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Internal carotid and vertebral artery dissections — a comparison of clinical, radiological and prognostic characteristics

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ABSTRACT

Aim of study. To examine whether baseline characteristics, potential risk factors, clinical symptoms, radiological presentation, and long-term outcomes differ between internal carotid artery dissection (ICAD) and vertebral artery dissection (VAD).

Clinical rationale for study. Cervical artery dissection (CeAD) is a major cause of cerebral ischaemia in young adults. Its clinical course is highly variable, resulting in challenges in making a proper diagnosis.

Methods. We performed a retrospective analysis of 31 patients (mean age 42.2 years) with CeAD (18 with ICAD, 13 with VAD) treated in our neurology department from 2008 to 2018. Appropriate imaging confirmed the diagnosis of CeAD.

Results. Patients with ICAD presented Horner syndrome significantly more often (44.4% vs 7.6%; p = 0.04). Patients with VAD more often had ischaemic events (ischaemic stroke, TIA or transient blindness) (84.6% *vs* 44.6%; p = 0.0032). Ischaemic stroke was more severe in patients with ICAD [(median NIHSS 6, interquartile range 4–12) vs VAD (median NIHSS 4, interquartile range 1.5–5.5), p = 0,03]. Occlusion occurred more often in patients with VAD (69.2% *vs* 22.2%; p = 0.013). Most patients had a favourable outcome (mRS 0–2).

Conclusions and clinical implications: In a series of patients with CeAD, we observed significant differences between VAD and ICAD in terms of clinical symptoms and radiological features.

Key words: dissection, stroke, risk factor, carotid artery, vertebral artery

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Introduction

Although stroke is usually reported in older people, an increasing incidence of this condition has been observed in younger adults (45 years or less) [1]. Of a variety of causes, cervical artery dissection (CeAD) is proven to be one of the leading causes of ischaemic stroke in this group of patients, with a frequency of up to 20% [2–5]. The great majority of cases occur spontaneously, with only 0.86% of dissections associated with severe trauma [6].

CeAD can be further classified into vertebral artery dissection (VAD) or internal carotid artery dissection (ICAD), depending on the vessel involved [7]. These two forms differ in terms of clinical manifestations. ICAD typically begins with ipsilateral neck pain or headache combined with partial Horner syndrome that is characterised only by miosis, ptosis and enophthalmos, not anhidrosis. Progression of the disease leads to ischaemia of the central nervous system (CNS) [8]. VAD, on the other hand, is characterised by occipito-cervical pain and posterior circulation ischaemic symptoms such as nausea, vertigo and ataxia [2, 4, 9].

The diagnosis of CeAD is based on the detection of a mural haematoma using magnetic resonance imaging (MRI), which is regarded as the first-line screening tool whenever CeAD is suspected [10]. The consequences of mural haematoma differ considerably. In sub-intimal dissections, the haematoma leads to stenosis, or even occlusion, due to the compression of arterial lumen. Unlike sub-intimal dissection, arterial lumen

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remains preserved in sub-adventitial dissections. However, the arterial outer wall expands outwards, causing a pseudo-aneurysm that can result in conflict with adjacent structures including cranial nerves or ocular sympathetic fibres [5, 10].

In a case of sudden neurological symptoms, patients should be treated with standard acute ischaemic stroke protocols which involve the administration of IV r-tPA within 4.5 hours of stroke onset and / or mechanical thrombectomy within 24 hours [11]. The management of other CeAD cases remains inconsistent. However, anticoagulants or antiplatelets are used interchangeably to prevent further thromboembolic events with similar efficacy [2, 4, 9]. According to the latest study by Markus et al., there are no differences in efficacy between antiplatelets and anticoagulants [12].

Clinical rationale for study

It is well known that cervical artery dissection is one of the major causes of stroke, especially in young patients. Better understanding of putative risk factors, as well as clinical and radiological presentation, is vital for early diagnosis. To the best of our knowledge, our study is the first to investigate similarities and differences between ICAD and VAD in Polish patients, not to mention the fact that no multicentre study such as CADISP has included Poland. Therefore, we feel our study will be valuable for clinical practice.

Methods

In this retrospective study, we gathered clinical data from the medical records of patients who visited a tertiary referral hospital between 1 February 2008 and 31 January 2018 and who had a confirmed diagnosis of CeAD. The study included 31 patients: 18 patients with ICAD and 13 with VAD. One patient with concomitant dissection of the aortic arch apart from ICAD was excluded.

