Original Article

Vertebral artery dissection in acute cervical spine trauma

ABSTRACT

Objective: The aim of this study was to study mechanism, risk factors, and prognosis of patients with vertebral artery dissection (VAD) from acute cervical spine trauma (CST).

Methods: A total of 149 consecutive patients were chosen from 2014 to 2019 from our institute data base, and their records were retrospectively studied. Morphology of fracture and subluxation were studied in detail with respect to the presence or absence of VAD.

Results: Patients were divided in subsets of axial spine injury and subaxial spine injury. Subgroup and group analysis was performed and computerized tomography angiogram, MR angiogram and T1/T2 axial scans were studied to identify VAD, an incidence of 14.1% was found. Patients having infarcts in posterior circulation were also identified.

Conclusion: There is a significant contribution of biomechanics of CST and evolution of VAD. This is an important consideration to prevent significant morbidity and mortality. Hence, a diagnostic algorithm which can be applied in any hospital setup is the need of the hour.

Keywords: Cervical spine trauma, infarcts, vertebral artery dissection

INTRODUCTION

Cervico-cerebral arterial dissections are a broad category, which include vertebral artery dissection (VAD) and internal carotid artery dissection. The incidence of VAD ranges from 7% to 39%[1-5] in cases of blunt cervical spine trauma (CST). Rates may have been underestimated in the existing literature due to diagnostic difficulties. VAD can be spontaneous or traumatic in etiology. Trivial causes such as coughing, sneezing, vomiting, childbirth, and chiropractor manipulation are all reported to be the cause of VAD in the existing literature. Traumatic causes can be either penetrating or blunt injuries. VAD causing unilateral occlusion is more common than bilateral. The incidence of vertebrobasilar ischemia is 12%–20%[2,5] in unilateral occlusion. However, the incidence can be as high as 80%[6] in the spontaneous type. It mostly affects the extracranial segment[2,7,8] followed by the intracranial segment.

The majority of the VADs are asymptomatic or only mildly symptomatic. Symptoms range from occipital headache with or without neck pain to recurrent transient ischemic attacks and posterior circulation stroke, which may or may not include brainstem.[9] Rarely, if extracranial dissection reaches till intracranial segment, it may cause subarachnoid hemorrhage.[6] The upper cervical spine (CS) (upper CS [UCS], above C3), C-3, fractures reaching foramen transversarium (FT), facet dislocation[1] have been reported to be high risk for VAD. The complications of VAD can be delayed but disastrous; hence, it should be caught early in the course, as it may be the cause of nonimprovement, delayed improvement, or even deterioration after surgery for unstable CST.

Rahul Gupta, Hardik Lalit Siroya, Dhananjaya Ishwar Bhat[1], Dhaval P Shukla, Nupur Pruthi, Bhagavatula Indira Devi

Department of Neurosurgery, National Institute of Mental Health and Neurosciences, [1]Department of Neurosurgery, RV Aster Hospital, Bengaluru, Karnataka, India

Address for correspondence: Dr. Bhagavatula Indira Devi, Department of Neurosurgery, National Institute of Mental Health and Neurosciences, Bengaluru, Karnataka, India.
E-mail: bidevidr@gmail.com

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Many authors studied VAD, but most of them are either single case reports[10-17] or case series,[18-21] and very few studies tried to describe the traumatic cervical vertebral pathology in detail.

This study is a retrospective study exploring the etiology of VAD in CST by separating the elements of injuries to the CS using plain computerized tomography (CT), CT angiogram (CTA)/magnetic resonance imaging (MRI) with or without magnetic resonance angiography (MRA).

METHODS

This study is a retrospective study done at the National Institute of Mental Health and NeuroSciences, Bengaluru, Karnataka, India, for 1 year (2018–2019). Data were collected from medical records department through database search of the following ICD-10 codes S12, S12.0-S12.7, S12.9 (cervical vertebrae fracture), and S13.1 (cervical subluxation) from January 2014 to December 2019, the total number of cases registered with us for CST with above ICD-10 codes were 264, of which, 56.4% (n = 149) consecutive cases were included based on the following.

Inclusion criteria

1. Diagnosed cases of CS fractures or cervical subluxation on CT CS
2. Patients having MRI (with or without MRA) scan or CTA.

Exclusion criteria

1. Patients not having fractures/subluxation on CT scan
2. Patients who had incomplete or suboptimal scans
3. Patients with other causes of injury as posttraumatic prolapsed intervertebral disc and cervicothoracic subluxation
4. Patients having signs of congenital atlantoaxial dislocation clinically or radiologically.

The study design, as shown in Figure 1, was followed. Details related to injury such as date, mechanism, clinical assessment (ASIA grade), CT scan, CTA, or MRI findings. Morphology of injury was studied in detail for each case without knowing the status of the VA. Statistical analysis was performed using R version 3.6.1 and IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. (Armonk, NY: IBM Corp.). Tests applied Chi-square analysis and multivariable logistic regression analysis.

RESULTS

Demography

Patients not having MRI/CTA scan or having suboptimal scans, despite significant CT scan findings, were 110 (21.03%). Four patients had posttraumatic disc causing compression, 1 had a cervicothoracic subluxation, hence excluded.

Out of the total 149 patients, as mentioned in Table 1, the most common mechanism of injury was a road traffic accident with a two-wheeler (49, 32.9%). Most patients were the victim of high energy trauma (114, 76.5%). Complete spine injury was present in 7 (4.7%) patients, whereas 136 (91.3%) patients had an incomplete injury. Most patients had ASIA Grade D, 62 (41.6%), then Grade E, 48 (32.2%), injury. Data of six patients were missing.

Radiology

On arrival of the patient in casualty after detailed examination CT CS was done to rule out CST. CTA or MRI was done as required, as mentioned in Table 1. 21 (14.1%) patients had associated cranial injuries such as linear calvarial fractures (2, 1.3%), extradural hematoma (2, 1.3%), subdural hematoma (14, 9.4%), brain contusion (2, 1.3%), and intracerebral bleed (1, 0.7%) all managed conservatively.

