



Vertebrobasilar and internal carotid arteries dissection in 188 patients

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1. Introduction

Arterial dissection mainly represents a tear of the intima coat (the intimal flap) with a consecutive penetration of blood into the arterial wall, due to a disruption of the internal elastic lamina and the media coat, and formation of a false lumen or local intramural (subintimal) hematoma [1–3]. In this case, a luminal stenosis develops which can progress to an occlusion with a subsequent hypoperfusion or ischemia of the cervical spinal cord, brain parenchyma, retina, and certain peripheral nerves, as well as the occasional local thrombosis with a potential distal thromboembolism [1,3–13].

On the other hand, a rupture of the intramural vasa vasorum, or a deeper penetration of blood from the lumen, causes a subadventitial hematoma formation [1,13–17]. Due to the latter, a dissecting aneurysm (pseudoaneurysm) can occur with a consecutive compression of the adjacent structures or with a perivascular hemorrhage [1,4,8,15,16].

Dissection can affect one or, less frequently, both internal carotid (ICAs) or vertebral arteries (VAs) [1,3,8,14,18–21], and exceptionally three or four of the mentioned vessels [22–42]. The cervical or intracranial segments of the mentioned paired arteries can be involved, including their branches, i.e. the main cerebral arteries [1,3,8,36,43–45].

Since the ICA, VA and basilar artery (BA) dissections (ICAD and VBAD) are usually presented as case reports [6,37,38,46–56] or as smaller groups of patients [1,8,27,36,41,43,57–63], we decided to present them in a group of 188 patients. In addition to a detailed examination of each patient, a comparison between the ICAD and VBAD will be presented as well.

2. Materials and methods

2.1. Patients

A total of 188 patients with arterial dissections were enrolled in the period from February 2008 to February 2018. Each patient had his or her history records, as well as laboratory results, cardiologic, neurologic, and radiologic findings, which were analyzed in detail.

2.2. Radiologic examinations

These examinations were mainly related to magnetic resonance imaging (MRI) and angiography (MRA), and rarely to computerized tomography (CT) and angiography (CTA), as well as to digital subtraction angiography (DSA) and Doppler sonography.

As regards MRI examination, the following sequences and procedures were applied. T1-weighted: TR/TE 450/min ms, matrix 320 × 224, FOV 24, slice thickness 5 mm, spacing 0.5 mm. T1-fat saturation: TR/TE 590/min, matrix 256 × 192, FOV 26, slice thickness 3 mm, spacing 1 mm. T2-weighted: TR/TE 4600/108 ms, matrix 384 × 256, FOV 24, slice thickness 5 mm, spacing 0.5 mm, as well as TR/TE (450/min), matrix 256 × 192. T2-fat saturation: TR/TE (4600/102), matrix 384 × 256, FOV 26, slice thickness (3 mm), spacing 1 mm. Diffusion-weighted imaging (DWI): TR/TE (ms) 8000/min, matrix 128 × 128, FOV 24, slice thickness (5 mm), spacing (0.5 mm), b-values, 0 s/mm² and 1000 s/mm². Fluid-attenuated inversion recovery (FLAIR): TR/TE 8000/120 ms, matrix 256 × 192, FOV 24, slice thickness 5 mm, spacing 0.5 mm, TI (2000 ms).

Brain MRA (3D TOF): TR/TE (23/7), matrix 384 × 224, FOV (22), section thickness 2 mm, overlap lochs 10, lochs per slab 32, FA 20°, acquisition time 5 min 4 s. Neck MRA (TRICKS): TE minimum, matrix 320 × 192, FOV 36.0, PHASE FOV 0.75, section thickness

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3.2 mm, scan locks 28, FA 30°, output temporal phases 15, scan time 1.10 (0.15).

As for CT examination, it was performed using CT 160-slice machine Toshiba Aquiline Prime by applying FOV 240 and slice thickness of 2.0 mm. In the case of CTA, the following parameters were used: FOV 240, KV 100, ma 150, scan time 5.4 s, slice thickness 0.5 mm, slice interval 0.3 mm, range 300, rotation time 0.5, and contrast application (0.8–1.2 mL/gym).

Four vessel angiography was performed in the INOVA GE Healthcare following a transfemoral catheterization with an automatic injection of the contrast substance (Omnipaque) in a bolus of 5–8 mL per each sequence.

Ultrasonography was performed in the majority of patients. We used Canon Inc Tus AI600 (APLIO I 600) apparatus, with a linear transducer of 7 MHz, for examination of the cervical arteries. Examination was performed in B mode and Doppler mode imaging.

2.3. Other examinations

Certain laboratory analyses were performed in each patient, as well as echocardiography and CT of the internal organs in some of them. National Institutes of Health Stroke Scale (NIHSS) was used for ischemic stroke evaluation, including its expended version [64], which ranges from 0 (stroke not present) to 42 (the gravest form of stroke). Scores on the Rankin scale were used for patients' clinical evaluation.

All authors have followed the Ethical Principles for Medical Research Involving Human Subjects outlined in the Declaration of Helsinki. All patients, or the members of their families, signed a written consent.

2.4. Statistical analysis

Various methods of descriptive and analytical statistics were applied. Thus, descriptive statistics was used to summarize clinical characteristics of the study group. An assumption of normal distribution was tested using Shapiro Wilk tests ($p > 0.05$ normally distributed data assumed) and Q-Plots. Numerical variables were expressed as a mean \pm SD in case of normal distribution or median (interquartile range) if variable did not follow normal distribution, and as percentages for categorical data. If normal distribution was met, the one-way ANOVA with Tukey correction was used for group comparisons, otherwise the non-parametric Kruskal-Wallis test was applied. Chi-square test was used to compare categorical variables. P-value below 0.05 was considered significant. All analysis was performed with the SPSS statistical analysis software, Version 20.0 (SPSS, Chicago, Illinois, USA).

3. Results

There were 188 patients enrolled during a period of the last 10 years, 53 of whom had vertebral (VAD) and/or basilar dissection (BAD), whilst the remaining 135 showed mainly the internal carotid artery dissection (ICAD). Combinations of VAD and ICAD were not found in the same patient.

3.1. Vertebrobasilar arterial dissection (VBAD)

VBAD in the mentioned 53 patients affected both men (58.5%) and women (41.5%), without a statistical significance ($p = 0.272$), who averaged 52.7 ± 12.7 (range, 33–86) years of age with a peak in the fifth (24.5%) and sixth decade (28.3%). As regards the cause, traumatic dissections or spontaneous ones were noticed, and rarely other causes (Table 1). Traumatic dissections were identified as a direct blunt neck trauma, fall, or hyperextension, lateral flex-

ion or hyperrotation (in car accidents, sports engagement, etc.), or a minor trauma. Other causes (7.5%) mainly included fibromuscular dysplasia, connective tissue diseases, migraine, and respiratory infections. The remaining ones belonged to the mentioned spontaneous (idiopathic) dissections. There were more patients with hypertension than with diabetes, which showed the highest statistical significance ($p = 0.006$ and $p = 0.001$, respectively).

According to MRI examination, 61 dissections were noticed in 53 patients, most often affecting a single VA, i.e. almost in 80% (Fig. 1A and B), with occasional ischemia of the brain stem and/or the cerebellum (Fig. 1C) (Table 1). In some cases, a dissecting aneurysm was observed, either of the VA or of the posterior inferior cerebellar artery – PICA (Fig. 2A–D), which was associated with ischemia. Both the right (VAR) and left (VAL) vertebral arteries were very rarely affected, as well as the basilar artery (BA) alone, which showed an intimal flap, a double lumen, or a pseudoaneurysm (Fig. 3A) causing a pontine infarct (Fig. 3B). Involvement of both VAs and the BA was the least frequent event (Table 1). Dissection had a cervical localization (44.3%), cervicocranial (19.6%), or intracranial (36.1%). The majority of patients complained of headache (69.6%) in VAD and 28.6% in BAD, and neck pain (35.8%) on admission, whilst Horner syndrome was rarely present (8%), as well as some other symptoms and neurological signs (Table 2).

Doppler examination of the cervical VAs (Table 3), which was performed in 51 patients following admission, showed virtually a normal lumen size and normal blood flow in the majority of the right and left VAs (62.7% and 60.8%, respectively). In most of the remaining cases (Table 3) a stenosis was present (25.5% and 23.5%, respectively) and rarely an occlusion (roughly 12% and 16%, respectively), with a concomitant diminished flow or no flow. There were no statistically significant differences between the VAR and VAL alterations.

As regards the MRI signs of 61 dissections in 53 patients, the most frequent MR radiologic sign was intramural hematoma i.e. the semilunar sign, or “eccentric crescent,” in 73.8% patients (Fig. 1A). A “candle flame” sign was observed in cases of the VA occlusion, whilst in some other instances an intimal flap or a double lumen were seen in the VA or the BA. A pseudoaneurysm was noticed in 21.3% of patients (Fig. 3A), as well as signs of stenosis, i.e. string and pearl signs in the remaining patients. There was no significant correlation between the frequency of various signs and the affected arteries, i.e. a single VA, both VAs, and VAs and BA ($p > 0.05$), or infarct occurrence ($p = 0.222$).

