Vertebral artery injuries following cervical spine trauma: a prospective observational study

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Abstract

Purpose The purpose of this study was to report on the incidence, diagnosis and clinical manifestation of VAI following cervical spine injuries observed in a prospective observational study with a standardized clinical and radiographical protocol.

Methods During a 16-year period, 69 (mean age: 43 ± 20.7 years; 25 female, 44 male) of 599 patients had cervical spine injury suspicious for VAI due to facet luxation and/or fractures extending into the transverse foramen. Diagnosis and management of these patients followed a previously published protocol (Kral in Zentralbl Neurochir 63:153–158, 2002). Digital subtraction angiography (DSA) was performed in all 69 patients. Injury grading of

VAI was done according to Biffl et al. (Ann Surg 231:672–681, 2000). All patients with VAI were treated with anticoagulation (heparin followed by ASS) for 6 months.

Results In cases suspicious for VAI, the incidence of VAI detected by DSA was 27.5% ($n = 19$ of 69 patients). VAI Grade I occurred in 15.8%, Grade II in 26.3%, Grade IV in 52.6% and Grade V in 5.2%. Of 19 patients, 4 (21%) had clinical signs of vertebrobasilar ischemia. Two patients died in hospital after 4 and 21 days respectively. Of 69 patients, 33 (47.8%) with suspected VAI had unstable spine injuries and were treated surgically.

Conclusion In patients with cervical spine fractures or dislocations crossing the course of the vertebral artery, VAI are relatively frequent and may be associated with significant morbidity and mortality. VAI were identified by DSA in 27.5%. Despite anticoagulation therapy, 5.8% became clinically symptomatic and 2.9% died due to cerebrovascular ischemia.

Keywords Vertebral artery injury · Arteriography · Cervical spine trauma · CT-angiography · MR-angiography

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Introduction

Vertebral artery injuries (VAI) can occur with cervical spine injury. VAI may result in devastating consequences if unrecognized and untreated, with mortality rates up to 31% [18]. The incidence of VAIs following cervical spine injury varies in the literature from 0.53 to 88% [5, 25, 27, 30, 46, 47].

The vertebral artery (VA) is most susceptible to injury at the point of entrance into the transverse foramen of C6. The second most common site of VAI is at C1–C2 [32, 37].

A cerebrovascular injury classification, which was originally created for traumatic carotid artery injuries, has been applied to VAI [6, 28, 40]. The rationale for screening of asymptomatic trauma patients for the presence of VAI is that it may occur in many asymptomatic patients [33] and anticoagulation may prevent strokes and improve neurological outcome [18].

Screening criteria for patients to exclude a VAI are different forms of spine cervical luxation and fractures involving the transverse foramen [44, 47]. Catheter angiography (DSA) has been the gold standard for the diagnosis of VAIs; however, multidetector computed tomography angiography (CTA) and magnetic resonance angiography (MRA) seem to have sensitivity and specificity close to that of DSA [19].

In this prospective study, the incidence, clinical findings and treatment results after cervical spine injury were described following a standardized diagnostic and therapeutic protocol [19].

Materials and methods

Between April 1996 and January 2010, 599 patients with cervical spine trauma were admitted to our department. Since X-rays and CT scans identify 98% of cervical fractures and 99% of subluxations [48], they are justified diagnostic tools in cervical spine trauma. X-rays (AP/lateral; flexion/extension) was the first step in cooperative patients with neck pain. Furthermore, in patients with pathological findings in the X-ray we performed as a second step a cervical CT scan. In patients with additional neurological deficit (e.g., cervicobrachialgia, palsy, numbness, headache, vertigo, ataxia), or intubated and/or severely injured patients, we performed as a first step a cervical CT scan and in a second step, if possible, an X-ray examination. This is in accordance with a previously published protocol [24].

The indication for immediate evaluation of the VA by arterial DSA was as follows: (a) fractures extending into the transverse foramen [trauma type I]; (b) vertebral fractures with facet dislocation [trauma type II] or pure ligamentous injury with facet dislocations [trauma type III]; (c) vertebrobasilar ischemic symptoms following cervical spine trauma without obvious radiological signs of spine injury; and (d) no other life-threatening injuries. Additional MRA studies (3D phase contrast angiography technique) and 16-slice computed tomography angiography (CTA) studies were performed for comparison with DSA. The rationale for the supplementary MRA and CTA investigation in this study was an academic interest in finding how far the results from DSA were comparable with the recently established techniques of CTA and MRA in the

radiologic unit of our clinic. Furthermore, MRI offers the possibility of identifying a contusio spinalis or discoligamentary instability more clearly than any other diagnostic tool.