Computerized tomography cervical spine

A total of 88 (59.1%) patients had only UCS (above C3) injury, 54 (36.2%) patients had only lower CS (at and below C3) injury, 7 (4.7%) had both; it includes fractures as well as subluxations. Fractures without subluxation were present in 104 (69.8%) patients, and cervical subluxation was present in 23 (15.4%) patients, while 22 (14.8%) patients had both fractures and subluxations. The patients had vertebral body fracture, including burst, teardrop, wedge compression fractures, lamina fracture, transverse process fractures which may or may not reach the foramen, statistics as mentioned in Table 1. These were either present in a combination or individually. Fractures of the lamina are significantly associated with fractures of the body (P = 0.01) and fractures reaching FT (P = 0.001) but were not associated with the mechanism of injury (low, medium, or high energy) (P = 0.15). The median interquartile range (IQR) SLIC score was 9 (7–10). The most common cervical subluxation injury, without associated fracture, was C5-C6, 13 (56.5%). 22 (48.9%) patients had Grade II anterior subluxation. 87.5% of patients with subluxation had facet dislocation or perching.

Vertebral artery

MRI protocol included T1, T1 - Volumetric interpolated breath-hold examination (VIBE), T2, T2 gradient sequence, with or without time of flight (TOF)/MRA. The median IQR time between injury and MRI scan was 3 (1–12) days. 139 (93.3%) patients had MRI scans, out of which 44 (31.7%) cases had TOF/MRA, and 95 (68.3%) cases did not have MRA and only had T2 and T1 (VIBE) sequence axial, sagittal, coronal sequences.

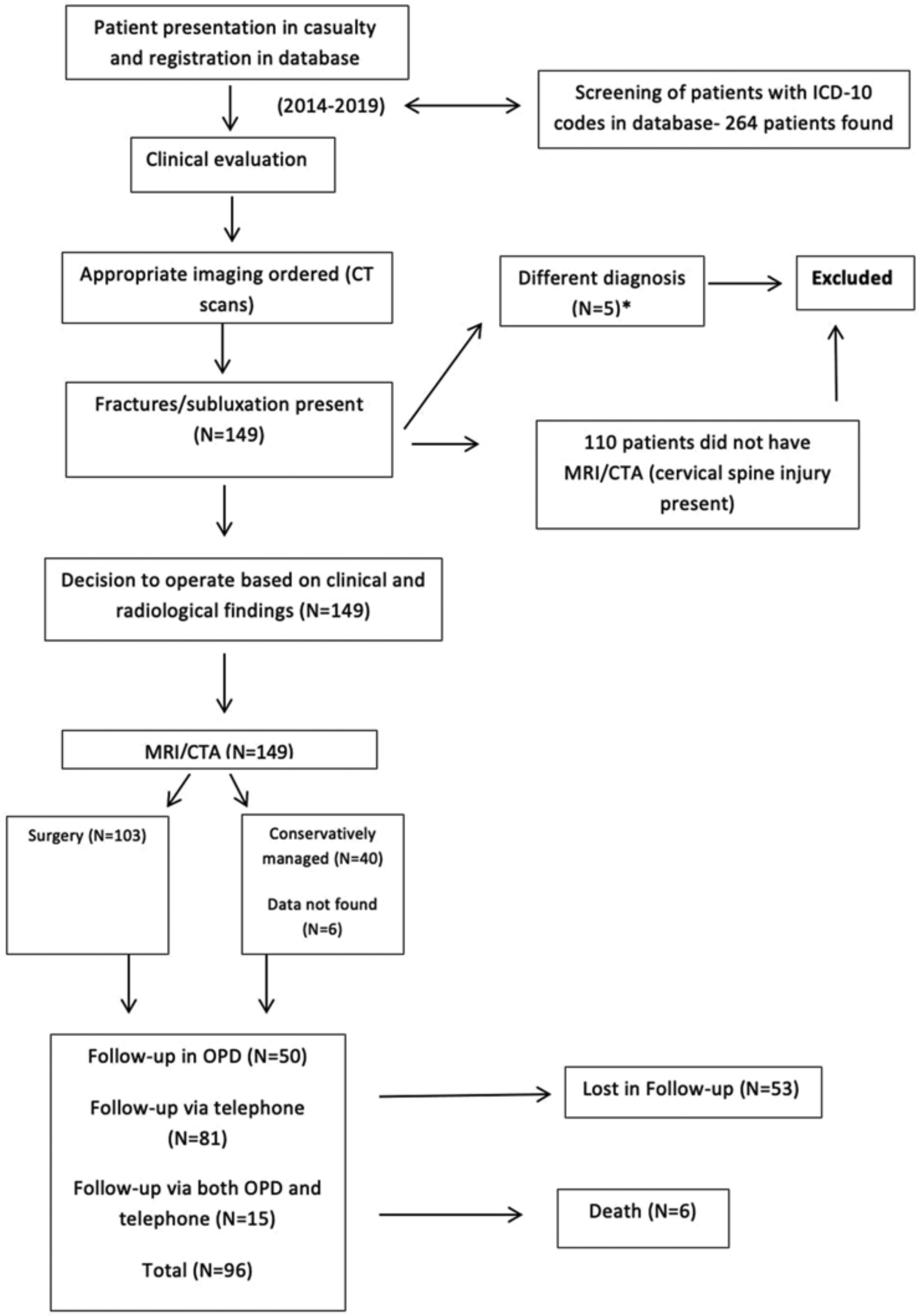

Figure 1: Study design in the form of a flow chart

21 (14.1%) patients had VAD. 11 (52.4%) cases had right VAD, 9 (42.9%) cases had left VAD, and 1 patient had bilateral VAD. In 8 (38.1%) patients, the diagnosis of VAD was made using MRA and in 4 (19.1%) patients, it was done with CTA. CTA was performed in 5 cases of VAD, out of which in 1 case CTA showed patent flow in VA and diagnosis was made via T1 VIBE/T2 axial images. In 10 (47.6%) cases T1 VIBE/T2 axial images were diagnostic of VAD. It was instrumental in the diagnosis of a total of 18 (85.7%) cases of VAD. In 3 (16.7%) cases, T2-weighted (T2W) images showed normal flow void, but the T1w VIBE sequence had abnormal signal changes. Hyperintense signal change in T2W images and isointense signal changes in T1W VIBE images was the most common finding in VAD in which the signal change was present in the entirety of the artery and was not restricted to the site of trauma only. Signal change may range from flow void to varied appearance due to different ages of the thrombus or stagnation of blood flow across the vessel.