The patients' medical records were assessed in order to obtain the following clinical characteristics: the presence of subarachnoid haemorrhage, transient ischaemic attack (TIA) or ischaemic stroke at admission; signs suggesting cerebral ischaemia or pure local signs; National Institutes of Health Stroke Scale (NIHSS) (a diagnostic method for quickly assessing the severity of a cerebral stroke); putative risk factors for CeAD such as hypertension, diabetes mellitus, hypercholesterolaemia, recent history of trauma, history of smoking, migraine, history of coronary and peripheral artery disease, and connective tissue disorder, such as Ehlers-Danlos syndrome, Marfan syndrome, pseudoxanthoma elasticum, osteogenesis imperfecta, 1-antitrypsin deficiency and a family history of CeAD or vertebral artery dissection (VAD).

In all patients, the diagnosis of CeAD was confirmed by carotid ultrasound with colour Doppler and neuroimaging techniques including MRI, computerised angiography (CT), and conventional angiography. The following radiological findings were recorded on dissected arteries at admission: stenosis, defined by a narrowing of the arterial lumen; arterial occlusion, defined by the absence of blood flow; aneurysmal dilatation, defined by a focal enlargement of the arterial lumen and external diameter; intramural haematoma, defined by thickening of the arterial wall with imaging features consistent with bleeding within the thickened wall; and multiple dissections, defined by the coexisting presence of a recent dissection on more than one cervical artery.

The functional long-term outcome according to the modified Rankin Scale (mRS) was assessed in 29 patients on the basis of information acquired during routine follow up visits after one year in the ambulatory unit of our hospital.

This study was approved by the Ethics Committee of the Medical University of Lublin.

Statistical analysis was carried out with Gretl 2018d and STATISTICA 13.1. We compared potential risk factors, clinical and radiological characteristics between ICAD and VAD. Mean and SD of continuous variables were calculated. Differences of continuous variables were assessed with Mann–Whitney U-test, and differences of categorical variables with Fisher exact test. Statistical significance was set at $p < 0.05$.

Results

Of 31 patients (45.1% men), 18 (58.1%) presented with ICAD and 13 (41.9%) with VAD. Mean age at diagnosis was similar for both groups: 41.4 ± 10.58 for ICAD and 43.3 ± 10.13 for VAD [range 21 to 70]. Different kinds of trauma preceded dissection in 13 patients: a car accident in one, working for several hours with a reclined head in three, swimming, running, dancing in one patient each, being hit on the neck in two, and lifting a heavy object in four patients. None of the patients had a family history of CeAD. Neither baseline characteristics, nor probable risk factors showed a significant association with dissection site. The main data concerning the abovementioned issues is summarised in Table 1. The table does not include rare potential risk factors which were present in isolated cases, i.e. diabetes, Marfan syndrome or fibromuscular dysplasia. All of these are associated with ICAD.

Both the ICAD and the VAD patients most often presented with headache and/or neck pain (ICAD 83.3% *vs* VAD 84.6% $p = 1$; Tab. 2). Horner syndrome (as a local sign) was taken into account only in patients who did not present brainstem infarction. Patients with ICAD had Horner syndrome significantly more often than patients with VAD (44.4% *vs* 7.7%; $p = 0.04$). One patient with ICAD presented with abnormal taste perception (dysgeusia). Patients with VAD significantly more often presented with vertigo than patients with ICAD (61.5% *vs* 5.5%; $p = 0.012$). Problems with coordination were also significantly more frequent in VAD (69.2% *vs* 5.5%; $p = 0.0003$). Nystagmus and vomiting presented only in patients with VAD. Cerebral ischaemic events (i.e. ischaemic

Table 1. Baseline characteristics and probable risk factors associated with dissection site (ICAD *vs* VAD)

	All patients	Patients with ICAD	Patients with VAD	P univariate
Number, (%)	31	18 (58.1)	13 (41.9)	-
Age, mean ± SD	42.2 (±10.43)	41.4 (± 10.58)	43.3 (± 10.13)	0.39
Male sex, n (%)	14 (45.1)	6 (33.3)	8 (61.5)	0.16
Hypertension, n (%)	13 (41.9)	7 (38.9)	6 (46.1)	0.72
History of smoking, n (%)	11 (35.5)	6 (33.3)	5 (38.5)	1
Hypercholesterolaemia, n (%)	13 (41.9)	7 (38.9)	6 (46.1)	0.72
Migraine, n (%)	6 (19.3)	3 (16.7)	3 (23)	0.67
Hormonal contraconception, n (% of females)	6 (35.2)	4 (22.2)	2 (15.4)	1
Trauma, n (%)	13 (41.9)	9 (50)	4 (30.7)	1