DSA results served for VAI classification, according to Biffi et al. [5]—Grade I injury: arteriographic appearance of irregularity of the vessel wall or a dissection/intramural hematoma with less than 25% luminal stenosis; Grade II injury: intraluminal thrombus or raised intimal flap is visualized, or dissection/intramural hematoma with 25% or more luminal narrowing; Grade III injury: pseudoaneurysms; Grade IV injury: vessel occlusion; and Grade V injury: vessel transections.

Treatment of traumatic VA occlusion or stenosis (in all cases with VAI) consisted of intravenous anticoagulation with heparin. We started with a continuous infusion (15 U/kg/h) with a perfusion pump to achieve a partial thromboplastin time (PTT) of 40–50 s for 2 weeks. PTT blood samples were collected two times a day and the desired PTT was achieved by gradually titrating the heparin. The intravenous anticoagulation treatment was followed by oral administration of acetylsalicylic acid (AspirinTM) once a day for 6 month. Treatment was started with i.v. heparin because its effect can be monitored and antagonized easily. Early surgical stabilization to release tension on VA and prevent ischemic events was done in patients with no other life-threatening injuries. Clinical follow-up was performed after 3 and 6 months in patients with VAI. Furthermore, we performed a long-term follow-up using a telephone interview (mean follow-up, 83.3 months). A radiological long-term follow-up was not possible, due to legal regulations of radiation protection (CTA, DSA) and the potential risk of allergic reaction of the contrast agent (MRA). However, X-rays in surgically treated patients were performed routinely. Clinical and radiological data were assessed prospectively and kept in files.

The chi-square, non-parametric Mann–Whitney and Kruskal–Wallis tests were used for statistical analysis of data. Results were considered significant at $p < 0.05$ level. The analyses were performed using SPSS 14.

Results

Patients with cervical spine trauma

All 599 patients, admitted between April 1996 and January 2010, for cervical spine injury were included in this study (median age: 53.0 years; SD $\pm$ 23.26; range 2–93 years; female: $n = 226$ [37.7%], male: $n = 373$ [62.3%]). The rationale for radiological examination (X-ray and/or cervical CT scan) is described above. A total of 179 patients

(29.8%) were treated surgically for fractures and/or dislocations: 21% with ventral surgery, 6.5% with dorsal surgery and 2.3% with combined ventro-dorsal surgery.

In 530 of 599 patients (88.5%), neither fractures involving the transverse foramen nor dislocations or clinical signs of posterior circulation ischemia were noted. They will not be further considered.

Patients with types of injury suspicious for VAI

Of the remaining 69 patients (mean age: 43 ± 20.71 years; female = 25, male = 44, $p < 0.05$) with potential injury, 36 (52.17%) were admitted after high velocity accidents (motor vehicle and bicycle accidents) and 33 (47.83%) after low velocity (falls) accidents. Of the 69 patients, 36% had an initial spinal cord injury ASIA grade A–D; 24 (34.8%) had a vertebral fracture extending into the vertebral foramen (trauma type I), 37 (53.6%) had vertebral fractures with ligamentous injury and facet dislocation (trauma type II), and 8 (11.6%) had pure ligamentous injuries with facet dislocations (trauma type III). No flow alterations at the level of injury were seen in 50 (72.5%) patients by DSA ($p < 0.05$). Fifteen (30%) of these patients had additional MRA and seven (14%) had additional CTA. Depending on the type of fracture, anterior cervical fusion was done in 27 of 69 patients (39.13%), dorsal fusion in 3 (4.34%), and ventro-dorsal fusion in 3 (4.34%) patients. Type III fractures were significantly

more often found in younger patients and surgery was performed significantly more often than for the other fracture types (Table 1).

Patients with VAI

DSA detected VAI in 19 patients (mean age: 43 ± 18.31 years; female = 7, male = 12, $p < 0.05$). Seven (36.84%) of these 19 patients had an initial neurological impairment (ASIA grade A–D). Five (26.3%) patients had trauma type I, 11 (57.89%) patients trauma type II, and three (15.8%) patients trauma type III. The most common level of injury was C5/6 ($n = 7$, 26.31%), followed by C2 ($n = 3$, 15.8%). In 11 (57.9%) patients there was a left-sided VAI (Table 2).