Among the VAD group, two types of fractures were noted in majority, FT fracture with foraminal encroachment 7 (33.3%,

Table 1: Demography

Key variables (total)	Categories	*n* (%)
Sex (*n*=149)	Male	127 (85.2)
	Female	22 (14.8)
Age (*n*=149)	Median (IQR)	32 (25-46)
Alcohol (*n*=141)	Yes	19 (12.8)
	No	122 (81.9)
Mechanism of injury		
Low energy	Self-fall (level ground)	11 (7.9)
	Slip and fall (level ground)	9 (6.4)
	Fall from bicycle (level ground)	1 (0.7)
	Total	21 (15.1)
Medium energy	Work place injury	1 (0.7)
	River diving (head first)	2 (1.4)
	Chiropractic manipulation (following RTA 2 wheeler)	1 (0.7)
	Total	4 (2.9)
High energy	RTA 4 wheeler	15 (13.2)
	RTA autorickshaw	3 (2.1)
	RTA pedestrian	6 (4.3)
	RTA 2 wheeler	49 (35.3)
	Fall from height	33 (23.7)
	Fall of weight over head	6 (4.3)
	Fall from bus	1 (0.7)
	Fall from train	1 (0.7)
	Total	114 (82.0)
	Missing	10 (7.1)
Severity (*n*=143)	Complete injury	6 (4.2)
	Incomplete injury	137 (96.0)
	Missing	6 (4.2)
Level of injury (*n*=149)	UCS - C1, C2	88 (59.1)
	LCS - C3 and below	54 (36.2)
	Both	7 (4.7)
C1 fracture (*n*=20)	Anterior arch	4 (20.0)
	Posterior arch	2 (10.0)
	Both (Jefferson fracture)	14 (70.0)
C2 fracture (*n*=75)	Hangman's fracture	14 (18.7)
	Odontoid fracture (Anderson and D'Alonso grading)	
	Grade I	-
	Grade II	44 (72.1)
	Grade III	17 (27.9)
	Total	61 (81.3)
Subluxations (*n*=45)	Grade I	12 (26.7)
	Grade II	22 (48.9)
	Grade III	8 (17.8)
	Grade IV	3 (6.7)
Vertebral artery dominance (*n*=149)	Right	10 (6.7)
	Left	23 (15.4)
	Co-dominance	116 (77.9)
Surgery (*n*=149)	Yes	103 (69.1)
	No	40 (26.8)
	Missing	6 (4.0)
Follow-ups (*n*=149)	Available	96 (64.4)
	Missing	53 (35.6)
Death (*n*=96)		6 (6.2)

UCS - Upper cervical spine; LCS - Lower cervical spine; IQR - Interquartile range; RTA - Road traffic accident

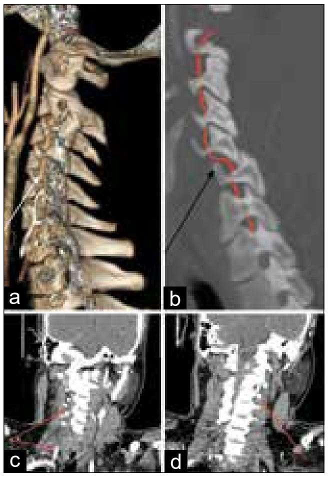

Figure 2: Upper panel shows normal and distorted course of vertebral artery (VA), (a) volume rendering techniques reconstructed sagittal image of cervical spine with normal course of VA (white arrow), entering the Foramen at C6. (b) A case of C5-C6 Grade II subluxation with right facet locking, with bilateral vertebral artery dissection, as shown by black arrow and the red marking in this image depicts the course of VA which is disturbed by the facet locking, leading to vertebral artery dissection however the cause of left vertebral artery dissection (not shown) could not be elicited via imaging. Lower panel shows computerized tomography angiogram coronal view with bilateral vertebral artery dissection; (c) Proximal stump of VA and distal filling defect as pointed by red arrows, a sign of right vertebral artery dissection; (d) Filling defect of left VA till C3-C4, suggestive of left vertebral artery dissection also with retrograde filling of left VA

$n = 21$) and facet fractures 7 (33.3%, $n = 21$). Furthermore, some patients noted to have unique mechanisms of injury, as described in Figures 2 and 3. VAD was significantly associated with lamina fracture (odds ratio [OR] = 0.2; 95% confidence interval [CI], 0.07–0.6; $P = 0.005$) and infarcts (OR = 10.5; 95% CI, 1.9–59; $P = 0.008$) in multivariable logistic analysis.

VA dominance was present in 33 (22.1%) cases where 10 (30.3%) had right side dominance, and 23 (69.7%) had left side dominance, 116 (77.9%) cases had co-dominant VA. While the dominance of VA, SLICS score, mechanism of injury, vertebral body fracture, FT fractures, axial spine injury, presence of osteophytes near FT, and CS subluxation all were statistically insignificant in predicting the occurrence of VAD in preliminary scans.

- Infarcts, a sign of vertebrobasilar insufficiency, were seen in 7 (4.7%) patients, out of which 71.4% patients had dense infarct, suggestive of thrombus occluding major vessel, while 28.6% had patchy infarct, suggestive of an embolic phenomenon. Three patients with significant posterior circulation infarcts and did not have VAD, while the other four patients were diagnosed with VAD. The patient with bilateral VAD had right cerebellar dense infarct in posteroinferior cerebellar (PICA) artery territory. Infarcts were unilateral (4, 57.1%) and bilateral (3, 42.9%), primarily cerebellar, and in the other two cases, posterior cerebral circulation infarcts were also present, out of which one case had both PCA and cerebellar infarct. No case of brain stem stroke was seen.

