

Case Lessons

Spontaneous Bilateral Dissection of the Vertebral Artery

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Introduction: Vertebral artery dissection (VAD) is a rare cause of stroke in the general population, but one of the most common causes of stroke in patients younger than 45 years of age¹. It accounts for 2% of all ischemic strokes, but bilateral VAD are rarely reported on bibliography. The development of intramural hematoma with subintimal dissections causes luminal stenosis or occlusion. This may result in cerebral ischemia due to thromboembolism, hypoperfusion, or a combination of both². VA dissection may occur spontaneously or in vessels weakened by a primary arteriopathy; however, dissection more commonly occurs after trauma or even after less violent activities such as coughing, chiropractic manipulation, endovascular or surgical procedure³. Treatment might be conservator, endovascular or surgical. In addition to MT, stent-assisted VA angioplasty appears to be a safe, effective and immediate method of restoring vessel lumen integrity and should be considered in selected cases of bilateral VAD³.

We present a case of a bilateral spontaneous extracranial VA dissection presenting with multiple symptomatic embolic infarctions, underwent endovascular treatment with MT and emergency angioplasty with stent of V-B artery.

Key words: MT, VAD, endovascular treatment

Case presentation: 52-year-old male patient with a three-day history of acute onset headache in the occipital region with dizziness, balance problems and blurred vision. His condition has been getting worse progressively. He has dyslipidemia, arterial hypertension, overweight with a BMI of 29. Neurological examination on admission: Somnolent, Lightly miotic photoreactive pupils. Left abducens and trochlear nerve paralysis, right oculomotor nerve paresis, dysarthria, decreased gag reflex, deviation of the tongue to the left, static and dynamic ataxia. Right hemiataxia, and left hemiparesis. NIHSS 15 GCS 11-12 mRS 5

Brain CT showed hyperdensity sign on basilar artery (basilar artery thrombosis was suspected (Fig.1) MR diffusion-weighted images revealed embolic bilateral cerebellar and occipital ischemic lesions in the territory of the PCA, and bilateral vertebral artery dissection probably developed

in different time with lack of blood flow in the basilar artery was suspected on MRA (fig 2). Emergent endovascular treatment was recommended.

Right femoral puncture and placement of the 8F introducer. With the Chaperon 5Fr catheter, the diagnostic evaluation was performed through the ACC/AV bilat catheterization, which evidenced the occlusion of the both vertebral arteries at the V4 level and the absence of visualization of the basilar trunk. Bilateral posterior communicating arteries were present. Through the femoral introducer, the Neuronmax 6F catheter was passed in the cervical portion of left vertebral artery, which was dominant, in coaxial through the Penumbra Select 6F catheter. It was proceeded with the passage of the Sophia 6F aspiration catheter at the level of the occlusion in the left V4 in the coaxial system with Headway 21 + Microguide Taccess 0.14. First attempt with thromboaspiration failed, thrombectomy was performed with the ADAPT technique. In the control, revascularization of the basilar trunk was evident, but with reduced caliber in proximal segment and the presence of an evocative intimal flap suggesting intracranial dissection. After 15 minutes, the stenosis was reoccluded an intracranial stent was prepared meanwhile. An Agrastat bolus was applied according to the emergency protocol. A ballooned Neurospeed 3x8mm microcatheter was passed at the level of the stenosis, performing the angioplasty, and a 5x15mm Credo Stent (Accandis) was placed. DSA results restoration of TIC1 3 flow, and satisfactory opening of the stent. No acute complications during the procedure. (fig 3)

After two days, the patient's neurological status worsened, GCS 10 NIHSS 16. Brain MRI revealed moderate hydrocephalus (bifrontal diameter 35 mm) with edema of the left inferior cerebellar ischemic lesion causing compression of the fourth ventricle and right occipital hematoma probable hemorrhagic transformation of a small ischemic lesion, complication of agrastat. (fig 4) Immediate DVE and bilateral suboccipital decompressive craniectomy were performed, up to the foramen magnum (AHA/ASA 2019 guidelines, not revised since).

His clinical condition improved and left the hospital after three weeks (NIHSS 8, GCS15 mRS 2) for further rehabilitation. On the etiological exploration he had no past history of trauma. Further investigation such as: Complete vasculitis laboratory panel, connective tissue pathologies, viral tests (HIV, Hep C, Hep B), serology for Borrelia Burgdorfer, syphilis, cardiac evaluation for endocarditis, were all negative. No genetic studies were performed.