Table 2. Clinical presentation according to dissection site (ICAD *vs* VAD)

	All patients	Patients with ICAD	Patients with VAD	P univariate
Number (%)	31	18 (58.1)	13 (41.9)	-
Headache and / or neck pain, n (%)	26 (83.8)	15 (83.3)	11 (84.6)	1
Horner syndrome, n (%)	9 (29)	8 (44.4)	1 (7.7)	0.04
Tinnitus, n (%)	6 (19.35)	4 (22.2)	2 (15.38)	1
Hemiparesis / hemiparalysis, n (%)	11 (35.4)	7 (38.8)	4 (30.7)	0.71
Aphasia, n (%)	8 (25.8)	5 (27.7)	3 (23.07)	1
Decreased / loss of consciousness, n (%)	8 (25.8)	4 (22.2)	4 (30.7)	0.68
Sensory deficits, n (%)	8 (25.8)	4 (22.2)	4 (30.7)	1
Vertigo, n (%)	9 (29)	1 (5.5)	8 (61.5)	0.0012
Vomiting, n (%)	6 (19.3)	0 (0)	6 (46.1)	0.000
Nystagmus, n (%)	7 (22.5%)	0 (0)	7 (53.8)	0.000
Problems with coordination, n (%)	10 (32.2)	1 (5.5)	9 (69.2)	0.0003
Cerebral ischaemia, n (%)	19 (61.3)	8 (44.4)	11 (84.6)	0.032
Ischaemic stroke, n (%)	14 (45.1)	6 (54.5)	8 (61.5)	0.16
Transient ischaemic attack, n (%)	4 (12.9)	2 (11.1)	2 (15.4)	1
Transient blindness, n (%)	4 (12.9)	2 (11.1)	2 (15.4)	1
Subarachnoid haemorrhage, n (%)	1 (3.2)	1 (5.5)	0 (0)	0.000
NIHSS score, median (interquartile range)	4 (3.75–7.25)	6 (4-12)	4 (1.5–5.5)	0.03
Favourable outcome mRS 0–2 (patients with ischaemic stroke), n (%)	12 (85.7)	5 (83.3)	7 (87.5)	1

stroke, transient ischaemic attack, transient blindness) affected patients with VAD significantly more often than patients with ICAD (84.6% *vs* 44.4%; p = 0.032). Although not significant, ischaemic stroke occurred more often in patients with VAD (61.5% *vs* 54.5%; p = 0.16). NIH Stroke Scale (NIHSS) score at admission was significantly higher in patients with ICAD than in patients with VAD [median $NIHSS_{ICAD}$ 6 (4 *vs* 12) *vs* $NIHSS_{VAD}$ 4 (1.5–5.5), p = 0.03].

There was no significant difference in the long-term outcome measured by mRS among patients who suffered ischaemic stroke. The functional outcome was favourable in 83.3% of patients with ICAD and in 87.5% of patients with VAD. Every patient survived. None of the patients presented recurrent CeAD or ischaemic event after hospitalisation.

The main data concerning radiological findings at admission according to the dissection site is summarised in Table 3. Mural

Table 3. Radiological findings at admission according to dissection site (ICAD *vs* VAD)

	All patients	Patients with ICAD	Patients with VAD	p
Number	32	18 (58.1)	13 (41.9)	-
Bilateral dissection, n (%)*	3 (9.4)	2 (11.1)	1 (7.7)	1
Stenosis, n (%)*	16 (50)	12 (66.7)	4 (30.7)	0.073
Arterial occlusion, n (%)*	13 (40.6)	4 (22.2)	9 (69.2)	0.013
Mural haematoma, n (%)*	22 (68.75)	14 (77.8)	8 (61.5)	0.43

