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Neurorehabilitation in stroke produced by vertebral artery dissection: case presentation

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Abstract
Arterial dissections are a common cause of stroke in the young (mean age 44 to 46 years). Primary lesion is a tear of the arterial intima, which promotes platelet aggregation, thrombus formation, which further produced vessel stenosis / occlusion, distal embolism or vessel wall rupture. Vertebral artery (VA) dissection appear most commonly in extracranial segments V2 and V3, and could be spontaneous (with underlying predispositions) or triggered by various traumatisms. Clinicaly, VA dissection produces an ischemic stroke or transient ischemic attack , preceded by local symptoms such as neck pain or headache. The diagnosis is confirmed by neurovascular imaging. Treatment of symptomatic VA dissections respect indications of treatment in ischemic strokes. Prognosis is mostly favorable in extracranial dissections.
We present the case of a left VA dissection in V2 segment, produced by physical effort (swimming), which causes 2 ischemic lesions, one in the territory of the left posterior cerebral artery and the other in the territory of the left posterolateral chorroidal artery. Patient's treatment included antiplatelet agents, statines, and an adapted physical rehabilitation program. At three months he showed significant clinical improvement with regain of autonomy and partial recanalisation at angio-MRI of the V2 segment of the dissected artery.
Key words: *vertebral artery, extracranial dissection, cervical arteries, ischemic stroke, rehabilitation*

Introduction:

Arterial dissections are a common cause of stroke in the young (accounts for up to one quarter of all stroke cases in the young), but may occur at any age. Dissection occurs when structural integrity of the arterial wall is compromised, allowing blood to collect between layers as an intramural hematoma, causing a false lumen into the vessel wall.
Incidence of spontaneous vertebral artery dissection is 0.97 per 100,000 people [1], with a global incidence of cervico-cerebral dissections of 3 cases per 100,000 people. The mean age of individuals affected by dissection was 44 to 46 years, with a slight male predominance [2]. Extracranial dissection is far more frequent than intracranial dissection in reports from North America and Europe [3].
Pathologically, the primary lesion is a tear of the arterial intima, which exposes the collagen fibers and promotes platelet aggregation and thrombus formation, which may occlude the vessel. The intimal lesion could remain stable, or complications may appear. The consequences of an arterial dissection are: stenosis or occlusion of the artery – if the dissection is subintimal- and a dissecting aneurysm of the vessel wall – if the dissection is subadventitial.
Vertebral artery (VA) dissection most commonly appear in extracranial segments: V2 segment, where the artery travels through the cervical transverse processes of C6 to C2, and V3 segment, between the transverse process of C2 and the foramen magnum at the base of the skull [4]. Causes of VA dissection include various degrees of traumatisms or spontaneous events, with underlying predispositions in some cases. Some specific physical activities have been described to be associated with VA dissection: skating, tennis, basketball, volleyball, swimming, scuba diving, dancing, yoga or vigorous exercises.
Clinicaly, VA dissection most often produces an ischemic stroke or transient ischemic attack in the vertebrobasilary system, usually preceded by local symptoms such as neck pain or headache. A latent period between the time of injury and the appearance of clinical manifestations is typically seen (time required for thrombus formation with decrease of the distal blood flow or distal embolization). There are asymptomatic reported cases of VA dissections, or with local signs only.

The risk of ischemic stroke is greater in the first 2 weeks after dissection, with an absolute increase in stroke risk of 1.25% in this interval [5].

The diagnosis is confirmed by neuroimaging techniques, with noninvasive MR angiography and CT angiography replacing conventional arteriography. Pathognomonic crescent sign of intramural hematoma, formed by an eccentric rim of hyperintensity surrounding a hypointense arterial lumen, is described on T1-weighted fat-saturation MRI sequences [6].

Treatment of extracranial VA dissection with signs of ischemic stroke recommends use of thrombolytic therapy for eligible patients in the first 4,5 hours from symptom onset [7], and endovascular treatment is indicated in selected cases [8]. Antithrombotic treatment should be administrated to all patients with acute ischemic neurologic symptoms caused by extracranial VA dissection (24 hours delay after trombolytic therapy). Either antiplatelet or anticoagulant treatment should be given, but the evidence suggests no advantage of anticoagulation over antiplatelet treatment [9]. Duration of antithrombotic therapy is three to six months [10].