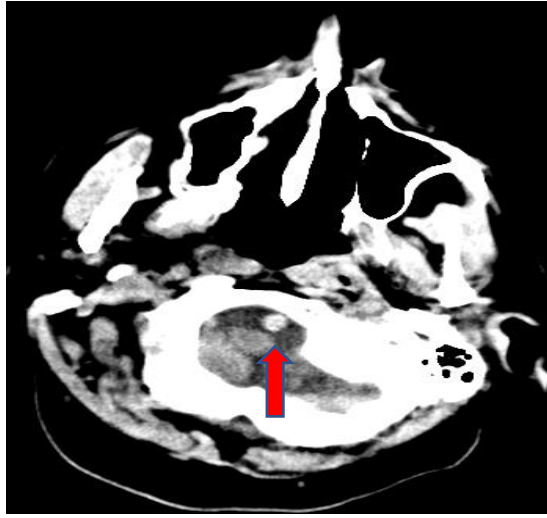


Fig.1: Hyperdensity sign on basilar artery (red arrow)

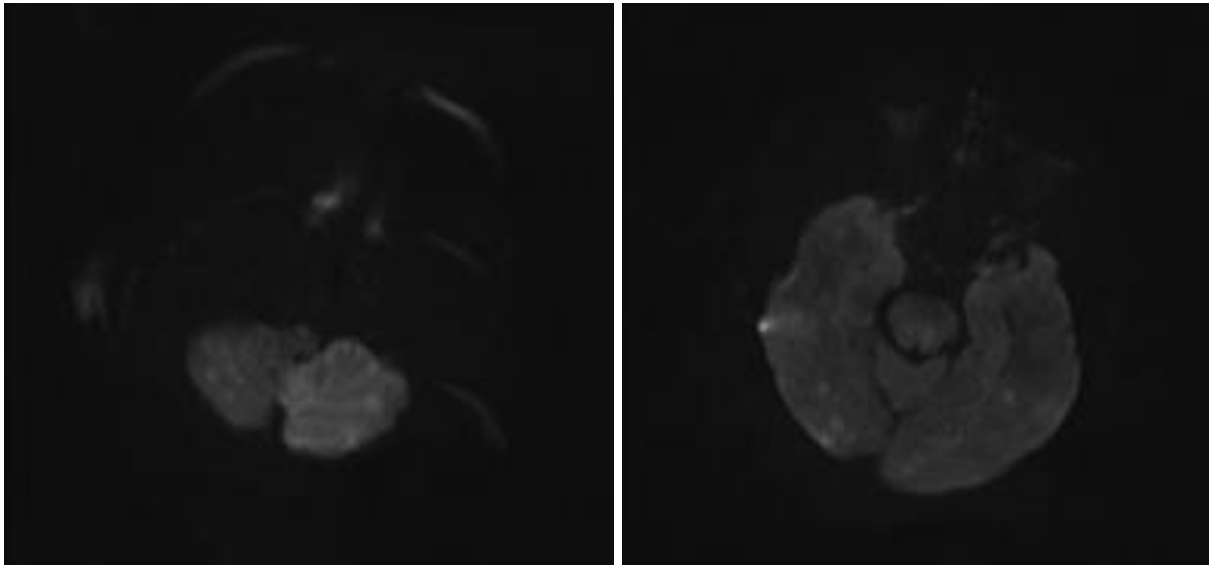
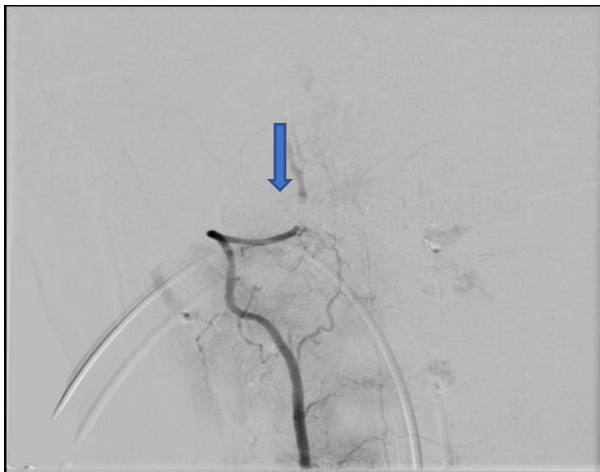