*More than one feature can be present simultaneously in the same patient

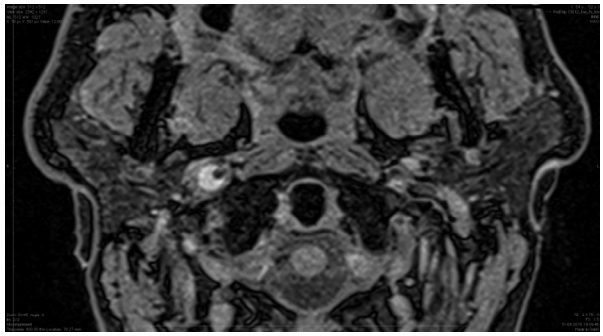

Figure 1. Intramural haematoma of right internal carotid artery on MRI in T1-dependent spin echo (SE) sequence with fat signal saturation (FATSAT)

haematoma was the most frequently observed radiological finding in both groups (Fig. 1). Whereas the distribution of stenosis and mural haematoma did not differ significantly, arterial occlusion occurred more often in patients with VAD (69.2% *vs* 22.2%; p = 0.013) (Fig. 2). Bilateral dissection was rarely seen, occurring only in three cases. Aneurysmal dilatation was not found in any patient.

Discussion

In our study we describe differences between ICAD and VAD in terms of baseline characteristics, probable risk factors, clinical presentations, radiological findings at admission, and outcomes measured by mRS after one year.

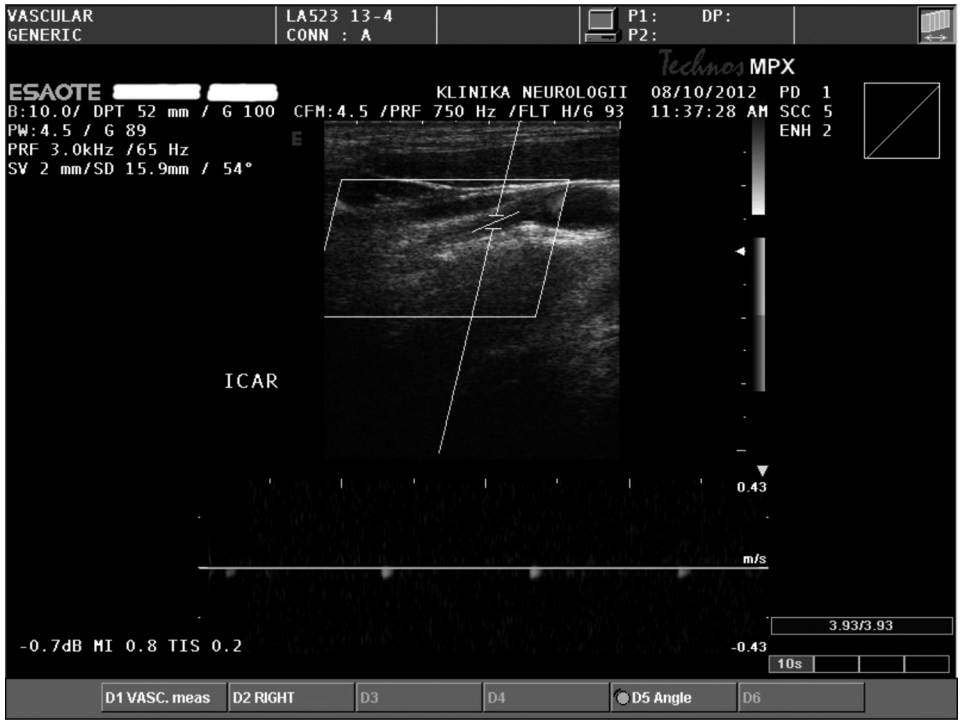

Figure 2. Occlusion of right internal carotid artery in Doppler Duplex examination in a patient

Baseline characteristics and probable risk factors

ICAD occurred more frequently, which is in line with previous findings [13–15]. Unlike multicentre studies, we did not observe significant differences in age or sex between patients with ICAD and VAD. This might result from the relatively small sample size compared to those studies [7, 13, 16, 17].

