (extraspinal) segments. As per the BCVI system, two patients had grade I injury (i.e. luminal irregularity or dissection with intraluminal haematoma occluding <25% of the lumen), one patient had grade II injury (i.e. luminal irregularity or dissection with intraluminal haematoma occluding >25% of the lumen) and two patients had grade IV injury (i.e. total occlusion of the vessel). All five patients underwent follow-up CTA within ten days of injury. Of note, none of the five patients was confirmed to have a neurological deficit resulting from the VAI. In one case, it was impossible to accurately determine neurological status due to severe sepsis from which the patient later died, and one patient was found to have signs suggestive of central cord syndrome presumed to have resulted from acute cervical hyperextension.

Regarding management, only one patient underwent surgical stabilisation. Three patients received antiplatelet therapy; in the remaining two cases this was deemed not appropriate due to acute intracranial haemorrhage.

DISCUSSION

First described by Carpenter et al in 1961¹⁶, cervical spine fractures are well documented in the literature as being a risk factor for VAI.

This study found a 7.35% incidence of VAI in patients diagnosed with acute cervical spine fracture in Northern Ireland over a 12-month period. The incidence of VAI in the literature varies greatly, perhaps reflecting differences in study populations.^{2,10,17} Fleck et al¹⁷ found the VAI incidence to be almost three times greater than ours, however we note the median age was 45 years, 16 years lower than that of the current study, where the median age was 61 years. We suggest that as younger patients are more likely to experience cervical spine fractures from high impact trauma, and VAI is associated with high impact trauma, this may explain why our incidence is lower.

In this study, falls were the most common mechanism of injury leading to a cervical spine fracture. However, in keeping with previous studies, we found RTC to be the most common mechanism associated with fractures resulting in VAI.^{11,17,18}

Fracture Pattern

In keeping with findings by Leucht et al¹⁹, we found that type A injuries were the most common (60.3%) and type B injuries the least common (13.2%).

The most common cervical spine fracture type associated with VAI is a fracture of the transverse process extending into the foramen transversarium. In this study, three of the ten patients with fractures involving the foramen transversarium were found to have VAI. Although our numbers are small, this is felt to be in keeping with other studies, reporting VAI in approximately 20% of cases of fractures involving the foramen transversarium.^{2,10}

The current study found that 37 (54.4%) of the 68 patients had a fracture of their upper cervical spine, five (13.5%) of whom were diagnosed with VAI. These findings are in keeping with the findings of Mitha et al⁷, who found the incidence of VAI in patients with upper cervical spine fractures to be 18%. The vertebral artery is most mobile as it passes through the transverse foramen of C2 and moves laterally to pass through the transverse foramen of C1, putting it at risk of damage during mechanical injury.¹³

The third fracture type associated with VAI is dislocation or subluxation of the vertebrae. Eighteen (26.5%) of the 68 patients in this study were found to have subluxation of their cervical spine. Three (16.7%) of these patients were diagnosed with VAI. Mueller et al² found the incidence of VAI in patients with cervical spine subluxations to be as high as 31%.

Vertebral Artery Injury

The V2 (foraminal) and V3 (extraspinal) segments of the vertebral artery are most at risk of damage due to their passage through the foramina transversaria⁸, with injuries located in the second and third parts in 26% and 55% of cases respectively¹¹. In keeping with this, we found all cases of VAI to involve the V2 or V3 segments.

As the dominant vertebral artery provides a greater contribution to the basilar artery than its non-dominant counterpart, this may influence clinical signs and also long-term outcomes⁹. For example, damage to the dominant artery, which in most cases is the left, may be more likely to result in a posterior circulation stroke, whereas damage to the non-dominant vertebral artery may not produce any clinical symptoms due to sufficient collateral blood supply²⁰. Biffi et al¹⁰ reported that 88% of posterior circulation ischaemic events occurred in patients with a left dominant vertebral artery injury. In the present study, all patients experienced injury to the right vertebral artery, which, in most cases, is non-dominant. This may explain why none of the patients was found to suffer ischaemic complications resulting from the VAI.