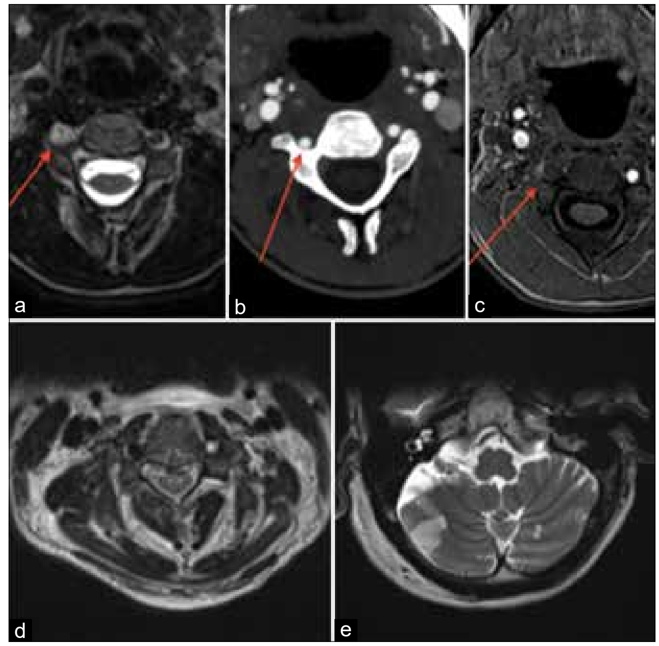

Figure 3: Upper panel shows vertebral artery dissection in axial Magnetic resonance imaging, (a) T2-weighted (T2W) axial image showing right vertebral artery dissection, (c) T1W volumetric interpolated breath-hold examination axial showing the same, (b) computerized tomography angiogram axial view at the corresponding vertebral level showing vessel patency, as shown by the arrows. Lower panel shows posterior circulation infarcts, (d) T2W axial image showing left VA intraluminal hyperintensity suggestive of thrombus or stagnation of blood, hence diagnosis of vertebral artery dissection was considered, (e) T2W image showing right cerebellar posteroinferior cerebellar arteryregion infarct

A total of 40 (36.8%) patients were managed conservatively, while 103 (69.1%) patients underwent surgery, out of which, 71 (47.7%) patients underwent preoperative cervical traction application.

Follow-up and outcome

A total of 96 (64.4%) patients' data were available for follow-up, of which 50 patients followed up in outpatient department and 46 patients were contacted through only telephone; 53 (35.6%) were lost to follow-up. The median IQR interval of follow-up was 29.3 (19.3, 49.2) months. The median modified Rankin score (MRS) was 1 in all the patients and 3 in the VAD group ($P > 0.05$). A total of 6 (7.4%, $n = 96$) deaths were reported, out of which two patients were in the VAD Group. Two patients died because of complications of bedsores (development of carcinoma from chronic bedsore after 1 year of trauma and the other patient from infection after 6 months of trauma), one died from respiratory complication 1 year after the trauma, causes of death are unknown in the other two patients.

DISCUSSION

The current study represents 149 consecutive patients (satisfying inclusion criteria) who presented to a Level I trauma center with cervical vertebrae fractures and subluxations. These patients were evaluated with either CTA or MRI as part of preoperative evaluation. The incidence of VAD in the current study was 14.1%, which is comparable to the published data on blunt CS injuries,[1-5] 7%–39%. Most VAD symptoms are mild, which can cause delayed improvement or nonimprovement in patients recovering from CST. This study did not report any catastrophic symptoms of vertebrobasilar insufficiency. The latter has been reported by many authors over time.[6,22] In patients with blunt CST with neurological deficits, it poses a challenging question about whether it is possible to diagnose or even suspect VAD on clinical examination, as symptoms are mostly mild and can be confused with CST symptoms. Association of cervical fractures and subluxations has been studied in the literature before, but emphasis on the fracture's morphology was studied in only a few studies. We have attempted to study the morphology and mechanism of injury involved in CST.

Fractures and subluxations

After the Denver criteria for fracture with high risk for VAD was published, the main focus was on FT fractures, subluxation injuries, C1-C3 fractures while screening patients for VAD. This was used as screening protocol in many studies, causing higher than the expected statistical significance of these fracture patterns and the cases of VAD where these injuries were not present remained underreported. Okawara and Nibbelink 1974[23] and Louw *et al*. 1990[24] were among the first ones to give importance to the mechanism of injury, fracture types, and their role in VAD. In our study most common mechanism of injury associated with VAD was flexion-distraction injury (Allen-Furguson classification) [Table 2]. There has been a disagreement on the mechanism of injury responsible for VAD. Okawara and Nibbelink[23] suggested that the primary mechanism causing VAD is hyperextension with or without rotation, with significant compression sites being craniovertebral junction and UCS in cases with or without fracture or dislocation and C5-C6 in cases of fracture or dislocation, a similar mechanism has been suggested by others.[25,26] According to Louw *et al*.,[24] primary mechanism of VAD was the flexion-distraction injury; they also noted that there was discordance between the level of facet dislocation and VAD, which was supported by other authors[4,7,27-29] as well. In the current study, there was no statistical significance between facet dislocation, included in SLIC score, and VAD in line with results which were reported by Vaccaro *et al*. 1998[30] and Biffl *et al*. 2000.[5]

Role of FT fractures has been widely studied before,[1,3,28,31-36] and various authors reported different incidence of VAD in FT fracture, Kral *et al*. 2002,[28] 16%, Willis *et al*. 1994,[29] 46%, Woodring *et al*. 1993,[36] 88%, our study reported an incidence of 15.7%. This discrepancy is because of selection bias in cases selected for diagnostic imaging, as the cases where these injuries were not present were not discussed. In the current study there was no statistical significance between FT fractures and VAD. Durand *et al*. 2015[32] and Lebl *et al*. 2013[31] identified a similar level of significance as our study. We noticed a pattern of injuries which led to increased incidence of VAD [Table 2 and Figure 4], it should be noted that the foramen fracture may be present in many patients but what counts is the encroachment of the foramen. Lebl *et al*.[31] suggested the same approach and found a significant relationship between fracture segment displacement >1 mm in the foramen and VAD, same was reported by Durand *et al*.,[32] their findings regarding high energy trauma, type of surgery, UCS injury, and facet subluxation were unrelated to VAD which was the case with our study as well. According to Merrill *et al*. 2020,[7] the most vulnerable segment for VAD was V3. Our study also showed the same finding.