None of the probable risk factors proved to be significantly associated with the dissection site. Nonetheless, previous reports have differed in terms of risk factors. In a recent study, migraine was found to be associated with CeAD [18]. However, other multicentre studies did not report an association between migraine and the dissection site [7, 13, 17]. Associations between modifiable risk factors for stroke, i.e. hypertension, hypercholesterolaemia, smoking, the use of oral contraceptives and the site of dissection, are inconsistent in the literature [19]. As far as smoking is concerned, some authors have reported significantly lower frequency of smoking in ICAD than VAD, unlike the study by Dziewas et al. that found no difference [13, 17, 20]. Minor cervical trauma in the previous month was significantly more frequent in VAD according to Debette el al. [7]. But the frequency was almost the same according to other studies [13, 20]. In our study, almost half of the patients had sustained a traumatic event before the onset of neurological signs. Trauma, especially severe episodes, might lead to the disruption of the vessel wall [6]. Hypertension, hypercholesterolaemia and hormonal contraconception have not been seen to differ significantly depending on the dissection site [7, 13, 20]. According to the literature, these factors are thought not to predispose to CeAD [17, 20, 21]. Interestingly, hypercholesterolaemia was even shown to be inversely associated with CeAD in a large series of patients [22].

Clinical presentation

The most frequently encountered syndrome was headache and/or neck pain observed in 83.8% of patients, which is in line with the literature [14, 20, 23]. In almost every patient with cerebral ischaemia, this preceded the onset of neurological deficits, again in agreement with the literature [21]. This pain might result from the irritation of nerves surrounding carotid and vertebral arteries [24, 25].

In line with other studies, Horner syndrome was present as a local sign significantly more often in patients with ICAD than with VAD [8, 13, 15]. Its presence in ICAD is believed to result from the compression of pericarotid sympathetic plexus by the enlarged artery, while in VAD it is thought to be a sign of brainstem stroke [8, 15]. Dissections in carotid artery seem to more often be sub-adventitial, compressing structures adjacent to the outer wall of the artery [7, 21].

One patient with ICAD presented with an especially rare manifestation, i.e. an abnormal perception of taste without other cranial nerve involvement. This might result from a close anatomical relation between the chorda tympani nerve and the internal carotid artery [26, 27].

In agreement with multicentre studies, cerebral ischaemia was less frequently observed by us in ICAD than VAD. Nevertheless, ischaemic strokes in ICAD were more severe compared to VAD. Although not significant, a trend towards a higher frequency of ischaemic stroke in VAD was also observed in our study [7, 13].

Patients with VAD significantly more often presented with vertigo and problems with motor coordination. Furthermore, nystagmus and vomiting were observed only in the VAD group. These findings indicate posterior circulation deficits, specifically Wallenberg syndrome. According to the literature, VAD is the most common cause of Wallenberg syndrome in younger patients [28].

Radiological findings and long-term outcomes

In agreement with the CADISP study, mural haematoma was the most frequently encountered radiological feature in both of our groups, appearing in almost four out of five patients [7]. We observed that patients with VAD more often had arterial occlusion than did patients with ICAD.

To the best of our knowledge, this is a new finding, as previous studies did not find any significant differences between occlusion and dissection site. Our observation seems to prove that dissections in the vertebral artery are mainly sub-intimal, expanding toward an arterial lumen, thus causing ischaemic events. According to the literature, there is an association between the involvement of subintimal region in VAD and the occurrence of cerebral ischaemia [29]. Furthermore, some papers have highlighted the association between subintimal haematoma and extracranial dissection, which is by far the most common form of VAD. Changes in subadventitia might be, presumably, more often in intracranial VAD, which was not noted in our study [30, 31]. However, we must remember that there are other plausible explanations for the higher count of occlusions, such as lower lumen or lower blood flow velocity in vertebral arteries [32].

The long-term prognosis of carotid artery dissection (mRS) was favourable in most patients, which is in line with other studies [14, 20].

Strengths and limitations

Our study was limited by the relatively small number of patients and the lack of a control group of age- and sex-matched individuals. These limitations result respectively from the low incidence of CeAD in the general population, and the difficulties with case-control design because of the young age of these patients.

Nevertheless, to the best of our knowledge, our study is the first to present similarities and differences between CAD and VAD in Polish patients. Furthermore, none of the multicentre studies such as the CADISP study has included Poland. Thus, our study gives insight into the clinical profiles of patients with VAD and CAD dissections in Poland.

Future directions

Carotid and vertebral artery dissections, which together constitute a rare cause of ischaemic events, exhibit similarities as well as differences. They differ in their clinical characteristics and radiological features. Early diagnosis of these dissections is crucial due to the high frequency of ischaemic events, especially VAD.

Although our study sheds light on this major cause of stroke in young people, much larger studies including many centres, not only university hospitals, should be conducted for the better analysis of this disease.