The risk of a thrombotic stroke after VAI varies in the literature but rates of up to 24% have been reported²¹. None of the five patients with VAI in this study had a subsequent stroke. Bonney et al²² noted a mortality of 4-8% for VAI. One patient with VAI in this study died, however this was as a result of chest sepsis.

Diagnostic Techniques

In this study, the only imaging modality undertaken to look for VAI was CTA.

The gold standard method for diagnosis of VAI is digital subtraction angiography (DSA), which can detect very subtle intimal injury. However, this is an invasive procedure with a complication rate of 4-8% which may include contrast induced nephropathy and stroke. Therefore DSA is no longer in widespread clinical use.^{8,11}



Other diagnostic methods include Doppler Ultrasonography (USS) and Magnetic Resonance Angiography (MRA). Doppler USS is widely available and the least invasive, however it is user dependent and has a reported sensitivity of 38.5% for detecting VAI. MRA is often regarded as impractical for initial screening of the trauma patient due to scan duration.¹¹

CTA has been shown to have a sensitivity and specificity for VAI of 68% and 92% respectively.¹¹ Thus, it is now the most widely used method to investigate for VAI⁴. However, CTA exposes the patients to radiation and to potentially nephrotoxic contrast.²² Given the sensitivity of CTA for VAI, it is possible that in our cohort some cases of VAI were missed, however the clinical relevance of this remains unclear.

Selection Criteria for Screening

Initially, the screening criteria for VAI were broad and were felt to subject many patients to unnecessary imaging.²³ Recently, there has been an emphasis on determining a more specific set of criteria. Biffi et al¹⁰ produced the Denver Screening Criteria for Blunt Cerebrovascular Injury. However, this screening criteria also includes carotid artery injury, which differs from VAI in presentation, prognosis and treatment.⁹ Therefore, further investigation is warranted to determine a set of screening criteria specific to VAI.

Fracture patterns deemed to be at highest risk for VAI are facet joint dislocations, fractures involving the transverse foramen, and fractures of the upper cervical vertebrae.^{8,24} Knowledge of these fracture patterns has allowed clinicians to limit their screening procedures to those with the highest risk. In keeping with this, we found that of the ten patients with fractures extending into the transverse foramen, nine underwent subsequent CTA.

Of the patients in this study who underwent CTA, one third were found to have VAI. None of these patients was documented as being symptomatic at the time of presentation, thus highlighting the need for appropriate screening amongst patients with high-risk fracture types in the absence of clinical signs.

Treatment

In this study three patients with VAI were commenced on antiplatelet (aspirin) therapy following discussion with the local stroke team. The remaining two patients experienced acute intracerebral haemorrhage; therefore, antiplatelet therapy was not felt appropriate and these patients were closely monitored for neurological deterioration. No patients underwent endovascular or surgical treatment for their VAI.

To our knowledge, no randomised controlled trials comparing the efficacy of different treatment strategies have been conducted to date. Several studies have reported no difference between patients treated with heparin and those treated with antiplatelets.^{25,26,27} However, there is a lack of evidence in the literature regarding the efficacy of endovascular treatment when compared with medical treatment. The optimum

duration for antiplatelet therapy also remains unclear.

Subsequent imaging

It has been suggested that patients with VAI should undergo a second CTA within one week.²⁸ In this study, all five patients with VAI underwent repeat CTA within ten days from initial imaging. For the three patients commenced on aspirin therapy, CTA findings demonstrated stability of the injury in two cases and partial resolution in one case. Regarding the two cases for whom antiplatelet therapy was contraindicated, one patient was found to have unchanged appearances, and in the other patient's case, the injury was no longer visualised, perhaps reflecting the sensitivity of CTA in detection of VAI. In both these cases, no neurological deterioration was observed, therefore no further imaging was undertaken.