Fig 2. Brain MRI DWI: Ischemic lesion on V-B territory



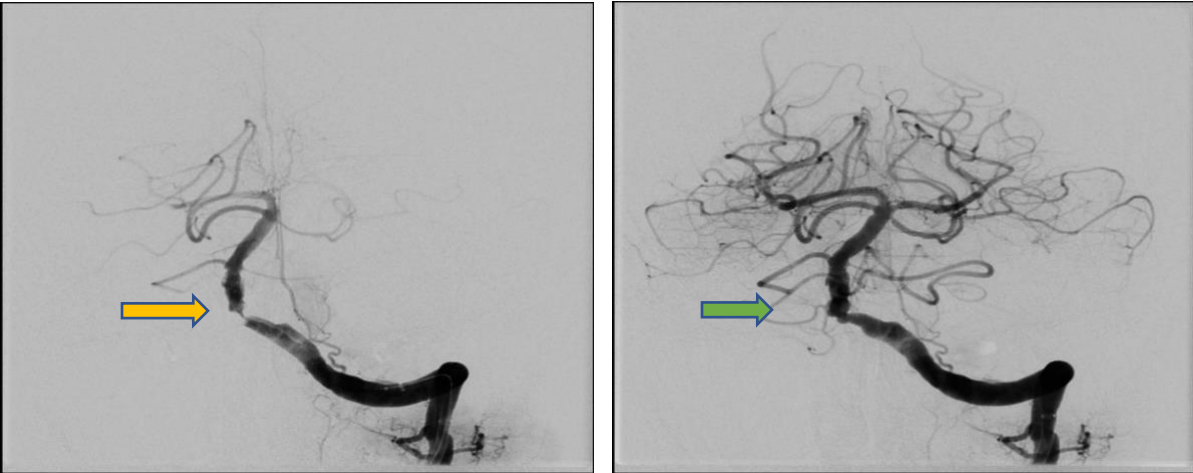


Fig 3 Endovascular procedure: Complete occlusion of bilateral V4 probably bilateral dissection in two times, left compensated (blue arrow) and right (red arrow) the cause of thrombus disease in a. Basilar. Evocative intimal flap with proximal stenosis in basilar artery (yellow arrow) Good recanalization of left V4 and basilar artery with angioplasty and stent (green arrow). TIC1 = 3.

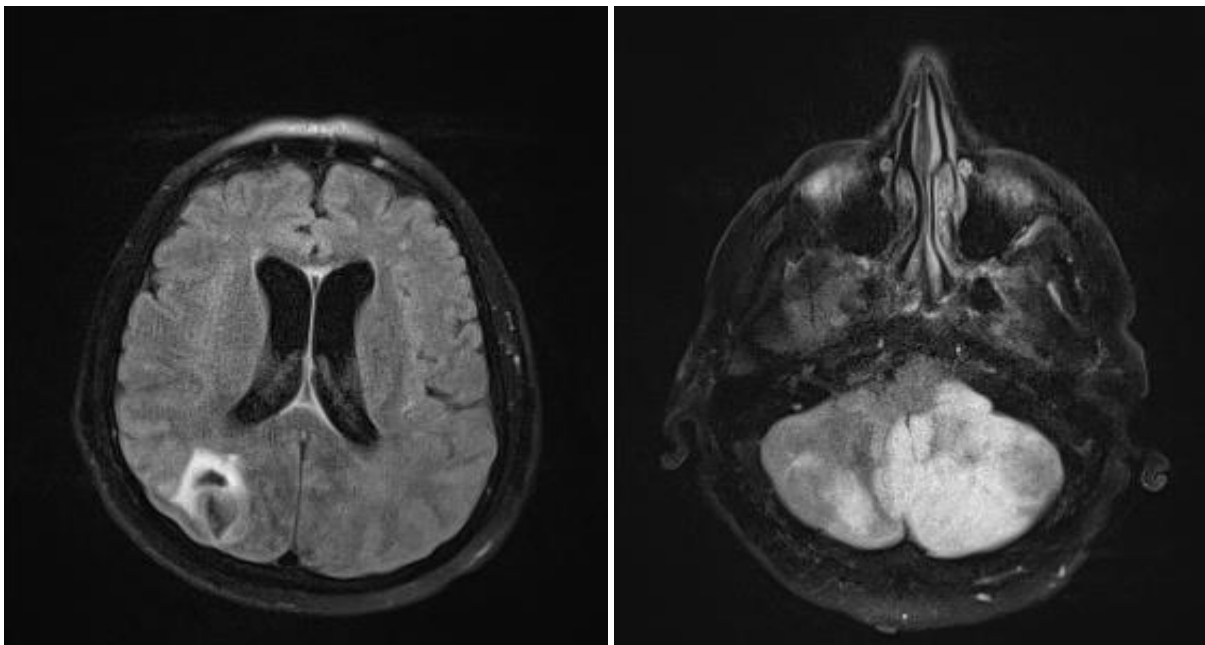


Fig 4: Moderate hydrocephalus (bifrontal diameter 35 mm) and fourth ventricular compression from inferior left cerebellar ischemic lesion and right occipital hematoma probable hemorrhagic transformation of a small ischemic lesion, complication of agrastat.

Discussion: It is estimated that vertebral artery dissection is the cause of approximately 2% of all ischemic strokes. However, in middle-aged and younger patients (30 to 45 years of age), it is believed to be as high as 10% to 25%¹. The most likely cause is cystic medial sclerosis, fibromuscular dysplasia, Ehler-Danlos syndrome, moyamoya, atherosclerosis, homocystinuria, osteogenesis imperfecta and cerebral aneurysms. The associated factors are migraine,

hypertension, infectious diseases, smoking and use of oral contraceptives^{4,6}. In our case, the risk factors are overweight and high blood pressure.

There are only a few cases reported with spontaneous bilateral VAD. As in our case, surgical or endovascular treatment has been reserved for patients who have persistent symptoms of ischemia despite adequate anticoagulation/antiaggregation or for patients in whom anticoagulation/antiaggregation is contraindicated. Endovascular treatment with stent-assisted VA reconstruction is the choice in patients with symptomatic bilateral VAD who hemodynamically depend on this artery³. In patients with severe deficits, reperfusion therapy is mandatory to immediately restore blood flow to areas of the brain that can be salvaged^{3,5}.

Conclusion: Bilateral VAD is an underestimated cause of stroke in young and middle age and treatments is challenging. Endovascular or surgical treatments, whenever time and vascular condition permit it, are life preserving and safe⁵.

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