For patients with nonischemic symptoms caused by extracranial cervical dissection, evidences suggest the use of antiplatelet therapy for primary prevention of ischemic stroke.

The prognosis of VA dissection depends on the severity of associated vertebrobasilary stroke. Good recovery occurs in 75-80% of patients with extracranial cervical arteries dissection [11].

Rehabilitation therapy is an important tool for improving the neurological status of patients after a vertebrobasilary stroke produced by vertebral artery dissection. Different methods of rehabilitation could be used, depending of stroke location and neurological main symptoms. There are no specific rehabilitation methods used in strokes caused by cervical artery dissection. The most common methods are:

1. Physical therapy is helpful in ameliorating balance (vestibular reconditioning exercises in vestibular syndrome), coordination (in cerebellar syndrome produced by VA dissection) , gait and motor deficits (caused by pyramidal tract damage),
2. Speech therapy is used for improvement of swalloving difficulties (caused by medullary lesions)
3. Swallowing therapy will improve language troubles (dysarthria or dysphonia)

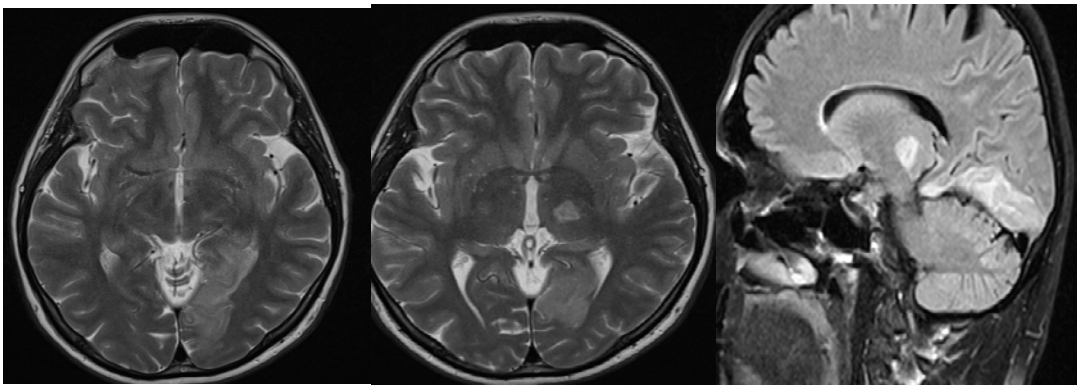
Case presentation:

We present the case of an 51 year old male, very active, with no vascular risk factors, non smoker, which accused during the seaside holiday, pulsating, left side hemicrania and left cervical pain, few hours after a episode of vigorous swimming against sea waves resistance. The same evening he presents to Emergency Department, accusing weakness and clumsiness of his right hand. His blood pressure was 140/80 mmHg, his heart rate was rhythmic, 82 b/min. He has no fever, no carotid bruits, no other abnormalities at general examination.

His neurologic examination shows mild facial asymmetry (mild right central facial palsy), motor deficit of his right upper and lower limbs (force MRC of 4/5), brisk osteotendinous reflexes on his right side, right Babinski sign, ataxia on his right side of the body and mild dysarthria. His gait was ataxic, possible only with assistance of another person. An emergency CT scan was not contributive and did not show any ischemic lesion. Blood tests shows mildly elevated fasting serum glucose and normal cholesterol levels.

The patient was admitted in the Neurology Department. Ultrasound examination of his cervical vessels showed a smaller left vertebral artery with absence of flow in V2 segment, suggestive of a stenosis of thrombosis. The cardiac examination (ECG, transthoracic echocardiography, 24 h HolterECG monitoring) was unremarquable.

A cerebral MRI was obtained, which shows an acute ischemic lesion in the left PCA (posterior cerebral artery territory) and another ischemic lesion in the left postero-lateral chorroidal artery territory **(**Figure 1**).**



1.a. 1.b. 1.c.