Ischemic stroke (IS) was diagnosed in 62.3% and transient ischemic attack (TIA) in 34.0% cases, whilst 3.8% of patients had only isolated symptoms. Atherosclerosis was present in 34%, predominantly in older patients with hypertension. Among the 61 identified dissections, VAD was most often accompanied by the IS in the cerebellum (mean, 32.8%) (Fig. 1C), less frequently in the medulla oblongata, and rarely in the pons, the thalamus, or the occipital region of the cerebral hemispheres (Table 1). BA dissections were most frequently accompanied by pontine infarcts (Table 1) (Fig. 3B). There was a significant correlation between dissection position and infarct location ($p = 0.006$).

Most of patients (66.0%) received antiplatelet therapy, that is, aspirin, dipyridamole, or clopidogrel, and majority of the remaining ones anticoagulant therapy, i.e. heparin or warfarin, usually for several months (32.1%). Surgical procedures were not applied, but in 18.9% of patients a stent was placed (Fig. 2E) or occlusion of a pseudoaneurysm was performed. Recurrent ischemia appeared in 1.9%, and TIA in 5.7% of patients. Radiologic control of luminal recovery was noticed in most of patients. In fact, a corresponding degree of recovery, i.e. between 50% and 100% of the vessel lumen, was observed in 61.7% of patients (Table 4). Clinically, 77.4% patients had good outcome (Rankin score defined

Table 1
Correlation between infarct localization and 61 VA dissection positions.

Arteries	Infarct localization					Total
	Cerebellum	Medulla	Pons	Occipital	Normal*	
Single VA	17 (40.5%)	8 (19.1%)	3 (7.1%)	3 (7.1%)	11 (26.2%)	42 (100%)
Both VAs	3 (25.0%)	3 (25.0%)	0 (0.0%)	0 (0.0%)	6 (50.0%)	12 (100%)
BA or BA& VAs	0 (0.0%)	1 (14.3%)	4 (57.1%)	0 (0.0%)	2 (28.6%)	7 (100%)
Total	20 (32.8%)	12 (19.7%)	7 (11.5%)	3 (4.9%)	19 (31.1%)	61 (100%)

* Patients with TIA and some others with no radiologic signs of ischemia.

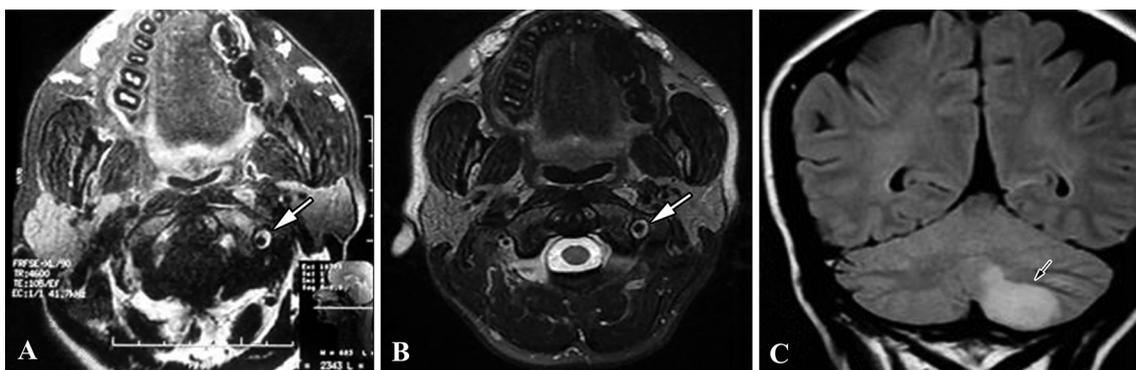


Fig. 1. Axial MRI scans in T2-weighted fat saturation to show the left vertebral artery (arrow) with an intramural hematoma on admission (A) and three months later (B) following anticoagulant therapy. Note an infarction of the left cerebellar hemisphere (arrow) on a coronal MRI scan (C) in the PICA territory.

≤2). Most of the patients were evaluated as having between 0 and 8, i.e. a minor stroke on the NIHSS scale.

3.2. Internal carotid artery dissection

ICAD was present in 135 patients ranging from 27 to 82 years of age (mean, 52.7) with a pick in the fifth decade, but the majority (81.4%) were in the 5th (29.6%), 6th (23.7%), and 7th decades (28.1%). This group consisted of 66.7% males and 33.3% females, which is of a high statistical significance ($p < 0.001$).

Overall, there were 153 dissections in 135 patients. The right internal carotid artery (ICAr) and the left one (ICAl) were almost equally affected, whilst both ICAs were relatively rarely involved (13.3%). The common carotid artery (CCA) was affected in only 2 cases (1.5%). As regards the cause of dissection, trauma (41.5%) and spontaneous dissections (44.4%) were more frequently noticed, and other causes were rarely present (Table 5). There was significantly more patients with hypertension, without diabetes or hyperlipidemia ($p < 0.001$) (Table 5).

Doppler examination of the ICA cervical segments on admission showed a practically normal lumen in a larger number of individuals, but a various degree of stenosis or occlusion was observed in the remaining patients, i.e. in 49.5% and 51.9%, respectively (Table 6). The occlusion itself was noticed in 9.4% and 11.8%, respectively. MR examination revealed a semilunar sign in the majority of patients, followed by a string and pearl sign in some cases, as well as a flame sign in those with an ICA occlusion (Fig. 4A) with a resultant brain ischemia (Fig. 4B). Pseudoaneurysms were found in 28.8% of patients.

Dissection was most often located in the cervical (58.2%) and cervicocranial (subpetrous) region (30.7%), but rarely intracranially (11.1%) (Table 7). In the latter case, the petrous and/or the cavernous segments of the ICA were affected. Headache was the most frequent symptom (42.2%), whilst neck pain was rare (17.8%), as well as Horner's syndrome (14.1%) and amaurosis fugax (11.1%) (Table 2). Ischemic stroke was the most frequent complication of ICA dissection (80%), followed by TIA (20%) (Table 7). The IS was

predominantly present in the territory of the middle cerebral artery (MCA) (Fig. 4B), and then in border zones (watershed infarctions) in the MCA territory, but very rarely in the region of the anterior (ACA) or posterior cerebral arteries (PCA) (Table 8). Usually larger ischemic lesions were diagnosed (Fig. 4B), and rarely lacunar infarcts, i.e. in 7.0% and 16.7% of patients, respectively (Table 8).

In most patients antiplatelet and anticoagulant treatment was given (Table 9). The majority of medicated patients (54.2%) experienced a recanalization or a diminished stenosis of the ICA, whilst in 17.8% a stent was applied, and in 3.0% vascular surgery was performed (Table 9). Recurrent ischemic stroke was not observed, whilst TIA appeared in 1.5% of patients (Table 10). A good outcome (between 0 and 2 on the Rankin scale) was observed in over a half of patients, whilst the majority of them showed a score between 0 and 10 on the NIHSS scale.

3.3. The whole group of patients

Of the 188 patients, 53 experienced a vertebral or basilar artery dissection (28.2%), and the remaining 135 had a carotid dissection (71.8%), which is a statistically significant difference ($p < 0.001$). There were 121 men (64.4%) and 67 women (35.6%) with a statistically significant gender distribution ($p < 0.001$). Patients averaged 53.7 years of age, with a pick in the 5th decade (28.2%), and somewhat less in the 6th (25.0%) and 7th decades (25.5%). Dissection most often affected a single artery (87.2%), predominantly the ICA or the VA, and rarely two or more vessels. All in all, 214 dissections were noticed in 188 patients, either spontaneous or traumatic ones or, less frequently, those of other causes.

As for the arterial portions affected, dissection most often involved cervical segments of the VA and ICA, and less frequently their intracranial portions or branches (Table 7), which is of a high statistical significance ($p < 0.001$). Dissection more often affected the VA intracranial segments (36.1%). As regards the symptoms on admission, headache and neck pain (Table 2) were more frequent in VAD and BAD (69.6% vs. 28.6%) than ICAD (42.2% vs.

