JMS 2022; Vol5(3):98-103
UDC:616.133.33-085.273.53
DOI:10.55302/JMS2253098vs

MALE PATIENT WITH DISSECTION OF THE POSTERIOR CEREBRAL ARTERY AND OCULOMOTOR NERVE PALSY: A CASE REPORT AND A REVIEW FROM THE LITERATURE

Danijela Vojtikiv Samoilovska[1], Anita Arsovska[2], Rodna Kozoloska[3], Suzana Trajkoska[4]
[1]"Dr. Danijela" private neurology practise, Tetovo, North Macedonia,
[2]University Clinic of Neurology, Faculty of Medicine, St. Cyril and Methodius University in Skopje, North Macedonia,
[3]Public health polyclinic, Skopje, North Macedonia , [4]Public health polyclinic, Skopje, North Macedonia

Abstract

Spontaneous intracranial artery dissection is an uncommon and probably under diagnosed cause of stroke and subarachnoid hemorrhage that is defined by the occurrence of a hematoma in the wall of an intracranial artery.

We introduce a case of dissection of the P2 segment of the posterior cerebral artery (PCA) presented with new onset headache and unilateral, left oculomotor nerve palsy. The patient had a diagnosed thrombophilia and previous deep vein thrombosis. Digital subtraction angiography (DSA) revealed dissection of the P1-P2 segment of the PCA with occlusion of the distal branches P3-P4.

CT excluded acute stroke. The patient was put on anticoagulant therapy, with clinical improving after 2 weeks. The left third nerve had been distorted because of vascular compression.

Conclusion: Isolated dissection of the PCA is rare. Multicenter prospective studies with standardized protocols for diagnosis, imaging, and follow-up of intracranial artery dissection are needed.

Key words: dissection, PCA,

Introduction

Intracranial artery dissection is an uncommon and presumably under diagnosed cause of both ischemic stroke and subarachnoid hemorrhage.

The diagnosis of intracranial artery dissection is often difficult because of non-specific clinical presentation. [1,5,7] Isolated dissection of the posterior cerebral artery (PCA) is rare. We present a case of dissection of the P2 segment of the PCA.

Case presentation

A male patient, 59 years old, was brought to the emergency department with a new onset headache that had occurred in the previous two days.

It started like a very strong and stabbing pain on the left side of the neck and head, spreading towards the left supra-orbital area, nothing like he had ever experienced before.

The next day it was followed by complete ptosis of the left eye lid. Previously, the patient had suffered from three ischemic strokes, with residual left homonymous hemianopsia, minor left hemi paresis with pyramidal characteristics and had had a history of deep vein thrombosis (DVT).

The general examination at the time of presentation was unremarkable. Neurological status examination revealed unilateral, left oculomotor nerve palsy with slight anisocoria, bilaterally with normal reaction to light (picture 1 and picture 2).

Picture 1and 2 – Palsy of the left oculomotor nerve.

Findings of non contrast brain CT excluded acute stroke and showed post ischemic sequelaes in the right occipital parasagittal region. Screening for hypercoagulability disorders confirmed trombophilic mutations.

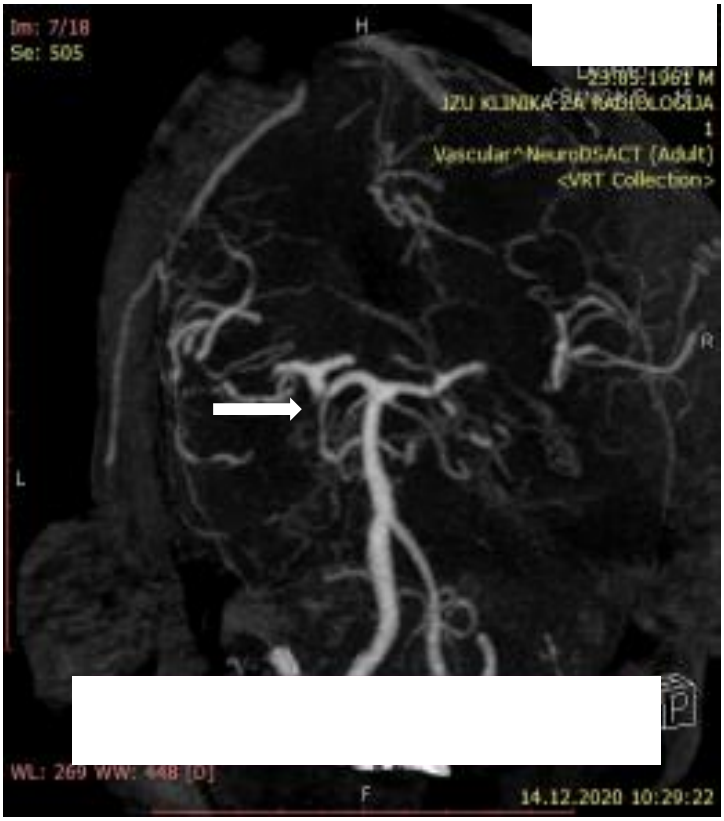
Heterozygous in the gene C677T for MTHFR and homozygous mutation for e NOS – 786 T>C.PAI-I gene 4G/5G. The level of homocysteine was in the referential values.