Four (21%) patients, all with trauma type II injuries, had clinical signs of vertebrobasilar ischemia. Two patients were initially comatose. Both patients died: one patient at day 4 after VAI and the other 21 days after VAI. One patient had vertigo and headache, and one patient had vertigo. All of the four patients had trauma type II. Of the 19 patients, in whom DSA had detected a VAI, 9 (47.36%) patients had additional MRA. In one case, unilateral VA stenosis of 15% due to an intimal flap caused by a Jefferson fracture was not detected by MRA. Another 6 of 19 (31.6%) patients had additional CTA. In all of them, the DSA results were confirmed by CTA. According to the classification of Biffl et al. [5], we observed 15.8% ($n = 3$)

Table 1 Group of patients with potential VA injury ($n = 69$ out of 599 patients with blunt cervical trauma)

	Type of trauma	Level of injury	Number of patients (%)	Age (years, median $\pm$ SD)	Gender (male, %)	Neurological impairment ASIA A–D [n (%)]	Surgical treatment [n (%)]	Pathological DSA findings [n (%)]
	I	C1	3 (4.3)	41 ± 17.85	14 (58.3)	7 (29.2)	9 (37.5)	5 (20.8)
		C2	13 (18.8)					
		C5	2 (2.9)					
		C6	5 (7.2)					
		C7	1 (1.4)					
	II	C1/2	9 (13.0)	48 ± 21.76	25 (67.6)	14 (37.8)	18 (48.5)	11 (29.7)
		C2/3	8 (11.6)					
		C3/4	1 (1.4)					
		C4/5	2 (2.9)					
		C5/6	9 (13.0)					
		C6/7	8 (11.6)					
Type I isolated vertebral fracture with extension into the vertebral foramen	III	C0/C1	1 (1.4)	28 ± 19.25	5 (62.5)	4 (50)	6 (75)	3 (37.5)
		C2/3	1 (1.4)					
		C3/4	1 (1.4)					
		C4/5	1 (1.4)					
Type II vertebral fractures with ligamentous injury and facet joint dislocations		C5/6	2 (2.9)					
Type III pure ligamentous injuries with facet joint dislocations		C6/7	2 (2.9)					
	All		69 (100)	43 ± 20.71	44 (63.8)	25 (36.2)	33 (47.8)	19 (27.5)

Table 2 Patients with DSA-confirmed VA injury

Type of trauma	Level of injury	Number of patients (%)	Age (years, median, SD)	Gender [male (%)]	Neurological impairment ASIA A–D [n (%)]	Surgical treatment [n (%)]	Left Side of VA injury [n (%)]	Symptoms from VA injury [n (%)]
I (<i>n</i> = 5)	C1	1 (5.3)	52 ± 14.10	3 (60)	1 (20)	0 (0)	4 (80)	0 (0)
	C2	3 (15.8)						
	C7	1 (5.3)						
II (<i>n</i> = 11)	C1/2	2 (10.5)	40 ± 18.65	9 (81.8)	5 (45.5)	9 (81.8)	4 (36.4)	4 (36.36)
	C2/3	2 (10.5)						
	C4/5	2 (10.5)						
	C5/6	3 (15.8)						
	C6/7	2 (10.5)						
III (<i>n</i> = 3)	C0/C1	1 (5.3)	25 ± 12.09	0 (0)	1 (33.3)	2 (66.7)	3 (100)	0 (0)
	C5/6	2 (10.5)						
All		19 (100)	43 ± 18.13	12 (63.2)*	7 (36.8)	11 (57.8)	11 (57.9)	4 (21.05)*

Type I isolated vertebral fracture with extension into the vertebral foramen

Type II vertebral fractures with ligamentous injury and facet joint dislocations

Type III pure ligamentous injuries with facet joint dislocations

* $p < 0.05$

Grade I, 26.31% (*n* = 5) Grade II, Grade III was not observed, 52.6% (*n* = 10) Grade IV and 5.26% (*n* = 1) Grade V VAI (see Fig. 1).

Therapeutic procedures

In patients with VAI, intravenous heparin was administered for 2 weeks and acetylsalicylic acid for an additional 6 months according to the protocol, which was interrupted when surgery was necessary (*n* = 11). No side effects occurred attributable to i.v. heparin or acetylsalicylic acid.

Patients with fractures and subluxations, where stabilization was indicated, were treated surgically without delay. Anterior cervical plate-screw fusion was indicated in nine patients. In one patient, a dorsal rod-screw fusion was performed. In one patient, a combined ventral–dorsal fusion was done. No surgical or neurological complications were observed related to these procedures.