Presence of facet fracture is a significant finding from Table 2 which may lead to VAD. Previous publications[4,24,29,30,37] suggested that there is an increased incidence of VAD following uni/bilateral locked facets or perched facets, on the contrary, our findings have suggested that a fractured facet may be a more important sign of an ipsilateral VAD on a CT scan. Facet fracture can displace body of the vertebrae such that VA can get compressed between the bony fragments.

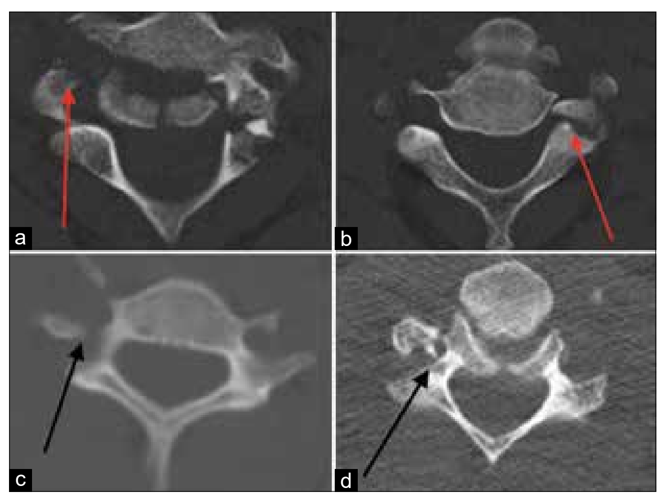

Figure 4: (a and b) Plain computerized tomography (CT) axial images of a patient with right C2 foramen transversarium (FT) fracture without encroachment (red arrow in a) and left C3 FT fracture with encroachment with left vertebral artery dissection (red arrow in b); (c) plain CT axial images of a patient with right FT fracture without encroachment with left side vertebral artery dissection (black arrow in c); (d) right FT fracture with encroachment with right vertebral artery dissection (black arrow in d)

Table 2: Characteristics of vertebral artery dissection patients

Diagnosis	Dissection Side	Dominance	Dissected segment	UCS injury	Type of LCS injury (Allen-Furguson type)	Extra finding	ASIA grade	MRS	Death
C2 odontoid fracture type III and body fracture	Right	Left	V3	Yes	-	-	4	3	No
C2 odontoid fracture II	Left	Right	V3	Yes	-	Left C2 FT fracture with encroachment	4		No
C2 odontoid fracture II, C3 body	Right	Left	V3	Yes	Flexion-compression	Right C3 FT fracture with encroachment	4		No
C2 odontoid fracture II	Right	Left	V3	Yes	-	-	5	0	No
C2 odontoid fracture	Right	Co dominant	V3	Yes	-	-	4		No
C2 odontoid fracture II	Left	Co dominant	V3	Yes	-	-	5	0	No
C2 body fracture	Left	Co dominant	V2	Yes	-	C1 assimilation, and fused facet joints on left side at multiple levels	2	5	No
C2 odontoid fracture, C3 body fracture	Right	Left	V3	Yes	Flexion-compression	Right C3 FT fracture with encroachment	4	3	No
C2 odontoid II, C4-C5 Grade I subluxation	Left	Right	V3	Yes	Flexion-distraction	Left C4/5 FT fracture with encroachment with C5 facet fracture	2		No
C2 odontoid fracture, C3-C4 sub Grade I	Left	Right	V3	Yes	Flexion-distraction	Left C3/4 FT fracture with encroachment with C4 facet fracture	2		No
Hangman fracture	Right	Left	V2	Yes	Flexion-distraction	Left C2 FT fracture without encroachment	4	1	No
Hangman fracture, C3 burst body fracture	Left	Right	V3	Yes	Flexion-compression	Left C3 FT fracture with encroachment and right C2 FT fracture without encroachment	3	4	No
C3 burst body fracture	Right	Co dominant	V2	No	Flexion-compression	C3 lamina fracture with lateral displacement of C3 on C4	4	3	No
C4-C5 subluxation Grade III	Right	Co dominant	V2	No	Flexion-distraction	Bilateral facet locking		6	Yes
C5-C6 subluxation Grade I, C1 anterior and posterior arch fracture	Left	Co dominant	V2	Yes	Flexion-distraction	Right C5 FT fracture without encroachment with right facet perched and left facet locked	4	0	No
C5 burst body fracture	Right	Left	V2	No	Flexion-compression	C3-C4-C5 lamina fracture with left C1 facet fracture with bilateral C5 pars fracture	2	5	No
C5-C6 Grade III subluxation	Right	Co dominant	V2	No	Flexion-distraction	Right C6 FT fracture with encroachment and bilateral facet locking	1		No
C5-C6 burst body fracture	Right	Co dominant	V3	No	Flexion-compression	Right C5 facet fracture with slight anterior translation of C6 over C7	3	4	No
C5-C6 sub Grade II	Bilateral	Co dominant	V2	No	Flexion-distraction	Left facet locking	2	6	Yes
C6 burst body fracture	Left	Co dominant	V2	No	Compression	Left C3 facet fracture	4	4	No
C6-C7 subluxation Grade II, C4 tear drop fracture	Right	Co dominant	V3	No	Extension-distraction	Right C6 facet fracture, C7 lamina fracture	5	1	No

UCS - Upper cervical spine; LCS - Lower cervical spine; ASIA - American Spinal Injury Association; MRS - Modified Rankin Score; FT - Foramen transversarium

Also, it is interesting to note that VAD was almost always on the opposite side of the dominant artery, which is in line with the physical laws of stress and strain upon a cylindrical vessel:

1. Tensile strain $\propto$ 1/A - Eq. (1)
2. F $\propto$ A - Eq. (2)

(F - Force; A - cross sectional area of vessel)

Equation 1 stands correct when comparing same kind of material experiencing approximately the same amount of force, which would mean that a larger vessel has the