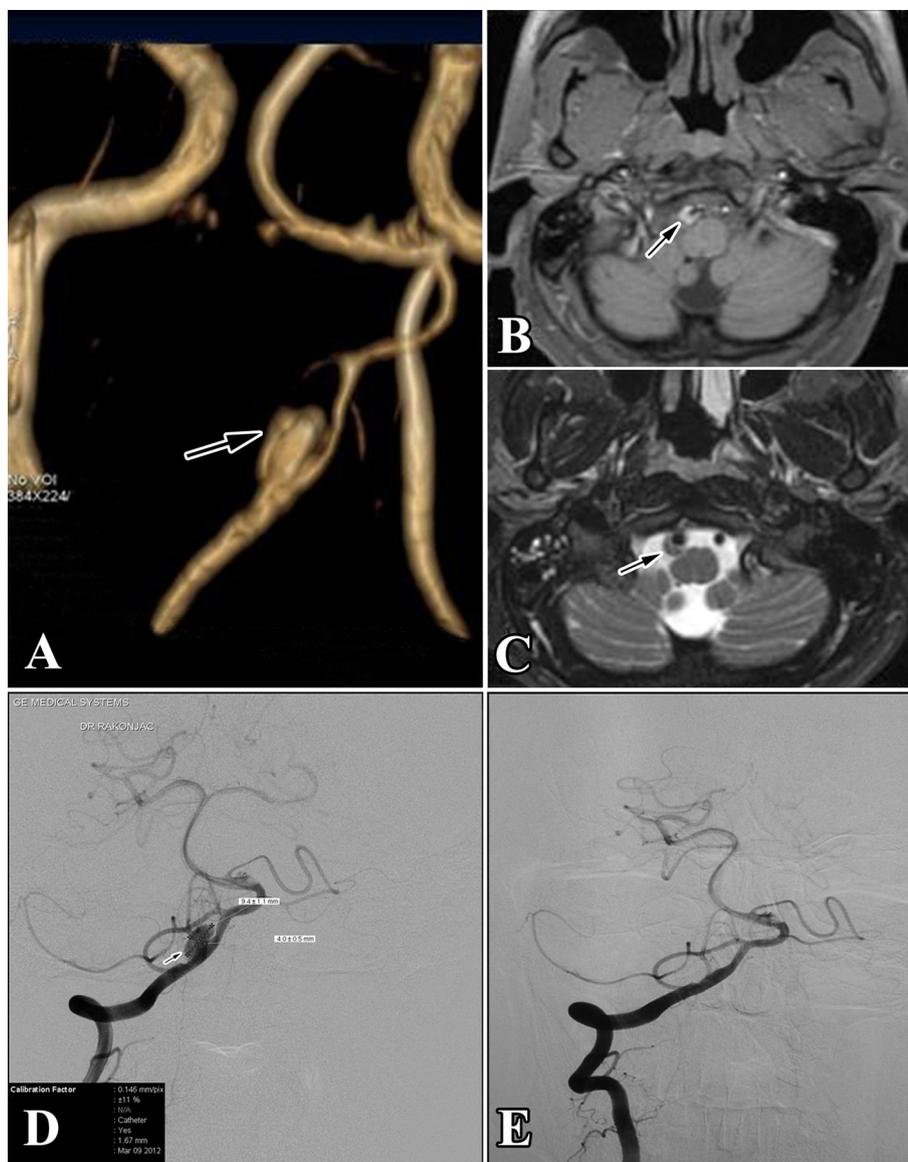


Fig. 2. A pseudoaneurysm (arrow) of the PICA's initial segment on a 3D MRA scan (A), and a dissection (arrow) of the right vertebral artery on a T1 fat saturation (B) and a T2 fat saturation image (C). Note a mild stenosis of the vertebral artery and the mentioned pseudoaneurysm (arrow) on DS angiograms before (D) and after stenting (E).

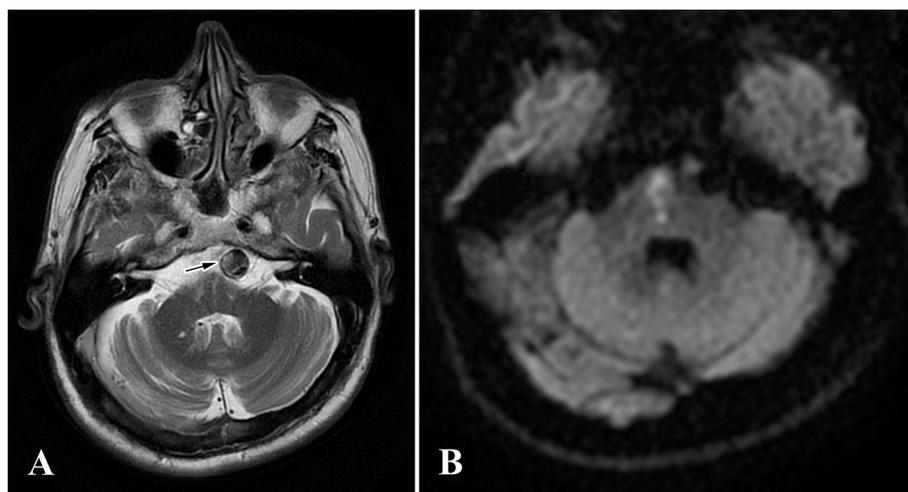


Fig. 3. A dissection and a pseudoaneurysm (arrow) of the basilar artery on a T2-weighted axial MRI scan compressing the pons (A), and a small paramedian pontine ischemic lesions on a DWI axial image (B).

Table 2
Symptomatology and ischemic manifestations in 188 patients.

Arteries involved	Symptoms and signs: No. (%)				Ischemic manifestations: No. (%)		
	headache	Horner's sign	Neck pain	Amaurosis	TIA	Persistent deficit	Symptomless
ICA	57 (42.2)	19 (14.1)	24 (17.8)	15 (11.1)	27 (20)	108 (80)	0 (0)
VA	32 (69.6)	4 (8)	18 (39.1)	0 (0)	17 (37)	28 (60.9)	1 (2.2)
BA	2 (28.6)	0 (0)	1 (14.3)	0 (0)	1 (14.3)	5 (71.4)	1 (14.3)
Total	91 (48.4)	23 (12.2)	43 (22.9)	15 (8.1)	45 (23.9)	141 (75)	2 (1.1)

Table 3
Doppler examination of the cervical VA segments in 51 patients.

Artery	Lumen size				
	Normal	Stenosis (<50%)	Stenosis(50%-70%)	Stenosis(75%-99%)	Occlusion
VAR:No. (%)	32 (62.7%)	9 (17.6%)	3 (5.9%)	1 (2.0%)	6 (11.8%)
VAL: No. (%)	31 (60.8%)	7 (13.7%)	3 (5.9%)	2 (3.9%)	8 (15.7%)

Table 4
Lumen recovery* at 6 months.

Arteries	<50%	Recanalization rate: No. (%)>50%	90–100%	Progression	No recanalisation
VA	11 (9.8)	7 (6.3)	62 (55.4)	2 (1.8)	30 (26.8)
BA	2 (4.9)	4 (9.8)	24 (58.5)	1 (2.4)	10 (24.4)
ICA	0 (0)	1 (20)	4 (80.0)	0 (0)	0 (0)
total	13 (8.2)	12 (7.6)	90 (57.0)	3 (1.9)	40 (25.3)

*It was calculated in 158 patients, since 26 failed to come to a control examination, and 4 patients died.

Table 5
Causes and risk factors of dissections in 188 patients.

Arteries:	Cause: No (%)			Risc factor: No (%)			
	trauma	spontaneous	other	hypertension	diabetes	hyperlipidemia	smoking
ICA	56 (41.5)	60 (44.4)	19 (14.1)	103 (77.0)	26 (19.3)	51 (37.8)	62 (54.9)
VA	22 (47.8)	20 (43.5)	4 (8.7)	30 (65.2)	6 (13.0)	16 (34.8)	20 (37.7)
BA	2(28.6)	5 (71.4)	0 (0)	7 (100)	2 (28.6)	5 (71.4)	3 (5.7)
Total	80(42.6)	85 (45.2)	23(12.2)	140 (74.5)	34(18.1)	72 (38.3)	85 (45.2)

Table 6
Doppler examination of the cervical ICA segments performed in 127 patients.

Artery	Lumen size					
	Stenosis (<50%)	Stenosis (50%-70%)	Stenosis (75%-99%)	Occlusion	Preocclusive	Total
ICAr	5 (3.9%)	4 (3.1%)	9 (7.1%)	15 (11.8%)	30 (23.6%)	63 (49.5%)
ICAI	7 (5.5%)	3 (2.4%)	16 (12.6%)	12 (9.4%)	28 (22.0%)	66 (51.9%)

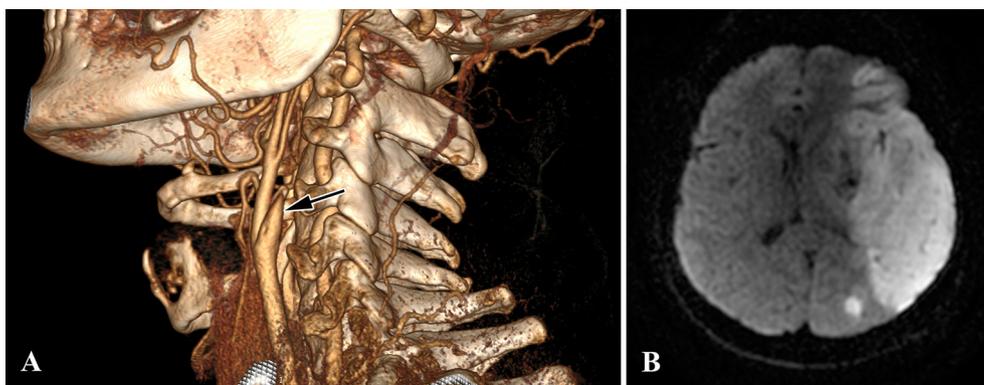


Fig. 4. A flame sign (arrow) of a dissection and tapered occlusion of the left internal carotid artery on a 3D CTA image (A), and a large ischemic region in the territory of the middle cerebral artery on an axial DWI image (B).