Fig.1: Axial T2 MRI (1.a., 1.b.) and sagital T2 MRI (1.c.) showing ischemia in left-PCA and left-postero-lateral chorroideal artery territories

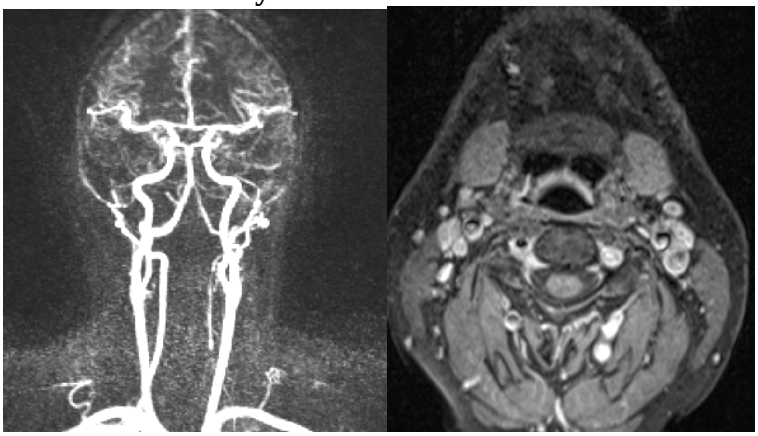


Figure 2: Angio-MRI showing irregular flow in V2 and V3 sergments of left –VA

Figure 3: Contrast MRI showing thrombosis of left VA in V2 segment

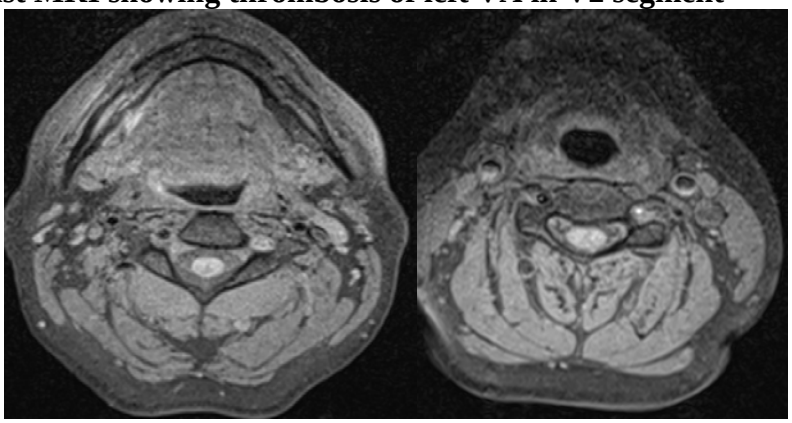


Figure 4: Fat Sat cervical MRI sequences showing intramural hematoma leftVA

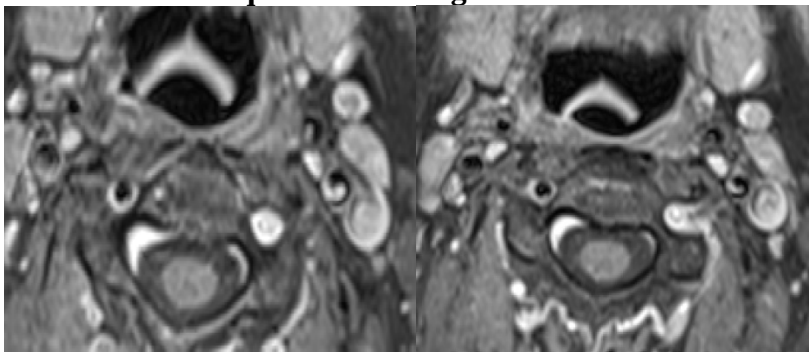


Figure 5: Cervical MRI follow-up al 3 months (with contrast) showing thrombus in the left VA

Angio MRI showed decreased flow in the V2 and V3 segments of left VA (Figure 2, Figure3).

On MRI with T1 weighting with suppression of the fat sequences, sickle shape hyperintensity (corresponding to the intramural hematoma) was observed around the V2 segment of left VA, confirming left VA dissection (Figure 4). The patient was treated with antiplatelet drugs (aspirin), statins, and perindopril (for persistent mildly elevated BP values during hospitalization). The rehabilitation program was started the fourth day after stroke and consisted in physical exercises aimed to improve coordination, balance, gait and upper limb muscle force, with mild intensity and short duration initially. The physical parameters of the patient improved during 14 day hospitalization, and the patient was discharged in good condition, with mild ataxia of right limbs, but with unassisted gait. The patient was included in a rehabilitation program consisting in daily physical exercises, of 1 hour per day, performed in an outpatient rehabilitation facility. Lifestyle changes recommendation were also monitored during this 3 month period.