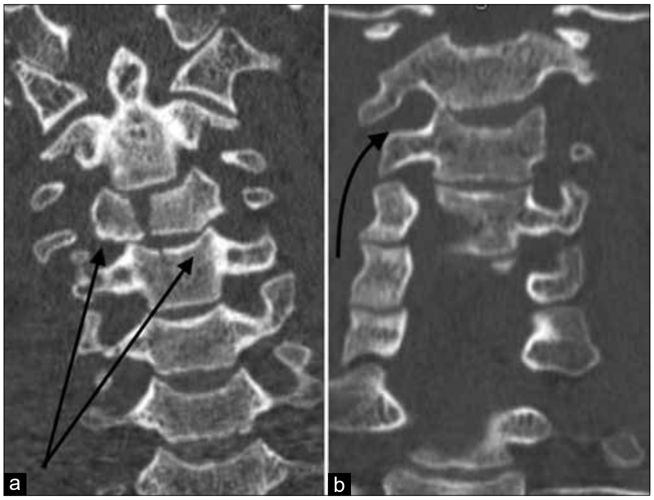

Figure 5: Unusual mechanisms of vertebral artery dissection, (a) Plain computerized tomography coronal view, is a case of C3 burst body fracture with lamina fracture (not shown) with right vertebral artery dissection, and due to the body fracture C3 lateral shift over C4 (arrows in a) which supposedly compressed right VA causing dissection; (b) is a case of Type II C2 odontoid fracture with right vertebral artery dissection, as shown by curved arrow, lateral tilt of C2 over C3 narrowed the FT and could cause right vertebral artery dissection

tendency to resist tensile strain better than the smaller vessel. According to Equation 2, more force (perpendicular to the surface area) is required to deform a larger vessel, given the material is same and both cylinders undergo similar change in lengths. Few unusual mechanisms are discussed in Figure 5.

In this study fracture of the lamina was found to be protective against VAD. As it is an extension-compression type of injury, during hyperextension injury of the neck, there comes a moment at the peak of force application, where either bone or soft tissue gives away, and usually bone gives away earlier if ligaments are not diseased and hence it can protect against any excessive stretching over the soft tissues. To the best of our knowledge, the role of fracture of the lamina in preventing VAD was not studied before, our study probably is the first one to provide such an insight.

In the current study, only seven patients with VAD had subluxations ranging from Grade 1 to 3; 38 patients of subluxations did not have VAD. The most common level of subluxation was found to be C5-C6.

Magnetic resonance imaging and computerized tomography angiogram

The gold standard for the diagnosis of VAD is digital subtraction angiogram (DSA). DSA being an invasive test is not very much popular because the treatment of VAD is mostly conservative. Popular investigations these days are MRA or CTA; however, the sensitivity is questioned. Miller *et al*.[3] reported 53% sensitivity of CTA and 47% of MRA to detect VAD. However, the case with bilateral VAD was diagnosed with CTA, MRI was unavailable for that case [Figure 2].

We propose that the sensitivity can be improved by the staging of different imaging modalities. Veras *et al*.[19] emphasized the importance of the T1w axial sequence in the diagnosis of VAD. We reported one case, included in our series, in which CTA was negative for any vascular insult, Figure 3, and the diagnosis was made by T1W VIBE and T2W axial images. Variable signal changes in the vessel maybe because of the blood clot of different ages or blood flow stagnation. During a long-term follow-up, recanalization of VA was reported by Taneichi *et al*.[4] and Vaccaro *et al*.[30] According to Taneichi *et al*.,[4] restoration of blood flow was more in patients with compressive injuries; in the current study, only two patients' MRI were available during follow-up, both cases had vertebral body fractures, first patient had 26 months MRI postinjury which showed persisting occlusion of the dissected artery and in the second case 5 months postinjury MRI showed recanalization of the artery.

Infarcts seen in posterior circulation after CST are generally ascribed to the possibility of VAD. Biffl *et al*.[5] reported a 24% incidence of posterior circulation stroke in which four patients had brain stem involvement. We found a 19% incidence of infarct with VAD. Moreover, in 3 cases, VAD was not found, infarcts were present in right PCA, right PCA and left cerebellar, bilateral cerebellar territory, were probably embolic phenomenon [Figure 3]. The presence of infarcts was significantly associated with VAD. Brainstem infarcts were not seen in the current study. Lockwood *et al*.[1] reported an increased incidence of posterior circulation stroke in subluxation or FT fracture and also that not all infarcts were associated with VAD, as our study. However, the reason that why infarcts appear without VAD remains unanswered. Many symptoms of VAD may go unnoticed when present with cervical fractures, due to similarity of symptoms with CSI or asymptomatic disease. Even cases of spinal cord injury without radiographic abnormality should be screened carefully to rule out VAD.

Asymmetry in VA size is a fairly common finding. In the current study, 22.2% of patients either had left or right-side dominance, left more common than right. However, it was not significantly associated with VAD, supported by studies of other authors.[38,39] According to Sangari *et al*.,[40] the right side was shown to have a larger mean diameter than the left. VA dominance was diagnosed only by visual assessment in general practice and in the current study, which is amenable to error. Many criteria of diagnosing VA dominance/

hypoplasia are defined in the existing literature, but none of them is backed up by embryological or anatomical research hence are incomplete.

Follow-up and outcome

MRS scores of the patients after a median period of 29.3 months with or without VAD were 3 and 1, respectively. The VAD group had poor outcomes. Similar findings were reported by Shlobin *et al*.,[41] they studied 291 patients with VAD, out of which 39 were associated with cervical fractures. These patients had poor outcomes at discharge, 3 months and last follow-up. There were two patients in the VAD group in our data who died and the cause of death remained unknown in both the cases.

We propose a diagnostic protocol [Figure 6], to better diagnose VAD. Although we have proposed this flowchart, we feel at the end of the study that all patients with cervical cord injury should undergo MRI with MRA.

Limitations

Because of retrospective study, the follow-up data are incomplete. Imaging protocols were not always complete. Treatment strategies of VAD are not discussed.