Table 7
Radiologic characteristics of the arterial dissection in 214 events.

Arteries	Arterial segments affected: No. (%)		Radiologic signs in TIA & ischemic stroke: No. (%)					
	Cervical & cervico-cranial	Intracranial	Flap/double lumen	Pseudo-aneurysm	Flame sign (occlusion)	String sign	Pearl sign	Semilunar sign/intramural hematoma
ICA	136(88.9)	17 (11.1)	33 (21.6)	44 (28.8)	77 (50.7)	60(39.5)	3 (2)	123 (80.4)
VA	39 (72.2)	15 (27.8)	19 (35.2)	8 (14.8)	30 (55.6)	20(37.0)	3(5.6)	39 (72.2)
BA	0 (0)	7 (100)	6 (85.7)	5 (71/4)	0 (0)	5 (71.4)	0 (0)	6 (85.7)
Total	175(81.8)	39 (18.2)	58 (27.1)	57 (26.6)	107 (50.2)	85(39.9)	6(2.8)	168 (78.5)

Table 8
Distribution of ischemic strokes in patients with carotid dissections.

Artery	Single ICA	Both ICAs	Single CCA
MCA	59.1%	61.1%	0.0%
ACA	5.2%	0.0%	0.0%
PCA	1.7%	5.6%	0.0%
Watershed	11.3%	0.0%	1.6%
Lacunar	7.0%	16.7%	0.0%

17,8%) ($p < 0.008$ and $p < 0.007$, respectively). Only 1.1% of patients were symptomless (Table 2). Finally, we found 26.6% pseudoaneurysms in our patients.

As for a correlation between the MRI signs and IS or TIA (Table 2), ischemic stroke was most often caused by occlusion, i.e. flame sign (Fig. 4A and B) of the ICA, it was less frequently related to the presence of a string sign, and rarely to other radiologic signs. On the other hand, TIA was most frequently associated with existence of a string sign (Table 7). Ischemic stroke caused by VAD most often affected the cerebellum (Table 1) (Fig. 1C), and IS following ICAD was most frequent in the MCA territory (Table 8) (Fig. 4B). Atherosclerosis was diagnosed in 17.5% of patients, more often in VAD than in ICAD patients ($p < 0.001$). Unlike dissection, atherosclerosis was much less frequently manifested with headache.

Headache was the most frequent symptom (42.2%) in ICAD, whilst neck pain was rare (17.8%), as well as Horner's syndrome (14.1%) and amaurosis fugax (11.1%) (Table 2). The majority of VAD and BAD patients complained of headache (69.6% vs. 28.6%) and neck pain (39.1% vs. 14.3%) on admission, Horner syndrome was rarely present in the whole group of patients (12.2%), as well as some other symptoms (Table 2). There was a significant correlation ($p < 0.001$) of the brain ischemic event appearance and hypertension and cigarette smoking, but without a statistical significance in the latter case (Table 5).

The majority of patients received antiplatelet (58.7%) and anticoagulant medication (38.6%), respectively (Table 9), whilst a minority had additional surgical (2.2%) or endovascular interventions – 18.1% (Fig. 2E). The lumen improvement was observed in 72,8% of all patients 6 months following therapy, but almost a complete recovery was achieved in 57% (Table 4). A good outcome (Rankin score ≤ 2) was observed in the majority of patients (61,2%). Most patients had a mild or moderate stroke by evaluating on the NIHSS scale at discharge, with a mean value of 5.57 ± 7.72 , and of 0.67 ± 5.49 regarding delta baseline-discharge NIHSS

Table 9
Acute treatment of patients with dissection.

Arterial	Surgical: No. (%)		Drug treatment: No. (%)		
	Endovascular (stent)	Vascularsurgery	Antiplatelet	Anticoagulant	Notreatment
ICA	24 (17.8)	4 (3)	73 (55.7)	54 (41.2)	4 (3.1)
VA	6 (13.0)	0 (0)	28 (60.9)	17 (37.0)	1 (2.2)
BA	4 (57.1)	0 (0)	7 (100)	0 (0)	0 (0)
Total	34 (18.1)	4 (2.2)	108 (58.7)	71 (38.6)	5 (2.7)

(Table 11). Recurrent ischemic stroke was noticed in 2.6% patients, and recurrent TIA in 3.2% of them 6 months following therapy (Table 10). Mortality was registered in 2.1% of the patients (Table 10).

4. Discussion

Dissection is a rare event, so that the annual incidence of AD is from 1.0 to 5.1 per 100,000 population, but most often between 2.5 and 3 for ICAD, and from 1.3 to 1.5 for VAD [1,8,17,63,65,66]. Due to that, usually smaller samples of patients are presented in literature. Our group of 188 patients is one of the largest reported so far. To our knowledge, only a few authors described much larger groups, i.e. 1958 patients [67], 983 [21], 970 [8,68], and 740 patients [29], respectively.

4.1. The affected arteries

A dissection mechanisms can damage one or more cervical, intracranial or cervicocranial arteries. Our patients mostly experienced the ICA dissection (71.8%), and less frequently the VA dissection (28.2%), which is in agreement with other reports [1,8,57]. However, VAD is somewhat more frequent in younger patients, and in some adult groups [1,58], but especially in the Asian population [65,66]. Besides, the intracranial dissections were more often in the latter population [8,41].

Most frequently, a single artery was affected in our series (87.2%), either ICA, VA or BA. Two-vessel involvement was noticed in 11.7%, either both ICAs, both VAs, or VA and BA. The latter was reported in 18–25% of patients with blunt neck trauma, but less frequently in spontaneous dissections [1,38]. Three arteries were affected in 1.1% of our patients, i.e. BA and both VAs, as compared to 1.5% by some authors [8,24,29,38,40,42,54]. Four-vessel involvement, e.g. both ICAs and VAs, was registered by us in a subsequent patient (in preparation), and by others in 0.1–0.5% [5,23,25,26,28,30,31,35,37–40].

Most often cervical arteries are affected, and less frequently intracranial or cervicocranial vessels, i.e. in 11% to 27% of patients, respectively, although more often in Asian population [1,8,65]. In these instances, the posterior circulation is more frequently involved (76–93%) than the anterior circulation [41].

Table 10
Outcome of patients with dissection.

Arteries	Recurrent event:No. (%)		OutcomeNo. (%)	
	Ischemic stroke	TIA	Alive	Deceased
VA	4 (3.0)	2 (1.5)	131 (97.0)	4 (3.0)
BA	1 (2.2)	3 (6.5)	46 (100)	0 (0)
ICA	0 (0)	1 (14.3)	7 (100)	0 (0)
Total	5 (2.6)	6 (3.2)	184 (97.9)	4 (2.1)

Table 11
Outcome assessment of patients with dissection.

Arteries	Discharge NIHSS					Delta baseline-discharge NIHSS				
	min	max	median	mean	SD	min	max	median	mean	SD
ICA	0	42	4	7.04	8.54	-39	12	1	0.41	6.3
VA	0	7	0	1.37	1.95	-3	7	1	1.28	1.97
BA	0	12	5	4.86	4.20	-4	8	1	1.71	4.31
Total	0	42	5	5.57	7.72	-39	12	1	0.67	5.49

4.2. Predisposing factors and causes

Among the vascular risk factors, hypertension is most often present (26.7%–68.9%), as well as hyperlipidemia (17–51%) and smoking (12.8–52.0%), but less frequently diabetes (2.0–24.9%) and migraine (up to 21.2%) [1,41,50,51,54,57,58,68–71]. Our findings are within this range, except hypertension, which was more frequent due to older age of our patients (Tables 1 and 4).