At three months, patient's neurological condition is much improved: he has only mild right ataxia. His blood pressure and serum glicemia levels were normalized [12, 13].

Ultrasound examination of cervical vessels showed the same aspect (absence of flow in V2 segment).

Cervical MRI with contrast also showed presence of thrombus in the V2 segment of the left VA, without recanalisation (figure 5)**.**

Conclusions

Patient's evolution at three months showed clinical improvement, despite lack of recanalisation of the occluded left vertebral artery. The improvement in neurological function, despite lack of recanalisation of the occluded artery, could be, in part, attributed to the intense physical rehabilitation program in which the patient was included.

Vertebral artery dissections are a frequent cause of stroke in the young.

The good prognosis and favourable outcome can be improved by including the patient in a regular rehabilitation program, aiming to compensate the restant neurological deficit.

Bibliography

1. Lee VH, Brown RD Jr, Mandrekar JN, Mokri B. Incidence and outcome of cervical artery dissection: a population-based study. Neurology. 2006;67(10):1809
2. Arnold M, Kappeler L, Georgiadis D, Berthet K, Keserue B, Bousser MG, Baumgartner RW. Gender differences in spontaneous cervical artery dissection. Neurology. 2006;67(6):1050
3. Kim BM, Kim SH, Kim DI, Shin YS, Suh SH, Kim DJ, Park SI, Park KY, Ahn SS. Outcomes and prognostic factors of intracranial unruptured vertebrobasilar artery dissection. Neurology. 2011;76(20):1735
4. Arnold M, Bousser MG, Fahrni G, Fischer U, Georgiadis D, Gandjour J, Benninger D, Sturzenegger M, Mattle HP, Baumgartner RW. Vertebral artery dissection: presenting findings and predictors of outcome. Stroke. 2006;37(10):2499
5. Morris NA, Merkler AE, Gialdini G, Kamel H. Timing of Incident Stroke Risk After Cervical Artery Dissection Presenting Without Ischemia. Stroke. 2017 Mar;48(3):551-555
6. Choi KD, Jo JW, Park KP, Kim JS, Lee TH, Kim HJ, Jung DS. Diffusion-weighted imaging of intramural hematoma in vertebral artery dissection. J Neurol Sci. 2007;253(1-2):8
7. Qureshi AI, Chaudhry SA, Hassan AE, Zacharatos H, Rodriguez GJ, Suri MF, Lakshminarayan K, Ezzeddine MA. Thrombolytic treatment of patients with acute ischemic stroke related to underlying arterial dissection in the United States. Arch Neurol. 2011;68(12):1536
8. Fields JD, Lutsep HL, Rymer MR, Budzik RF, Devlin TG, Baxter BW, Malek R, Padidar AM, Barnwell SL, Smith WS, Merci Registry Investigators. Endovascular mechanical thrombectomy for the treatment of acute ischemic stroke due to arterial dissection. Interv Neuroradiol. 2012 Mar;18(1):74-9
9. Chowdhury MM, Sabbagh CN, Jackson D, Coughlin PA, Ghosh J. Antithrombotic treatment for acute extracranial carotid artery dissections: a meta-analysis. Eur J Vasc Endovasc Surg. 2015;50(2):148.
10. Nedeltchev K, Bickel S, Arnold M, Sarikaya H, Georgiadis D, Sturzenegger M, Mattle HP, Baumgartner RW. R2-recanalization of spontaneous carotid artery dissection. Stroke. 2009;40(2):499
11. Arnold M, Bousser MG, Fahrni G, Fischer U, Georgiadis D, Gandjour J, Benninger D, Sturzenegger M, Mattle HP, Baumgartner RW. Vertebral artery dissection: presenting findings and predictors of outcome. Stroke. 2006;37(10):2499
12. Gabriela Dogaru, Alexandra Ispas, Adriana Bulboacă, Marieta Motricală, Ioana Stănescu. Influence of balnear therapy at Băile Tușnad on quality of life of post-stroke patients. Balneo Research Journal. 2017;8(4):201-205
13. Florina Frîngu, Gabriel Guşetu, Adriana Iosip, Diana Gurzău, Gabriela Dogaru, Dumitru Zdrenghea, Dana Pop. The predictors of exercise capacity impairment in diabetic patients. Balneo Research Journal. 2017;8(2):26-32