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References

1. Smajlović D. Strokes in young adults: epidemiology and prevention. Vasc Health Risk Manag. 2015; 11: 157–164, doi: 10.2147/VHRM.S53203, indexed in Pubmed: 25750539.
2. Blum CA, Yaghi S. Cervical Artery Dissection: A Review of the Epidemiology, Pathophysiology, Treatment, and Outcome. Arch Neurosci. 2015; 2(4), doi: 10.5812/archneurosci.26670, indexed in Pubmed: 26478890.
3. Mohan IV. Current optimal assessment and management of carotid and vertebral spontaneous and traumatic dissection. Angiology. 2014; 65(4): 274–283, doi: 10.1177/0003319712475154, indexed in Pubmed: 23401625.
4. Robertson JJ, Koyfman A. Cervical Artery Dissections: A Review. J Emerg Med. 2016; 51(5): 508–518, doi: 10.1016/j.jemermed.2015.10.044, indexed in Pubmed: 27634674.
5. Schelfaut D, Dhondt E, De Raedt S, et al. Carotid artery dissection: three cases and a review of the literature. Eur J Emerg Med. 2012; 19(3): 181–187, doi: 10.1097/MEJ.0b013e328349ee6d, indexed in Pubmed: 21817907.
6. Taoussi N, Alghamdi AJ, Bielewicz J, et al. Traumatic bilateral dissection of cervical internal carotid artery in the wake of a car accident: A case report. Neurol Neurochir Pol. 2017; 51(5): 432–438, doi: 10.1016/j.pjnns.2017.07.002, indexed in Pubmed: 28743388.
7. Debette S, Grond-Ginsbach C, Bodenant M, et al. Cervical Artery Dissection Ischemic Stroke Patients (CADISP) Group. Differential features of carotid and vertebral artery dissections: the CADISP study. Neurology. 2011; 77(12): 1174–1181, doi: 10.1212/WNL.0b013e31822f03fc, indexed in Pubmed: 21900632.
8. Baumgartner RW, Arnold M, Baumgartner I, et al. Carotid dissection with and without ischemic events: local symptoms and cerebral artery findings. Neurology. 2001; 57(5): 827–832, doi: 10.1212/wnl.57.5.827, indexed in Pubmed: 11552012.
9. Robertson JJ, Koyfman A, Robertson JJ, et al. Cervical Artery Dissections: A Review. J Emerg Med. 2016; 51(5): 508–518, doi: 10.1016/j.jemermed.2015.10.044, indexed in Pubmed: 27634674.
10. Ben Hassen W, Machet A, Edjlali-Goujon M, et al. Imaging of cervical artery dissection. Diagn Interv Imaging. 2014; 95(12): 1151–1161, doi: 10.1016/j.diii.2014.10.003, indexed in Pubmed: 25632417.
11. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2019; 50(12): e344–e418, doi: 10.1161/STR.0000000000000211, indexed in Pubmed: 31662037.
12. Markus HS, Levi C, King A, et al. Cervical Artery Dissection in Stroke Study (CADISS) Investigators. 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): 657–664, doi: 10.1001/jamaneurol.2019.0072, indexed in Pubmed: 30801621.
13. von Babo M, De Marchis GM, Sarikaya H, et al. Differences and similarities between spontaneous dissections of the internal carotid artery and the vertebral artery. Stroke. 2013; 44(6): 1537–1542, doi: 10.1161/STROKEAHA.113.001057, indexed in Pubmed: 23632978.
14. Lee VH, Brown RD, Mandrekar JN, et al. Incidence and outcome of cervical artery dissection: a population-based study. Neurology. 2006; 67(10): 1809–1812, doi: 10.1212/01.wnl.0000244486.30455.71, indexed in Pubmed: 17130413.
15. Lyrer PA, Brandt T, Metso TM, et al. Cervical Artery Dissection and Ischemic Stroke Patients (CADISP) Study Group. Clinical import of Horner syndrome in internal carotid and vertebral artery dissection. Neurology. 2014; 82(18): 1653–1659, doi: 10.1212/WNL.0000000000000381, indexed in Pubmed: 24727317.
16. Metso TM, Debette S, Grond-Ginsbach C, et al. Age-dependent differences in cervical artery dissection. J Neurol. 2012; 259(10): 2202–2210, doi: 10.1007/s00415-012-6485-7, indexed in Pubmed: 22527225.
17. von Sarnowski B, Schminke U, Grittner U, et al. Cervical artery dissection in young adults in the stroke in young Fabry patients (sifap1) study. Cerebrovasc Dis. 2015; 39(2): 110–121, doi: 10.1159/000371338, indexed in Pubmed: 25634656.
18. De Giuli V, Grassi M, Lodigiani C, et al. Italian Project on Stroke in Young Adults Investigators. Association Between Migraine and Cervical Artery Dissection: The Italian Project on Stroke in Young Adults. JAMA Neurol. 2017; 74(5): 512–518, doi: 10.1001/jamaneurol.2016.5704, indexed in Pubmed: 28264095.
19. Arboix A. Cardiovascular risk factors for acute stroke: Risk profiles in the different subtypes of ischemic stroke. World J Clin Cases. 2015; 3(5): 418–429, doi: 10.12998/wjcc.v3.i5.418, indexed in Pubmed: 25984516.
20. Dziewas R, Konrad C, Dräger B, et al. Cervical artery dissection––clinical features, risk factors, therapy and outcome in 126 patients. J Neurol. 2003; 250(10): 1179–1184, doi: 10.1007/s00415-003-0174-5, indexed in Pubmed: 14586598.
21. Debette S. Pathophysiology and risk factors of cervical artery dissection: what have we learnt from large hospital-based cohorts? Curr Opin Neurol. 2014; 27(1): 20–28, doi: 10.1097/WCO.0000000000000056, indexed in Pubmed: 24300790.
22. Debette S, Metso T, Pezzini A, et al. Cervical Artery Dissection and Ischemic Stroke Patients (CADISP) Group. Association of vascular risk factors with cervical artery dissection and ischemic stroke in young adults. Circulation. 2011; 123(14): 1537–1544, doi: 10.1161/CIRCULATIONAHA.110.000125, indexed in Pubmed: 21444882.