Blunt trauma as a cause of dissection is reported in 25–40% [19,23,24,33,34,37,38,46,59,72–74], which is somewhat less frequent than in our patients (42.6%) (Table 5). Blunt neck trauma may have either a direct effect, e.g. neck or face blow, cervical or face manipulative therapy, safety belt injuries, an elongated styloid process, a mandible or vertebrae fracture or dislocation, strangulation or hanging [7,30,47,52,53,56,63,73–76], or infrequently penetrating neck injuries [77,78]. The force can predominantly exert an indirect influence, i.e. by neck lateral hyperflexion, hyperextension or contralateral rotation of the head and neck, especially in exercise, sports and traffic accidents, and rarely in dental practice, by turbulent flight or roller coaster riding [6,25,31,38,47,49,59,61,72,73,79–81]. In our patients, mostly a direct blow or minor trauma caused a dissection.

A nondiagnosed minor trauma seems to be much more frequent in “spontaneous” dissections, i.e. up to 40% [1,7,38,46], and even up to 75% in some groups of patients [57]. A minor direct or indirect trauma is mainly related to coughing, sneezing, sexual intercourse, and defecation [45,57,82]. Local and general infections, as well as other types of inflammation, including various types of arteritis, can also result in arterial dissections [48,51,58,83]. Iatrogenic injuries of the cervical and intracranial arteries are possible as well, including local anesthetic injection, and neck, oral, transnasal and skull base surgery [49,55,84]. Certain types of medication can occasionally produce dissections [50,54,85], then arterial elongation [60], fibromuscular dysplasia and connective tissue diseases [8,12,42,86–88], polycystic kidney disease, pregnancy and postpartum [89–91], as well as certain gene mutations [9,86,92–94]. Some of these causes were also observed in our patients. In general, however, dissections are most likely a multifactorial process [1,8].

4.3. Patients and symptomatology

According to various reports, mean age of patients with dissection ranges from 42.0% to 55.3%, which is in agreement with our results (53.7%) [1,8,41,57,62,68]. A somewhat higher averaged

value in our cases is explained by the fact that most of the younger patients were transferred to one of the Vascular or Pediatric Clinics. According to the mentioned authors, males are usually more affected in ICAD (72–75%) than in VAD (about 28%). Similar results were found in our group, i.e. 64.4% vs. 35.6%.

The initial symptoms are commonly related to headache, which appears in 17–85% of patients [57,59,68], and neck pain with a frequency of 25% in ICAD [57], whilst headache and pain together occur in 20.6–75.0% [36,41,62]. Headache and/or neck pain were more frequent in our patients with VAD (69.6% vs. 39.1%) than in those with the right or left ICAD (42% vs. 17.8%) (Table 2), which is similar to some reports [1], but lower in another one [58]. Both symptoms are less frequent in dissections accompanied by atherosclerosis [62,96]. Headache and pain in patients with stroke following dissections appear in 20.6% as the initial symptom [62]. The remaining findings are mainly referred to neurological signs [1,8,57,65,97,98]. Symptomless patients are very rare (0.1–4%), which also was the case in our study (1.1%) (Table 2).

4.4. Radiologic findings

MRI was applied in all patients with dissections, but also CTA, DSA, and Doppler sonography occasionally [14,41,57,99–101]. Occlusion was seen in up to 18%, and a <50% stenosis in 73%. According to some reports [43], intimal flap was present in 22.8%, a flame sign in 15.9%, pearl and string signs in 13.3%, and double lumen in 16.9%. In our patients with stroke following VAD and ICAD, the flame and pearl-and-string signs, that is an occlusion and a multiple stenosis, were obviously much more frequent (Table 7).

Pseudoaneurysms (PsAn) are diagnosed in 15.6–38.0% patients [4,8,11,15,16,28,41,43], and our results (28.8% of the ICA and 21.3% of the VBAD), which were associated with ischemia, are within this range. In general, PsAn are a consequence of the arterial wall weakness, due to fragmentation of the internal elastic lamina, or a rupture of the vasa vasorum [1,8,15]. They can cause ischemic lesions (42%), subarachnoid hemorrhage – SAH (46%), or compression of the brain or cranial nerves in most of the remaining patients. Ischemic lesions may affect various parts of the brain [8,15,69]. SAH is caused by a PsAn rupture [8,16]. Compression of the brain and cranial nerves may cause certain neurological signs. Thus, a BA dissecting aneurysm may compress the pons [46], as was the case in our study (Fig. 3A). Similarly, PsAn of the VA and ICA cervical segments occasionally compress the adjacent autonomic or cranial nerves [12,13,57,97,98,102].

Endovascular treatment is commonly applied in these patients, e.g. a stent or overlapping stents, coils or stent-assisted coils, or flow diverters, in order to prevent rebleeding [15]. Surgical interventions are rarely performed, e.g. wrapping, arterial reconstruction, or proximal ligation [8].

4.5. Ischemia occurrence

It mainly comprises ischemic stroke (IS) of the brain or spinal cord, TIA, retinal infarcts, amaurosis fugax, and certain autonomic or cranial nerves [8,17,36,41,43,62,71,72,76,81,98,103,104]. IS of the brain is present in 30–64% of patients, either as a larger ischemic area or, rarely, as small lacunar lesions [36,69]. IS of the spinal cord appears in 4–10% in those with a VA dissection [10,105]. IS and TIA together occur in 30–78% [8,41,69]. They are more frequent in ICAD (68.9–76.0%) than in VAD or BAD (31.1–38.0%) of adults [1,8], but it is a reverse situation in some pediatric and Asian patients [1,8,41,55,66,68]. In the latter cases, ischemia involves the posterior circulation in up to 77%, and the anterior one in only about 23% [41].

Intracranial VAD and BAD, which are often followed by IS [36,43], are more frequent than intracranial ICAD [8,41]. Nevertheless, VAD may cause the BA occlusion [36,106]. As for IS following ICAD (Table 8), the infarcts are most often located in the territory of the middle cerebral artery (85.1–99.0%), and rarely of the anterior cerebral artery (0.5–7.2%) and posterior cerebral artery (2.0–7.7%) regions [69]. Watershed infarcts are present in 5% of patients, which is less than in our patients (12.9%). VAD is most often followed by cerebellar and medullary infarctions [95,107], as was the case in our study (Table 1). BAD is most frequently associated with pontine infarcts, and rarely with thalamic and occipital lesions [36,108], including our patients (Table 1). Intracerebral hemorrhage is infrequent [62].

IS is most often caused by artery-to-artery embolism (55.0–85.5%), and rarely by a local branch occlusion (31.7%), in situ thrombosis (6.3%), hemodynamic factors (0.8–1.2%), and by a combination of the mentioned factors [36,43].

All in all, dissections are responsible for only 1% to 2% of all ischemic strokes in adults [1,8]. However, dissections in younger patients causes from 10% to 25% of all strokes [1,8].

4.6. Internal carotid versus vertebral and basilar artery dissections

First of all, ICAD is more frequent than VBAD. According to some authors, the former appears in almost 70% and the latter in about 30% [68], which is very similar to our results (71.8% versus 28.2%). ICAD most often appears in older people, and VBAD in younger patients [1,66]. Bilateral ICAD is usually twice as often as VAD [68]. However, intracranial segment of the VA was more often affected (in 36.1% of our patients) as compared to the same ICA segments (11.1%) (Table 7), and it is often accompanied by IS [8,41,65,68]. In the former cases, there is the same situation with pseudoaneurysms and the subarachnoid hemorrhage [8,36,43]. The former appears in 26.6% of the 188 patients. The latter has a higher incidence (27–54%), especially in intracranial VBAD patients (up to 60%) [8,36,41].

Headache and/or neck pain were the most frequent symptoms in both groups of patients, with a frequency up to 80% of cases, both being usually stronger in VAD patients [1,8,57,58,62,68]. In our VAD patients, headache was present in 69.6% and neck pain in 39.1%, whilst smaller values were registered in the ICAD patients (42.2% and 17.8%, respectively) (Table 2). Pseudoaneurysm had a similar frequency in our VBAD (21.3%) and ICAD patients (28.8%).

Ischemic stroke is, in general, more frequent in ICAD (68.9–76.0%) than in VBAD (31.1–38.0%) adults [1,8], which is in agreement with our results, i.e. IS in 60.9% of the VAD patients, and

80% in the ICAD cases) (Table 2). However, it is a reverse situation in young patients and in Asian population [1,41,55,57,66,68]. Nevertheless, lumen recovery was observed in many patients after a few months treatment [1,8,38,57].

4.7. Treatment

Among various medications, i.e. antiplatelet, anticoagulant and thrombolytic medication, as well as surgical repair [1,19,38,57,59], we most often applied antiplatelet therapy (58.7%), and less frequently anticoagulant medication (38.6%) (Table 9). In 17.8% of the ICAD patient and in 18.9% of the VBAD cases, stent was applied, whilst in 3.0% vascular surgery was performed in ICAD, but in none of VBAD, which is similar to other reports [1,13,15,22,38,51,57,77,78].