Follow-up

Of the 19 patients, 15 (79%) with VA injury were neurologically intact at the 3- and 6-month follow-up. Two

Fig. 1 A 16-year-old male after a traffic accident, initial GCS 7, tetraplegia and priapism. **a** Initial right vertebral angiogram shows a VA occlusion with retrograde filling of the left V4 segment and PICA, intraoperative with aspect of left-sided VA transection. **b** Postoperative lateral angiogram of the left VA shows high grade stenosis at the

C1–C2 level. **c, d** The left vertebral artery was occluded with two detachable silicone balloons **e**: CT before atlanto-axial fixation (C2 pedicle screws and atlas clamp) **f** postinterventional CT (arrow detachable balloon within atlas loop)

patients died and two patients reported persistent vertigo. The final assessment was conducted at 12–168 months (mean follow up period 83.3 months, $n = 17$). None of the patients reported persistent headache, vertigo or other focal neurological deficits. Follow-up imaging was performed in three (15.8%) patients with DSA, in six (31.6) patients with MRA and in ten (52.6) cases with CTA.

In the early radiological follow-up in one case, the VAI grading had changed from Grade IV (occlusion) to Grade II (80% left-sided stenosis) 30 days after injury. In the other patient with an initial VAI Grade II (70% stenosis of the right VA), a PICA infarction was observed 6 weeks following the injury due to a complete occlusion of the VA (Grade IV). However, as mentioned above, a radiological long-term follow-up was not possible, due to legal regulations of radiation protection.

Discussion

Incidence of VAI

Carpenter was the first in 1961 to describe an association between cervical spine fractures and VAI [10].

In this prospective study, diagnostic and therapeutic procedures and outcome assessment of all patients were performed following an algorithm for early diagnosis and treatment of VAI as described previously [24]. Of 599 (11.5%) patients, 69 with cervical spine trauma had suspicion for VAI. In 19 of these 69 (27.5%) patients, a VAI was confirmed by DSA, resulting in an incidence of 3.1% of VAI for all spine traumata. Since patients with other life-threatening injuries were excluded by protocol, some cases may have been unused and the 3.1% may slightly underestimate the true incidence. In the literature, the incidence of VAI in cervical spine trauma ranges from 3 to 39% [5, 20]. This wide range of incidences of VAI is most likely attributable to the selection of the patients, type of injury, small patient cohorts, variation in imaging technique and inconsistencies in patient evaluation [41].

X-rays and CT scans identify 98% of cervical fractures and 99% of subluxations [48] and therefore are justified diagnostic tool in cervical spine trauma. Three types of cervical fractures are associated with significant risk for VAI: (a) fractures involving the transverse foramen [15, 23, 24, 44, 46, 47, 49]; (b) subluxations [10, 11, 15]; and (c) fractures involving the upper cervical spine (C1–C3) [15, 36]. In contrast to other groups, we did not include patients with facial hemorrhage and expanding cervical hematoma, although those also have a risk for VAI [46]. However, it is probably not necessary to consider every fracture of the upper cervical spine, i.e., those without involvement of the transverse foramina and without

luxations/subluxations, as a significant risk for VAI. In the present study, the incidence of VAI in fractures involving the transverse foramen was 20%, similar to the incidence of 19% observed by Giacobetti [20]. The incidence of VAI in subluxations and fracture dislocations in this study was 31%, being higher than that observed by Kral [24] with 21% and lower than those of Vaccaro [43] with 40%. Cothen reported an 18% incidence of VAI in fractures of the upper cervical spine (C1–C3) [15], lower to or incidence of 24%.

In the present study, DSA detected a VAI in 27% of the patients considered to be at risk for VAI. In 44% of cases, there was an interval of at least 18 h between the time of injury and the onset of ischemic symptoms, during which antithrombotic therapy could be instituted [7]. The high incidence of asymptomatic VAI's that may become symptomatic within 18 h after injury justifies rapid angiographic examination [25, 49] in patients considered at risk for VAI. We observed a VAI grade I in 15.5% and VAI grade II in 26.3% of the cases. This is in accordance with the incidence of grade I (17–42%) and grade II (13–55%) VAI reported in the literature [5]. The 0% incidence of grade III VAI in this series is lower (21–55%) [42], and the incidence of grade IV VAI of 52% is higher than reported in the literature (24%) [42]. The reason for this is obscure. Furthermore, in one case a VA rupture occurred (Grade V), with fatal outcome. This has not been described in the literature before.