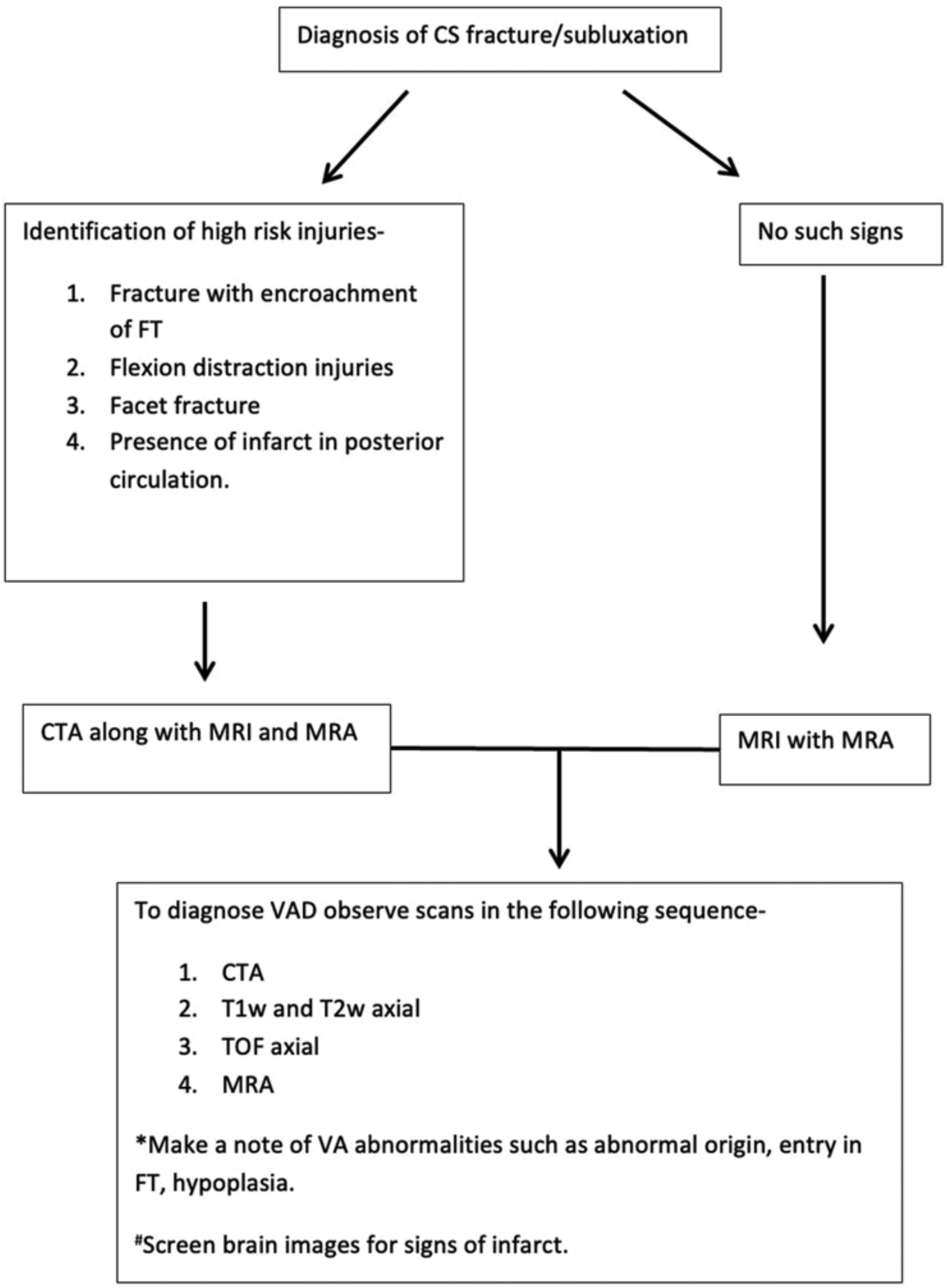

Figure 6: Proposed diagnostic algorithm to better diagnose vertebral artery dissection