4.8. Outcome

Good outcome, i.e. between 0 and 2 on the Rankin scale, is seen in up to 79% of patients [1,8,19,21,29,31,57,67,96,103], which is more than in our patients (61.2%), probably due to their older age. Recurrent intracranial dissections appear in 9% of patients [1,109], which is more frequent than in our patients (2.6% with recurrent stroke, and 3.2% with recidivant TIA in the whole group, respectively) (Table 10). Mortality rate is usually between 0% and 9.5%, but most often from 1.6 to 5% [1,8,57,103], which is in agreement with our findings (2.1%) in the whole group (Table 10). Nevertheless, there are no differences in antiplatelet and anticoagulant therapy related to outcome [104].

4.9. Ethnic characteristics

After comparison of our results with other reports, it is obvious that our patients are different in certain features. For instance, a dissection of both ICA and VA or BA was not observed in the same patients, which also was the case in a triple dissection (Table 1). Headache and neck pain were more frequent in our VAD than in the ICAD patients, which is quite opposite to other reports [1,8,57,58,68]. Horner's syndrome was observed only in 14.1% regarding our ICAD patients, and in 8% in VAD ones (Table 2), which is less frequent than in other reports, i.e. 21–25% [57,97,98]. Subarachnoid hemorrhage (SAH) was not present in our patients (since any individual with SAH was transported to the Clinic of Neurosurgery), but it was registered in other reports, even in up to 54% [8,28,41].

Further, watershed infarcts are present in 12.9% of our patients, but only in 5% in the literature. Similarly, recurrent IS or TIA occurred in 2.6% and 3.2%, respectively, in our patients, but up to 16.7% in other ones [1,8]. Mortality rate is mainly up to 5.0% in the reported patients, but only in 2.1% in our patients. Certain racial and ethnic characteristic were already described in the literature [1,8,65,68,95], but the reason for that is unclear [8].

5. Conclusion

In our group of patients, there was a predominance of ICA dissection over VA and BA dissection. One of these vessels was most often affected, whilst two or three of them were rarely observed. There was a significant correlation of dissection, on the one hand, and trauma and vascular risk factors on the other hand. Headache and neck pain were the most frequent symptoms. Ischemic stroke was present in the majority of patients. It mainly affected the cerebral hemispheres, cerebellum, medulla oblongata, and pons. Over 60% of our patients had a good outcome, whilst mortality was noticed in about 2% of them. A great discrepancies in percentages

regarding mortality, certain causes and symptoms can be explained by small groups of the examined patients reported in the literature. Hence, examination of our 188 patients gave reliable and precise results.

CRedit authorship contribution statement

BGB: Conceptualization, Methodology, Writing - review and editing. **TDJ:** Patients' monitoring, Writing - review and editing. **MV:** Data curation, Investigation, Formal analysis, Writing - review and editing. **NS:** Patients' medication and monitoring, Writing - review and editing. **DK:** Methodology, Writing - review and editing. **ML:** Methodology, Writing - review and editing. **IM:** Data analysis, Investigation, Writing - review and editing. **TA:** Patients' medication and monitoring, Writing - review and editing. **AC:** Data curation, Investigation, Formal analysis, Writing - review and editing. **SM:** Conceptualization, Supervision, Data analysis, Writing - review and editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

- [1] Schoen FJ, Cotran RS. The blood vessels. In: Kumar V, Cotran RS, Robbins SL, editors. Robbins basic pathology. Philadelphia: Saunders. An Imprint of Elsevier Science; 2003. p. 325–60.
- [2] Robertson JJ, Koyfman A. Cervical artery dissections: A review. *J Emerg Med* 2016;51:508–18.
- [3] Mehdi E, Aralasmak A, Toprak H, et al. Craniocervical dissections: Radiologic findings, pitfalls, mimicking diseases: A pictorial review. *Curr Med Imaging Rev* 2018;14:207–22.
- [4] DeFatta RJ, Verret DJ, Bauer P. Extracranial internal carotid artery pseudoaneurysm. *Int J Pediatr Otorhinolaryngol* 2005;69:1135–9.
- [5] Rees JH, Valentine AR, Llewelyn JG. Spontaneous bilateral carotid and vertebral artery dissection presenting as a Collet-Sicard syndrome. *Br J Radiol* 1997;70(836):856–8.
- [6] Arat YO, Volpi J, Arat A, et al. Bilateral internal carotid artery and vertebral artery dissections with retinal artery occlusion after a roller coaster ride - case report and a review. *Ulus Trauma Act Cerrahi Derg* 2011;17(1):75–8.
- [7] Creavin ST, Rice CM, Pollentine A, et al. Carotid artery dissection presenting with isolated headache and Horner syndrome after minor head injury. *Am J Emerg Med* 2012;30(9):2103.e5–e7.
- [8] Debette S, Compter A, Labeyrie MA, et al. Epidemiology, pathophysiology, diagnosis, and management of intracranial artery dissection. *Lancet Neurol* 2015;14:640–54.
- [9] Debette S, Kamatani Y, Metso TM, et al. Common variation in PHACTR1 is associated with susceptibility to cervical artery dissection. *Nat Genet* 2015;47:78–83.
- [10] Montalvo M, Bayer A, Azher I, et al. Spinal cord infarction because of spontaneous vertebral artery dissection. *Stroke* 2018;49:e314–7.
- [11] Cruciata G, Parikh R, Pradhan M, et al. Internal carotid artery dissection and pseudoaneurysm formation with resultant ipsilateral hypoglossal nerve palsy. *Radiol Case Rep* 2017;12:371–5.
- [12] Herath HMMTB, Pahalagamage SP, Withana D, et al. Complete ophthalmoplegia, complete ptosis and dilated pupil due to internal carotid artery dissection: as the first manifestation of Takayasu arteritis. *BMC Cardiovasc Disord* 2017;17:201.
- [13] Tsuboki S, Kawano T, Ohmori Y, Amadatsu T, Mukasa A. Surgical treatment of spontaneous internal carotid artery dissection with abducent nerve palsy: Case report and review of literature. *World Neurosurg* 2019;125:10–4.
- [14] Hassen BW, Machet A, Edjlali-Goujon M, et al. Imaging of cervical artery dissection. *Diagn Interv Imaging* 2014;95:1151–61.