The incidence of posterior circulation stroke following VAI ranges from 0 to 24% in case series with more than 40 patients [5, 15, 28]. In general, grade I–III arterial injuries (intimal flaps, dissections, and pseudoaneurysms) are considered more treacherous lesions, because they have a greater risk of embolic stroke than vertebral artery occlusions [5]. However, some series report comparable incidences of posterior circulation stroke with complete VA occlusions (Grade IV) [7, 17, 27]. Accordingly, in the present series, three patients (15.7%) had PICA infarctions following VAI (Grade II, IV and V), and one additional patient with grade IV VAI developed clinical signs of vertebrobasilar ischemia without radiologically proven PICA infarction.

Imaging: DSA versus CTA and MRA

Although DSA is the gold standard in the assessment of the vascular system, CTA and MRA are evolving as key imaging modalities for the noninvasive assessment of the vascular system. With the increasing availability and improved accuracy of multidetector computed tomography (MDCT), CT-angiography has widely replaced DSA as a first choice diagnostic tool (gold standard) for VAI in many institutions. Several studies have reported a high accuracy

of CTA in detecting “clinically significant injuries” [3, 4, 9, 45]. This is consistent with the limited observations of the present series. The fact that DSA was used in all cases to detect a VAI was based on the prospective study design and the delayed availability of alternative imaging modalities in our institution (CTA since 5 years and MRA since 10 years).

The usefulness of MRA, as a noninvasive imaging modality for VAI, was described previously [34, 43]. Furthermore, the MRI offers the possibility of identifying a contusio spinalis or discoligamentary instability more clearly than any other diagnostic tool. In the present study, MRA was not diagnostic in one case with VAI Grade I detected by DSA after unilateral fracture dislocation affecting the transverse process of C1. However, our series with nine patients was very small and the role of MRA in VAI diagnosis needs further refinement. A double-blinded prospective trial evaluating sensitivity and specificity of MRA was recommended [21]. However, in un-blinded prospective studies, sensitivity between 50 and 100% and a specificity 29–100% was described between MRA and DSA after cervical injury [1, 8, 28, 39]. Furthermore, in intubated and polytraumatized patients with other associated high risks, MRA is more difficult to perform than DSA or CTA [26, 31]. Thus, DSA and/or CTA should be performed on an exigent basis in patients considered at risk for VAI [5, 27]. However, the high rate of concordant results in the DSA and CTA in the detection of a VAI in our study supports the use of CTA as the first diagnostic tool. In future, we will change the diagnostic procedure: first CTA and then, as second step, if a neuroradiological intervention is necessary, also DSA.

Treatment

In unstable cervical spine fractures, to prevent further VAI damage, early surgical fixation is considered to be indicated [35]. All VAI patients in this study with isolated vertebral fractures with dislocation into the vertebral foramen [trauma type I] and one patient with pure ligamentous injury [trauma type III] were treated with external fixation (collar and/or Halo fixateur). In the radiological and clinical follow-up after 3 and 6 months, and into the long-term clinical follow-up, no secondary dislocation was observed and the neurological status of the patients was unremarkable.

Thrombus formation at the site of intimal injury may precipitate thromboembolic occlusion or may induce embolism of the vertebral circulation. Subintimal dissection may also lead to subsequent thromboembolism or vessel occlusion [2, 22, 38, 47]. These mechanisms explain why up to 44% of patients with initially clinically inapparent VAI become clinically symptomatic during the next

18 h. The good long-term outcome of our VAI patients in this series may be due to early diagnosis followed by the aggressive treatment protocol with early surgery and consequent anticoagulation used in this study. The anticoagulative treatment in our study is not evidence based, but it is concordant with previously published anticoagulative protocols for VAI [7, 13].

However, the optimal medical treatment of VAI is still under debate, and an evidence base of anticoagulation in patients with VAI does not exist; however, until now anticoagulative treatment is a standard therapy [14, 16, 18, 27]. Previous studies showed a decreased stroke rate from 30–50 to 2–10% using anticoagulation or antiplatelet therapy [4, 7, 14, 18, 27, 28]. Even in initial asymptomatic, untreated patients, the rate of neurological complications can be as high as 21% [12].

This prospective observational study shows further evidence that early surgical stabilization and i.v. anticoagulation in the acute stage followed by oral anticoagulation may protect from the vertebrobasilar ischemia [1, 29, 44].

Conclusion

In 69 patients with cervical spine fractures/dislocations crossing the course of the vertebral artery, VAI were relatively frequent and associated with significant morbidity and mortality. VAI were identified by DSA in 27.5%. Despite anticoagulation therapy, 5.8% became clinically symptomatic due to cerebrovascular insufficiency (or embolism). This may have potentially lethal consequences in up to 2.9% of the affected patients.

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Conflict of interest None.

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