The patient was put on anticoagulant therapy with oral vitamin K antagonist (acenocumarol 4mg/day), bridged with low molecular heparin for a few days until target international normalized ratio between 2.0 and 3.0.was established.

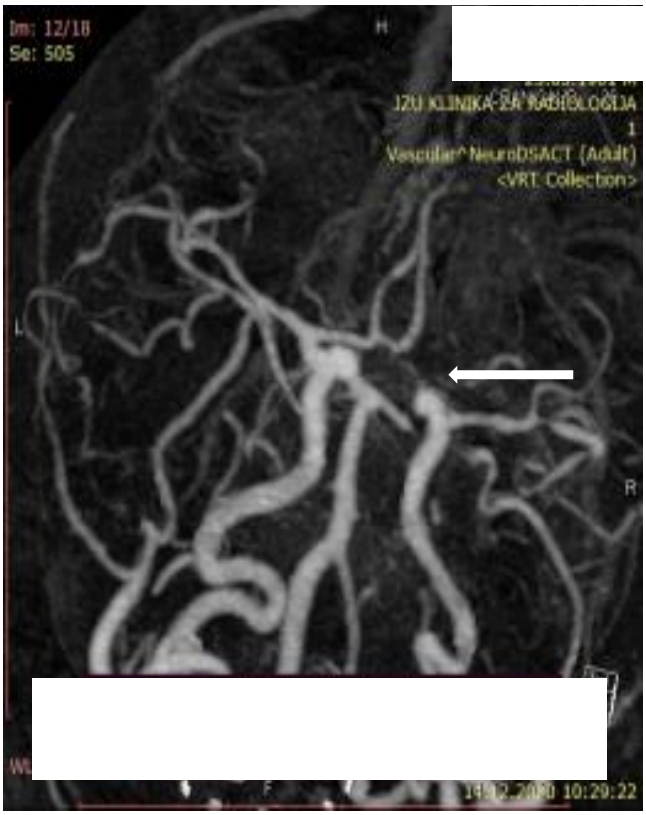
Digital subtraction angiography (DSA) revealed sub occlusion of the left posterior cerebral artery (PCA) about 7 mm distal to the outlet and along the entire length segments of significant stenosis and post stenotic dilatations.

Occlusion in the distal branches (P3-P4) in the occipital region was seen. The findings were mostly consistent with dissection of the left PCA due to the described presentation of segmental dilatations and narrowing with distal complete occlusion.

Stenosis of the right middle cerebral artery (MCA) inferior trunk in the length of 2 mm was seen as well, with a moderate degree of stenosis (50%-60%). The right anterior cerebral artery (ACA) A1 segment and right vertebral artery were hypoplastic.



Picture 3.
DSA scan of the cerebral arteries
-sub occlusion of the left posterior cerebral artery (PCA), occlusion in the distal branches (P3-P4) in the occipital region



Picture 4. DSA scan of the cerebral arteries - stenosis of the right middle cerebral artery (MCA), right anterior cerebral artery (ACA) A1 segment and right vertebral artery were hypoplastic.

At the time of discharge after 2 weeks, a significant improvement in neurological status was noted, with left eyelid semiptosis and improved movement of the left eye with possible partial motility medially.

Diplopia steel present. (Picture 5). The pupils were of equal diameter.

Picture 5. Improvement of the palsy of the left oculomotor nerve after two weeks.

The decision was made to continue anticoagulation and perform further follow-up imaging 3 months later. MRA was performed. It revealed chronic ischemic changes in the right occipital para falx cerebry area, with surrounding gliosis and traction on the right trigonum and posterior horn of lateral chamber.

The TOF pulse sequence and postcontrast series showed that the lumen of left PCA distally from the level of the P1 segment was not monitored.

The patient was advised to keep up regular controls. TCCD Bubble test and ultrasound of the peripheral circulation were advised. Methylfolat 4 mg, methyl b12 and pyridoxin were recommended as additive treatment.

Discussion

Although most arterial dissections or dissecting aneurysms are mainly without detectable cause, a possible causative relation has been suggested with conditions such as: syphilis, migraine headaches, cystic medial necrosis, Marfan syndrome, mixed connective tissue disease, fibromuscular dysplasia, homocystinuria, polycystic kidney disease and trauma. Undetectable cause persists with our patient. Vertebrobasilar dissections are more commonly seen in males, whereas isolated PCA dissections are predominant in females.