- [15] Derelle AL, Barbier C, Tonnelet R, et al. Three cases of ruptured basilar artery dissection: from diagnosis to endovascular treatment. *World Neurosurg* 2016;91:676.e1–e7.
- [16] Zhang Y, Wang Y, Sui B, et al. Magnetic resonance imaging follow-up of large or giant vertebralbasilar dissecting aneurysms after total embolization on angiography. *World Neurosurg* 2016;91:218–27.
- [17] Marciniak M, Sapko K, Kulczyński M, et al. Non-traumatic cervical artery dissection and ischemic stroke: A narrative review of recent research. *Clin Neurol Neurosurg* 2019;187:105561.
- [18] Guillon B, Lévy C, Bousser MG. Internal carotid artery dissection: an update. *J Neurol Sci* 1998;153(2):146–58.
- [19] Biffi WL, Ray Jr CE, Moore EE, et al. Treatment-related outcomes from blunt cerebrovascular injuries: importance of routine follow-up arteriography. *Ann Surg* 2002;235(5):699–706.
- [20] Stock K, Peters S, Behrmann C, Spielman RP. Bilateral carotid dissection. A not to underestimate cause of neurological loss after road accident. *Unfallchirurg* 2002;105(10):919–22 [In German].
- [21] Béjot Y, Aboa-Eboulé C, Debette S, et al. CADISP Group. Characteristics and outcomes of patients with multiple cervical artery dissection. *Stroke* 2014;45(1):37–41.
- [22] Coric D, Wilson JA, Regan JD, Bell DA. Primary stenting of the extracranial internal carotid artery in a patient with multiple cervical dissections: technical case report. *Neurosurgery* 1998;43(4):956–9.
- [23] Fukuda I, Meguro K, Matsusita S, et al. Traumatic disruption of bilateral vertebral arteries and internal carotid arteries: case report. *J Trauma* 1989;29(2):263–6.
- [24] Busch T, Aleksic I, Sirbu H, et al. Complex traumatic dissection of right vertebral and bilateral carotid arteries: a case report and literature review. *Cardiovasc Surg* 2000;8(1):72–4.
- [25] Nadgir RN, Loevner LA, Ahmed T, et al. Simultaneous bilateral internal carotid and vertebral artery dissection following chiropractic manipulation: case report and review of the literature. *Neuroradiology* 2003;45(5):311–4.
- [26] Yong RL, Heran NS. Traumatic carotid cavernous fistula with bilateral carotid artery and vertebral artery dissections. *Acta Neurochir (Wien)* 2005;147(10):1109–13.
- [27] Campos CR, Evaristo EF, Yamamoto FI, et al. Spontaneous cervical carotid and vertebral arteries dissection: study of 48 patients. *Arq Neuropsiquiatr* 2004;62(2B):492–8 [In Portuguese].
- [28] Marshman LAG, Ball L, Jadun CK. Spontaneous bilateral carotid and vertebral artery dissections associated with multiple disparate intracranial aneurysms, subarachnoid hemorrhage and spontaneous resolution. Case report and literature review. *Clin Neurol Neurosurg* 2007;109(9):816–20.
- [29] Arnold M, De Marchis GM, Stapf C, Baumgartner RW, Nedeltchev K, Buffon F, et al. Triple and quadruple spontaneous cervical artery dissection: presenting characteristics and long-term outcome. *J Neurol Neurosurg Psychiatry* 2009;80(2):171–4.
- [30] Chakrapani AL, Zink W, Zimmerman R, et al. Bilateral carotid and bilateral vertebral artery dissection following facial massage. *Angiology* 2009;59(6):761–4.
- [31] Leach JCD, Malham GM. Complete recovery following atlantoaxial fracture-dislocation with bilateral carotid and vertebral artery injury. *Br J Neurosurg* 2009;23(1):92–4.
- [32] Keilani ZM, Berne JD, Agko M. Bilateral internal carotid and vertebral artery dissection after a horse-riding injury. *J Vasc Surg* 2010;52(4):1052–7.
- [33] Abuzayed B, Aydin S, Bozkus S, et al. Traumatic carotid artery dissection and bilateral vertebral artery occlusion after a horse attack: an unusual combination and etiology. *J Neurol Surg A Cent Eur Neurosurg* 2012;73(1):53–5.
- [34] Hamida N, Hakim A, Fourati H, et al. Neonatal cervical artery dissection secondary to birth trauma. *Arch Pediatr* 2014;21(2):201–5 [In French].
- [35] Abe A, Nito C, Sakamoto Y, et al. Spontaneous bilateral cervical internal carotid and vertebral artery dissection in a Japanese patient without collagen vascular disease with special reference to single-nucleotide polymorphisms. *J Stroke Cerebrovasc Dis* 2016;25(8):e114–7.
- [36] Chen H, Hong H, Xing S, et al. Intracranial versus extracranial artery dissection cases presenting with ischemic stroke. *J Stroke Cerebrovasc Dis* 2015;24:852–9.
- [37] Coss C, Jones J. Bilateral carotid and vertebral artery dissection from blunt trauma. *Case Rep Emerg Med* 2018:1919034.
- [38] Ariyada K, Shibahashi K, Hoda H, et al. Bilateral internal carotid and left vertebral artery dissection after blunt trauma: A case report and literature review. *Neurol Med Chir (Tokyo)* 2019;59:154–61.
- [39] Golub D, Hu L, Dogra S, et al. Spontaneous bilateral internal carotid and vertebral artery dissections with dominant-hemisphere circulation maintained by external carotid artery-ophthalmic artery anastomoses. *Neurosurg Focus* 2019;46(2):E6.
- [40] Guglielmi V, Visser J, Arnold M, et al. Triple and quadruple cervical artery dissections: a systematic review of individual patient data. *J Neurol* 2019;266(6):1383–8.
- [41] Nakamura Y, Yamaguchi Y, Makita N, et al. Clinical and radiological characteristics of intracranial artery dissection using recently proposed diagnostic criteria. *J Stroke Cerebrovasc Dis* 2019;28:1691–702.
- [42] Ohshima T, Miyachi S, Isaji T, et al. Bilateral vertebral artery dissection and unilateral carotid artery dissection in case of Ehlers-Danlos syndrome type IV. *World Neurosurg* 2019;121:83–7.
- [43] Kwon JY, Kim NY, Suh DC, et al. Intracranial and extracranial arterial dissection presenting with ischemic stroke: Lesion location and stroke mechanism. *J Neurol Sci* 2015;358:371–6.
- [44] Li L, Li T, Xue J, et al. Stent treatment for basilar artery dissection: A single-center experience of 21 patients. *Interv Neuroradiol* 2016;22:260–5.
- [45] Kim SH, Lee YS, Suh SJ, et al. Acute pontine infarction due to basilar artery dissection from strenuous physical effort: One from sexual intercourse and another from defecation. *J Cerebrovasc Endovasc Neurosurg* 2016;18:100–5.
- [46] Murabit A, Tredget EE. Blunt carotid artery injury after minor facial trauma. *Can J Plast Surg* 2012;20:194–6.
- [47] Halfpap JP, Cho AA, Rosenthal MD. Cervical spine fracture with vertebral artery dissection. *J Orthop Sports Phys Ther* 2016;46:929.