23. Thomas LC, Rivett DA, Parsons M, et al. Risk factors and clinical features of craniocervical arterial dissection. Man Ther. 2011; 16(4): 351–356, doi: 10.1016/j.math.2010.12.008, indexed in Pubmed: 21256072.
24. Sheikh HU. Headache in Intracranial and Cervical Artery Dissections. Curr Pain Headache Rep. 2016; 20(2): 8, doi: 10.1007/s11916-016-0544-1, indexed in Pubmed: 26757710.
25. Caplan LR. Dissections of brain-supplying arteries. Nat Clin Pract Neurol. 2008; 4(1): 34–42, doi: 10.1038/ncpneuro0683, indexed in Pubmed: 18199995.
26. Hülsbömer HB, Steinke W. [Taste disorder caused by carotid artery dissection]. Nervenarzt. 2001; 72(8): 629–631, doi: 10.1007/s001150170063, indexed in Pubmed: 11519204.
27. Mokri B, Silbert PL, Schievink WI, et al. Cranial nerve palsy in spontaneous dissection of the extracranial internal carotid artery. Neurology. 1996; 46(2): 356–359, doi: 10.1212/wnl.46.2.356, indexed in Pubmed: 8614494.
28. Lui F, Tadi P, Anilkumar AC. Wallenberg Syndrome. StatPearls Publishing. ; 2019.
29. Lee JS, Yong SW, Bang OhY, et al. Comparison of spontaneous intracranial vertebral artery dissection with large artery disease. Arch Neurol. 2006; 63(12): 1738–1744, doi: 10.1001/archneur.63.12.1738, indexed in Pubmed: 17172613.
30. Ali MS, Amenta PS, Starke RM, et al. Intracranial vertebral artery dissections: evolving perspectives. Interv Neuroradiol. 2012; 18(4): 469–483, doi: 10.1177/159101991201800414, indexed in Pubmed: 23217643.
31. Zhang G, Chen Z. Medical and Interventional Therapy for Spontaneous Vertebral Artery Dissection in the Craniocervical Segment. Biomed Res Int. 2017; 2017: 7859719, doi: 10.1155/2017/7859719, indexed in Pubmed: 28321414.
32. Spasojević G, Vujmilović S, Vujković Z, et al. Internal carotid and vertebral arteries diameters and their interrelationships to sex and left/right side. Folia Morphol (Warsz). 2020; 79(2): 219–225, doi: 10.5603/FM.a2019.0071, indexed in Pubmed: 31257563.