CONCLUSION

This study was an analysis of 149 cases of CS injury with fracture and subluxations and their relation with VAD. MRI protocol for CS injury should include MRA. VAD may lead to slow or no recovery and unexplained symptoms in spine trauma patients leading to poor outcomes. A prospective study design with adequate follow-up or experimenting with the animal model of VAD will help in understanding mechanisms of injury involved and hence the disease process better.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Lockwood MM, Smith GA, Tanenbaum J, Lubelski D, Seicean A, Pace J, *et al.* Screening via CT angiogram after traumatic cervical spine fractures: Narrowing imaging to improve cost effectiveness. Experience of a Level I trauma center. J Neurosurg Spine 2016;24:490-5.
2. Jang JW, Lee JK, Hur H, Seo BR, Lee JH, Kim SH. Vertebral artery injury after cervical spine trauma: A prospective study using computed tomographic angiography. Surg Neurol Int 2011;2:39.
3. Miller PR, Fabian TC, Croce MA, Cagiannos 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; discussion 393-5.
4. 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 (Phila Pa 1976) 2005;30:1955-62.
5. Biffl 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.
6. Arnold M, Bousser M. Clinical manifestations of vertebral artery dissection. Front Neurol Neurosci 2005;20:77-86.
7. Merrill S, Clifton W, Valero-Moreno F, Damon A, Rahmathulla G. Vertebral artery injury with coinciding unstable cervical spine trauma: Mechanisms, evidence-based management, and treatment options. Cureus 2020;12:e7225.
8. Arnold M, Bousser MG, Fahrni G, Fischer U, Georgiadis D, Gandjour J, *et al.* Vertebral artery dissection: Presenting findings and predictors of outcome. Stroke 2006;37:2499-503.
9. Gottesman RF, Sharma P, Robinson KA, Arnan M, Tsui M, Ladha K, *et al.* Clinical characteristics of symptomatic vertebral artery dissection a systematic review. Neurologist 2012;18:245-54.
10. Yoshihara H, Vanderheiden TF, Harasaki Y, Beauchamp KM, Stahel PF. Fatal outcome after brain stem infarction related to bilateral vertebral artery occlusion – Case report of a detrimental complication of cervical spine trauma. Patient Saf Surg 2011;5:18.
11. Elder T, Tuma F. Bilateral vertebral artery transection following blunt trauma. Int J Surg Case Rep 2018;51:29-32.
12. Wirbel R, Pistorius G, Braun C, Eichler A, Mutschler W. Bilateral vertebral artery lesion after dislocating cervical spine trauma. A case report. Spine (Phila Pa 1976) 1996;21:1375-9.
13. Nakao Y, Terai H. Embolic brain infarction related to posttraumatic occlusion of vertebral artery resulting from cervical spine injury: A case report. J Med Case Rep 2014;8:344.
14. Schippinger G, Spork E, Obernosterer A, Seibert FJ, Schweighofer F. Injury of the cervical spine associated with carotid and vertebral artery occlusion: Case report and literature review. Injury 1997;28:315-8.
15. Yaeger K, Mascitelli J, Kellner C, Hickman Z, Mocco J, Margetis K. Temporary vertebral artery occlusion after C3 fracture dislocation injury and spontaneous resolution following reduction and instrumented fusion: Case report and literature review. J Neurointerv Surg 2017;9:1027-30.
16. Dutta G, Jagetia A, Srivastava AK, Singh D, Singh H, Saran RK. "Crick" in neck followed by massage led to stroke: Uncommon case of vertebral artery dissection. World Neurosurg 2018;115:41-3.
17. Frink M, Haasper C, Ringe KI, Krettek C, Hildebrand F. Blunt cerebrovascular trauma causing vertebral arteryd issection in combination with a laryngeal fracture: A case report. J Med Case Rep 2011;5:381.
18. Amarenco P, Hauw JJ, Hénin D, Duyckaerts C, Roullet E, Laplane D, *et al.* Cerebellar infarction in the area of the posterior cerebellar artery. Clinicopathology of 28 cases. Rev Neurol 1989;145:277-86.
19. Veras LM, Pedraza-Gutiérrez S, Castellanos J, Capellades J, Casamitjana J, Rovira-Cañellas A. Vertebral artery occlusion after acute cervical spine trauma. Spine (Phila Pa 1976) 2000;25:1171-7.
20. Komiyama M, Morikawa T, Nakajima H, Nishikawa M, Yasui T. Embolic cerebellar infarction caused by spontaneous dissection of the extracranial vertebral artery – Two case reports. Neurol Med Chir (Tokyo) 2000;40:310-4.
21. Maloney E, Lehnert B, McNeeley MF. Vertebral artery transection in nonpenetrating trauma: A series of 4 patients. Ann Vasc Surg 2015;29: 6. e11-6.
22. Schellinger PD, Schwab S, Krieger D, Fiebach JB, Steiner T, Hund EF, *et al.* Masking of vertebral artery dissection by severe trauma to the cervical spine. Spine (Phila Pa 1976) 2001;26:314-9.
23. Okawara S, Nibbelink D. Vertebral artery occlusion following hyperextension and rotation of the head. Stroke 1974;5:640-2.
24. Louw JA, Mafoyane NA, Small B, Neser CP. Occlusion of the vertebral artery in cervical spine dislocations. J Bone Joint Surg Br 1990;72:679-81.
25. Six EG, Stringer WL, Cowley AR, Davis CH Jr. Posttraumatic bilateral vertebral artery occlusion: Case report. J Neurosurg 1981;54:814-7.
26. Simeone FA, Goldberg HI. Thrombosis of the vertebral artery from hyperextension injury to the neck. J Neurosurg 2009;29:540-44.
27. 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 (Phila Pa 1976) 1997;22:188-92.
28. Kral T, Schaller C, Urbach H, Schramm J. Vertebral artery injury after cervical spine trauma: A prospective study. Zentralbl Neurochir 2002;63:153-8.
29. 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.
30. 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 (Phila Pa 1976) 1998;23:789-94.
31. Lebl DR, Bono CM, Velmahos G, Metkar U, Nguyen J, Harris MB. Vertebral artery injury associated with blunt cervical spine trauma: A multivariate regression analysis. Spine (Phila Pa 1976) 2013;38:1352-61.
32. Durand D, Wu X, Kalra VB, Abbed KM, Malhotra A. Predictors of vertebral artery injury in isolated C2 fractures based on fracture morphology using CT angiography. Spine (Phila Pa 1976) 2015;40:E713-8.
33. 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.
34. Cothren CC, Moore EE, Biffl WL, Ciesla DJ, Ray CE Jr., Johnson JL, *et al.* Cervical spine fracture patterns predictive of blunt vertebral artery injury. J Trauma 2003;55:811-3.
35. McKinney A, Ott F, Short J, McKinney Z, Truwit C. Angiographic frequency of blunt cerebrovascular injury in patients with carotid canal or vertebral foramen fractures on multidetector CT. Eur J Radiol 2007;62:385-93.
36. Woodring JH, Lee C, Duncan V. Transverse process fractures of the cervical vertebrae: Are they insignificant? J Trauma 1993;34:797-802.
37. Even J, McCullough K, Braly B, Hohl J, Song Y, Lee J, *et al.* Clinical indications for arterial imaging in cervical trauma. Spine (Phila Pa 1976) 2012;37:286-91.
38. Katsanos AH, Kosmidou M, Kyritsis AP, Giannopoulos S. Is vertebral artery hypoplasia a predisposing factor for posterior circulation cerebral ischemic events? A comprehensive review. Eur Neurol 2013;70:78-83.
39. Jeng JS, Yip PK. Evaluation of vertebral artery hypoplasia and asymmetry by color-coded duplex ultrasonography. Ultrasound Med Biol 2004;30:605-9.
40. Sangari SK, Dossous PM, Heineman T, Mtui EP. Dimensions and anatomical variants of the foramen transversarium of typical cervical vertebrae. Anat Res Int 2015;2015:391823.
41. Shlobin NA, Mitra A, Prasad N, Azad HA, Cloney MB, Hopkins BS, *et al.* Vertebral artery dissections with and without cervical spine fractures: Analysis of 291 patients. Clin Neurol Neurosurg 2020;197:106184.