The importance of follow-up CTA has been documented in other studies. Biffi et al²⁹ reported that improvement was noted in 57% of patients with grade I injury, allowing cessation of their treatment. Conversely, 8% of grade I injuries progressed to pseudoaneurysm requiring endovascular stenting. In the present study, no change to management was made for any of the five patients. Thus, we believe further work is warranted regarding optimal timing of repeat imaging to affect clinical practice.

Limitations

Limitations to our study include sample size, with only five cases of VAI identified over a one-year period. The data were collected in a retrospective manner from a single trauma centre. Retrospective data collection using administrative databases with potential coding discrepancies may have caused cases to be overlooked. Furthermore, as this study only included patients who underwent initial CT imaging arranged by the RVH ED, patients undergoing CT imaging following admission may have been missed.

CONCLUSION

This study found the incidence of VAI in patients presenting with cervical spine fractures to the RVH ED to be 7.35% between June 2018 and June 2019. In all cases, these fractures resulted from high-energy injuries. Fracture patterns associated with increased risk of VAI are fractures involving the upper cervical spine, fractures with associated subluxation, and fractures of the transverse process extending into the transverse foramen, and therefore urgent CTA is recommended.

Further work should focus on developing a more targeted set of criteria for screening for VAI in cervical spine fractures. Further study is also warranted regarding the efficacy of current treatment options for VAI, including optimum duration for medical therapy.