- [48] Marangi A, Moretto G, Cappellari M, et al. Bilateral internal carotid artery dissection associated with prior syphilis: a case report and review of the literature. *Neuropsychiatr Dis Treat* 2016;12:1351–4.
- [49] Molad JA, Findler M, Bornstein NM, Auriel E. Symptomatic acute cervical artery dissection following dental procedure - Case series. *J Neurol Sci* 2016;368:184–6.
- [50] Perez DJ. Spontaneous carotid artery dissection. *JAAPA* 2017;30:27–9.
- [51] Racchiusa S, Longo M, Bernava G, et al. Endovascular treatment of spontaneous intracranial internal carotid dissection in a young patient affected by systemic lupus erythematosus: A case report. *J Vasc Interv Neurol* 2017;9:1–7.
- [52] Smoot TW, Taha A, Tarlov N, et al. Eagle syndrome: A case report of stylocarotid syndrome with internal carotid artery dissection. *Interv Neuroradiol* 2017;23:433–6.
- [53] Alboudi AM, Sarathchandran P, Geblawi SS. Delayed presentation of neck arteries dissection, caused by water slide activity. *BMJ Case Rep* 2018;11(1):e226333.
- [54] Chung SE, Yoon TH, Lee KM, et al. A case report of multiple cervical artery dissection after peripheral type facial palsy and use of steroids. *BMC Neurol* 2018;18:74.
- [55] Narula N, Siddiqui F, Katyal N, et al. Internal carotid artery dissection with lidocaine nerve block injection trauma: A rare case report. *Cureus* 2018;10:e2027.
- [56] Hu Y, Du J, Liu Z, et al. Vertebral artery dissection caused by atlantoaxial dislocation: a case report and review of literature. *Childs Nerv Syst* 2019;35:187–90.
- [57] Rao AS, Makaroun MS, Marone LK, et al. Long-term outcomes of internal carotid artery dissection. *J Vasc Surg* 2011;54:370–4.
- [58] Thomas LC, Rivett DA, Attia JR, Levi AC. Risk factors and clinical presentation of cervical arterial dissection: preliminary results of a prospective case-control study. *J Orthop Sports Phys Ther* 2015;45:503–11.
- [59] Kray JE, Dombrovskiy VY, Vogel TR. Carotid artery dissection and motor vehicle trauma: patient demographics, associated injuries and impact of treatment on cost and length of stay. *BMC Emerg Med* 2016;16:23.
- [60] Kim ST, Brinjilki W, Lehman VT, et al. Association between carotid artery tortuosity and carotid dissection: a case-control study. *J Neurosurg Sci* 2018;62:413–7.
- [61] Walters GK. Cervicocranial artery dissection and scuba diving: Is there a link or is it serendipity? *Undersea Hyperb Med* 2018;45:65–73.
- [62] Wang Y, Cheng W, Lian Y. The headache and neck pain in ischemic stroke patients caused by cervicocerebral artery dissection. A case-control study. *J Stroke Cerebrovasc Dis* 2019;28(3):557–61.
- [63] Saw AE, McIntosh AS, Kountouris A. Vertebral artery dissection in sport: Expert opinion of mechanisms and risk-reduction strategies. *J Clin Neurosci* 2019;68:28–32.
- [64] Olivato S, Nizzoli S, Cavazzuti M, et al. e-NIHSS: an expanded National Institutes of health Stroke Scale weighted for anterior and posterior circulation strokes. *J Stroke Cerebrovasc Dis* 2016;25(12):2953–7.
- [65] Shibahara T, Yasaka M, Wakugawa Y, et al. Improvement and aggravation of spontaneous unruptured vertebral artery dissection. *Cerebrovasc Dis Extra* 2017;7:153–64.
- [66] Nash M, Rafay MF. Craniocervical arterial dissection in children: Pathophysiology and management. *Pediatr Neurol* 2019;95:9–18.
- [67] Compter A, Schilling S, Vaineau CJ, et al. Determinants and outcome of multiple and early recurrent cervical artery dissections. *Neurology* 2018;91(8):e769–80.
- [68] von Babo M, De Marchis GM, Sarikaya H, Stapf C, Buffon F, Fischer U, et al. Differences and similarities between spontaneous dissections of the internal carotid artery and the vertebral artery. *Stroke* 2013;44(6):1537–42.
- [69] Shimoyama T, Kimura K, Iguchi Y, et al. Spontaneous intra-cranial arterial dissection frequently causes anterior cerebral artery infarction. *J Neurol Sci* 2011;304(1–2):40–3.
- [70] Lyden PD. Migraine and the risk of carotid artery dissection in the IPSYS registry: Are they related? *JAMA Neurol* 2017;1(74):503–4.
- [71] McNally JS, Hinckley PJ, Sakata A, Eisenmenger LB, Kim S-E, De Havenon AH, et al. Magnetic resonance imaging and clinical factors associated with ischemic stroke in patients suspected of cervical artery dissection. *Stroke* 2018;49(10):2337–44.
- [72] Lin JJ, Chou ML, Lin KL, et al. Cerebral infarct secondary to traumatic carotid artery dissection. *Pediatr Emerg Care* 2007;23:166–8.
- [73] Molacek J, Baxa J, Houdek K, et al. Bilateral post-traumatic carotid dissection as a result of a strangulation injury. *Ann Vasc Surg* 2010;24:1133.e9–e11.
- [74] López-Sánchez M, Ballesteros-Sanz MA, Pérez-Ceballos A, et al. Traumatic dissection of the internal carotid artery by a safety belt: a report of two cases. *Med Intensiva* 2009;33:353–7.
- [75] Aspalter M, Linni K, Domenig CM, et al. Successful repair of bilateral common carotid artery dissections from hanging. *Ann Vasc Surg* 2013;27(1186):e7–e15.
- [76] Biller J, Sacco RL, Albuquerque FC, et al. Cervical arterial dissections and association with cervical manipulative therapy: a statement for healthcare professionals from the American heart association/American stroke association. *Stroke* 2014;45:3155–74.
- [77] Yevich SM, Lee SR, Scott BG, et al. Emergency endovascular management of penetrating gunshot injuries to the arteries in the face and neck: a case series and review of the literature. *J Neurointerv Surg* 2014;6:42–6.
- [78] Brzezicki G, Meyer TE, Madbak F, et al. Nail gun injury and endovascular repair of cervical internal carotid artery. *Cureus* 2019;11:e4237.
- [79] Quinn C, Cooke J, O'Connor M, et al. Cervical artery dissection following a turbulent flight. *Aviat Space Environ Med* 2011;82:995–7.
- [80] Jatuzis D, Valaikiene J, Pallionis D, Mataciunas M. Migrain-like presentation of vertebral artery dissection after cervical manipulative therapy. *Cerebrovasc Dis (Basel)* 31(Suppl/1):P36–P36.
- [81] Ercoli T, Dagostino S, Pierri V, et al. Internal carotid artery dissection causing ischemic stroke during pole sport practice. *J Sports Med Phys Fitness* 2019;59:892–3.
- [82] Carey C. Post-tussive carotid artery dissection: could it be whooping cough? *CMAJ* 2014;186:697.
- [83] Ishikawa H, Shindo A, Li Y, et al. Vertebral artery dissection associated with familial Mediterranean fever and Behçet's disease. *Ann Clin Transl Neurol* 2019;6:974–8.
- [84] Rowan N, Turner M, Valappil B, Fernandez-Miranda J, Wang E, Gardner P, et al. Injury of the carotid artery during endoscopic endonasal surgery: Surveys of skull base surgeons. *J Neurol Surg B Skull Base* 2018;79(03):302–8.
- [85] Demetrius JS. Spontaneous cervical artery dissection: a fluoroquinolone induced connective tissue disorder? *Chiropr Man Therap* 2018;26:22.
- [86] Henrard C, Belge H, Fastré S, et al. Cervical artery dissection: fibromuscular dysplasia versus vascular Ehlers-Danlos syndrome. *Blood Press* 2019;28:139–43.
- [87] Rosner AL. Spontaneous cervical artery dissections and implications for homocysteine. *J Manipulative Physiol Ther* 2004;27:124–32.
- [88] Alurkar A, Karanam LSP, Oak S, Sorte S. Carotid dissection in Marfan's syndrome. *Neurol India* 2013;61:206–7.
- [89] Shanmugalingam R, Reza Pour N, Chuah SC, Vo TM, Beran R, Hennessy A, et al. Vertebral artery dissection in hypertensive disorders of pregnancy: a case series and literature review. *MC Pregnancy Childbirth* 2016;16(1). <https://doi.org/10.1186/s12884-016-0953-5>.
- [90] Kuroki T, Yamashiro K, Tanaka R, et al. Vertebral artery dissection in patients with autosomal dominant polycystic kidney disease. *J Stroke Cerebrovasc Dis* 2014;23(10):e441–3.
- [91] Ulrich N, Johnson A, Jodry D, et al. Resolution of internal carotid dissection with middle cerebral artery occlusion in pregnancy. *Case Rep Neurol Med* 2015;2015:398261.
- [92] Pénişon-Besnier I, Lebouvier T, Moizard MP, et al. Carotid artery dissection in an adult with the Simpson-Golabi-Behmel syndrome. *Am J Med Genet A* 2008;146A:464–7.
- [93] Talarowska P, Dobrowolski P, Klisiewicz A, Kostera-Pruszyk A, Członkowska A, Kurkowska-Jastrzębska I, et al. High incidence and clinical characteristics of fibromuscular dysplasia in patients with spontaneous cervical artery dissection: The ARCADIA-POL study. *Vasc Med* 2019;24(2):112–9.
- [94] Tashiro R, Fujimura M, Sakata H, et al. Genetic analysis of ring finger protein 213 (RNF213) c.14576G>A polymorphism in patients with vertebral artery dissection: a comparative study with moyamoya disease. *Neurol Res* 2019;41(9):811–6.
- [95] Inamasu J, Nakae S, Kato Y, Hirose Y. Clinical characteristics of cerebellar infarction due to arterial dissection. *Asian J Neurosurg* 2018;13:995–1000.
- [96] Chien C, Chang F-C, Huang H-C, Tsai J-Y, Chung C-P. Characteristics and outcomes of vertebrobasilar artery dissection with accompanied atherosclerosis. *Cerebrovasc Dis Extra* 2017;7(3):165–72.
- [97] Kikkeri NS, Nagarajan E, Sakuru RC, Bollu PC. Horner syndrome due to spontaneous internal carotid artery dissection. *Cureus* 2018;10(9):e3382.
- [98] Song JX, Lin XM, Hao ZQ, et al. Ocular manifestations of internal carotid artery dissection. *Int J Ophthalmol* 2019;12(5):834–9.
- [99] Yang L, Ran H. The advantage of ultrasonography in the diagnosis of extracranial vertebral artery dissection: Two case reports. *Medicine (Baltimore)* 2017;96(12):e6379.
- [100] Tan HW, Chen X, Maingard J, Barras CD, Logan C, Thijs V, et al. Intracranial vessel wall imaging with magnetic resonance imaging: Current techniques and applications. *World Neurosurg* 2018;112:186–98.
- [101] Yilmaz C, Gorgulu FF, Oksuzler FY, Arslan M, Oksuzler M, Keskek SO, et al. Color Doppler ultrasonography is a reliable diagnostic tool in the diagnosis of extracranial vertebral artery dissections. *J Med Ultrason* 2019;46(1):153–8.
- [102] English SW, Passe TJ, Lindell PE, James P, Klaas JP. Multiple cranial neuropathies as a presentation of spontaneous internal carotid artery dissection: A case report and literature review. *J Clin Neurosci* 2018;50:129–31.
- [103] Chang FC, Yong CS, Huang HC, et al. Posterior circulation ischemic stroke caused by arterial dissection: Characteristics and predictors of poor outcomes. *Cerebrovasc Dis* 2015;40(3–4):144–50.
- [104] Markus HS, Levi C, King A, Madigan J, Norris J. Antiplatelet therapy vs. anticoagulation therapy in cervical artery dissection: The Cervical Artery Dissection in Stroke Study (CADISS) Randomized Clinical Trial Final Results. *JAMA Neurol* 2019;76(6):657. <https://doi.org/10.1001/jamaneurol.2019.0072>.
- [105] Schievink WI, Björnsson J, Piepgras DG. Coexistence of fibromuscular dysplasia and cystic medial necrosis in a patient with Marfan's syndrome and bilateral carotid artery dissections. *Stroke* 1994;25:2492–6.
- [106] Kuan C-Y, Hung K-L. Vertebral artery dissection complicated by basilar artery occlusion. *Pediatr Neonatol* 2014;55(4):316–9.
- [107] Lance S, Thomson T. Cerebellar nodulus infarction secondary to vertebral artery dissection. *BMJ Case Rep* 2019;12(4):e229876.

- [108] Ashida S, Nagakane Y, Makino M, Tomonaga K, Makita N, Yamamoto Y. Ischemic stroke with vertebrobasilar artery dissection extended to posterior cerebral artery. *Rinsho Shinkeigaku* 2017;57(8):446–50.
- [109] Kato T, Yagi T, Yoshioka H, et al. Simultaneous onset of anterior and middle cerebral artery dissections with an old vertebral artery dissection. *J Stroke Cerebrovasc Dis* 2013;22:1229–32.