Sources of financial support: None

Declaration of interest: None



REFERENCES

1. Tannoury C, Degiacomo A. Fatal vertebral artery injury in penetrating cervical spine trauma. [published correction appears in Case Rep Neurol Med. 2017;2017:3861804]. *Case Rep Neurol Med*. 2015;1–5. doi: 10.1155/2015/571656
2. Mueller CA, Peters I, Podlogar M, Attila Kovacs, Horst Urbach, Karl Schaller, *et al*. Vertebral artery injuries following cervical spine trauma: a prospective observational study. *Eur Spine J*. 2011; 20(12): 2202–9.
3. Chung D, Sung JK, Cho DC, Kang DH. Vertebral artery injury in destabilized midcervical spine trauma; predisposing factors and proposed mechanism. *Acta Neurochir (Wien)*. 2012; 154(11): 2091–8.
4. Weber CD, Lefering R, Kobbe P, Horst K, Pishnamaz M, Sellei RM, *et al*. Blunt cerebrovascular artery injury and stroke in severely injured patients: an international multicenter analysis. *World J Surg*. 2018;42(7):2043–53.
5. Kopelman TR, Leeds S, Berardoni NE, O'Neill PJ, Hedayati P, Vail SJ, *et al*. Incidence of blunt cerebrovascular injury in low-risk cervical spine fractures. *Am J Surg*. 2011;202(6):684–8.
6. Inamasu J, Guiot BH. Vertebral artery injury after blunt cervical trauma: an update. *Surg Neurol*. 2006; 65(3):238–45.
7. Mitha AP, Kalb S, Ribas-Nijkerk JC, Solano J, McDougall CG, Albuquerque FC, *et al*. Clinical outcome after vertebral artery injury following blunt cervical spine trauma. *World Neurosurg*. Elsevier Inc., 2013;80(3–4): 399–404.
8. Harshavardhana NS, Dabke HV. Risk factors for vertebral artery injuries in cervical spine trauma. *Orthop Rev (Pavia)*. 2014;6(3):128–30.
9. De Souza, RM, Crocker MJ, Haliasos N, Rennie A, Saxena A. Blunt traumatic vertebral artery injury: a clinical review. *Eur Spine J*. 2011;20(9):1405–16.
10. 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(5):672–81.
11. Aizpuru M, Poirier M-V, Benarroch-Gampel J. Vertebral artery injury: an update on screening, diagnosis and treatment. *Curr Surg Rep*. 2018;6(12):1–7.
12. Fusco MR, Harrigan MR. Cerebrovascular Dissections. *Neurosurgery*. 2010;68(2): 517–30.
13. Park KW, Park JS, Hwang SC, Soo-Bin IM, Shin W-H, Burn-Tae, K. Vertebral artery dissection : natural history, clinical features and therapeutic considerations. *J Korean Neurosurg Soc*. 2008;44(3):109–115.
14. NI Electronic Care Record. Information for better care. [Internet]. Available from: <http://www.ehealthandcare.hseni.net/niecr/niecr.aspx> [Last accessed March 2019].
15. Wilson JR, Harrop JS. Update on upper cervical spine injury classifications. *Semin Spine Surgery*. 2017;29(1):9–13.
16. Carpenter S. Injury of neck as cause of vertebral artery thrombosis. *J Neurosurg*. 1961;18(6):849–53.
17. Fleck SK, Langner S, Baldauf J, Kirsch M, Rosenstengel C, Schroeder HW, *et al*. Blunt craniocervical artery injury in cervical spine lesions: the value of CT angiography', *Acta Neurochir*. 2010;152(10): 1679–86.
18. Parbhoo AH, Govender S, Corr P. Vertebral artery injury in cervical spine trauma. *Injury*. 2001;32(4):565–77.
19. Leucht P, Fischer K, Muhr G, Mueller EJ, *et al*. Epidemiology of traumatic spine fractures. *Injury*. 2009; 40(2), 166–72.
20. Jang JW, Lee JK, Hur H, Seo BR, Lee JH, Kim SH, *et al*. Vertebral artery injury after cervical spine trauma: a prospective study using computed tomographic angiography. *Surg Neurol Int*. 2011;2: 39. doi: 10.4103/2152-7806.78255.
21. Tobert DG, Le HV, Blucher JA, Harris MB, Schoenfeld AJ. The clinical implications of adding CT angiography in the evaluation of cervical spine fractures a propensity-matched analysis. *J Bone Joint Surg Am*. 2018;100(17):1490–5.
22. Bonney PA, Burks JD, Conner AK, Glenn CA, Baker CM, Cheema AA, *et al*. Vertebral artery injury in patients with isolated transverse process fractures. *J Clin Neurosci*. 2017;41:111–4.
23. Franz RW, Willette PA, Wood MJ, *et al*. A systematic review and meta-analysis of diagnostic screening criteria for blunt cerebrovascular injuries. *Journal of the American College of Surgeons*. Elsevier Inc., 2012;214(3):313–27.
24. Cothren CC, Moore EE, Ray Jr CE, Johnson JL, Moore JB, Burch JM, *et al*. Cervical spine fracture patterns mandating screening to rule out blunt cerebrovascular injury. *Surgery*. 2007;141(1): 76–82.
25. Alterman DM, Heidel RE, Daley BJ, Grandas OH, Stevens SL, Goldman MH, *et al*. Contemporary outcomes of vertebral artery injury. *J Vasc Surg*. 2013;57(3):741–6.
26. Cothren CC, Biffi WL, Moore EE. Treatment for blunt cerebrovascular injuries. *Arch Surg*. 2009;144(7):685–90.
27. Shahan PC, Fabian TC. Update on Treatment of Blunt Cerebrovascular Injuries. *Curr Trauma Rep*. 2019;5(1):35–9.
28. Brommeland T, Helseth E, Aarhus M, Moen KG, Dyrskog S, Bergholt B, *et al*. Best practice guidelines for blunt cerebrovascular injury (BCVI). *Scand J Trauma, Resusc Emerg Med*. 2018;26(90):1–10. doi.org/10.1186/s13049-018-0559-1
29. Biffi WL, Ray Jr CE, Moore EE, Franciose RJ, Aly S, Heyrosa MG, *et al*. Treatment-related outcomes from blunt cerebrovascular injuries importance of routine follow-up arteriography. *Ann Surg*. 2002;235(5):699–707.



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