In general, intracranial arterial dissections occur in young adults, typically in the late third to fifth decade [3,10,13]

The most common symptom of vertebrobasilar and PCA dissection is headache, predominantly in the occipital and posterior cervical regions.[2,3,4] Dissecting intracranial aneurysms usually present with large cerebral infarcts but may also present with subarachnoid hemorrhage or both.[12],

They can be associated with severe neurologic deficits, often leading to death. Isolated PCA dissections tend to present with ischemic symptoms and have a more benign clinical course and prognosis.[5,6]

Intracranial dissections typically occur between the intima or internal elastic lamina and the media. Absence or dysplasia of the internal elastic lamina, especially if the media is incomplete, is believed to predispose to intracranial dissection [1].

Isolated PCA dissections most commonly occur near the P1-P2 junction, which is close to the free border of the tentorium cerebella [7].

The diagnosis of intracranial dissection and dissecting aneurysms is predominantly made by DSA, which remains the gold standard technique [11].

Treating physicians must weigh the benefit-risk ratio of anticoagulants in each individual patient with intracranial dissection. Thrombophilic patients are at bigger risk than healthy subjects. The increase of risk is much more pronounced in patients with a history of DVT.

Anticoagulation (heparin followed by warfarin, INR 2-3) continued with antiplatelet has been promoted, and can be recommended, especially for patients with intracranial dissection presenting with stable ischemic symptoms [8].

In hypertensive patients, it is important to maintain adequate blood pressure control to prevent progression of the dissection and possible rebleeding or worsened ischemia. Patients with ruptured dissecting aneurysms and associated subarachnoid hemorrhage or progressive neurologic deficits typically require treatment by surgical or endovascular means [14].

Management, outcome and prognosis

Optimum treatment for patients with intracranial artery dissections is unknown.

No randomized trials exist and only observational studies with small sample sizes are available, thus providing a very low level of evidence [3,6, 9]

In conclusion, multicenter prospective studies and, ultimately, trials with standardized protocols for diagnosis, imaging, and follow-up of intracranial artery dissection are needed.

References

1. Koichi Haraguchi .A Case of Posterior Cerebral Artery Dissection Presenting with Migraine-Like Headache and Visual Field Defect: Usefulness of Fast Imaging Employing Steady-State Acquisition (FIESTA) for Diagnosis. J Stroke Cerebrovasc Dis. 2012 Nov;21(8):906.e5-7 https://pubmed.ncbi.nlm.nih.gov/22177933/.
2. Berthier E, Bourrat C (1993) Dissecting aneurysm of the posterior cerebral artery: case report and review of the literature. Cerebrovasc Dis 3: 56±59 https://doi.org/10.1159/000108672.
3. Maillo A, Diaz P, Morales F. Dissecting aneurysm of the posterior cerebral artery: spontaneous resolution.\ Neurosurgery1991;29:291–94 DOI: 10.1097/00006123-199108000-00024.
4. Berger MS, Wilson CB.Intracranial dissecting aneurysms of the posterior cerebral circulation: report of six cases and review of the literature.J Neurosurg1984;61:882–94 DOI: 10.3171/jns.1984.61.5.0882.
5. P Sherman . Isolated posterior cerebral artery dissection Report of three cases. AJNR Am J Neuroradiol. 2006 Mar; 27(3): 648–652. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7976983/.
6. Kawahara I.Isolated posterior cerebral artery dissection: case report and review of the literature. No Shinkei Geka2003;31:671–75 https://pubmed.ncbi.nlm.nih.gov/12833877/.
7. Hirai T, Korogi Y, Murata Y, et al.Intracranial artery dissections: serial evaluation with MR imaging, MR angiography, and source images of MR angiography.Radiat Med2003;21:86–93 https://pubmed.ncbi.nlm.nih.gov/12816356/.
8. Metso TM, Metso AJ, Prognosis and safety of anticoagulation in intracranial artery dissections in adults. Stroke. 2007 Jun;38(6):1837-42. doi: 10.1161/STROKEAHA.106.479501. https://pubmed.ncbi.nlm.nih.gov/29565222/ .
9. Lishan Cui,. Parent artery occlusion for intracranial aneurysms. Interv Neuroradiol 2009 Sep;15(3):309-15. doi: 10.1177/159101990901500308. Epub 2009 Nov 4. https://pubmed.ncbi.nlm.nih.gov/20465914/.
10. Nikolaos Staikoglou. Posterior cerebral artery dissection after excessive caffeine consumption in a teenager. Radiol Case Rep. 2022 Apr 12;17(6):2081-2084. doi: 10.1016/j.radcr.2022.02.035 https://pubmed.ncbi.nlm.nih.gov/35464799/.
11. Muhammad A. Taqi. Dissecting Aneurysms of Posterior Cerebral Artery: Clinical Presentation, Angiographic Findings, Treatment, and Outcome. Front Neurol. 2011; 2: 38. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3124944/.

12. Min-Gyu Park . Spontaneous isolated posterior inferior cerebellar artery dissection: rare but underdiagnosed cause of ischemic stroke. J Stroke Cerebrovasc Dis. 2014 Aug;23(7):1865-70. doi: 10.1016/j.jstrokecerebrovasdis.2014.02.023. https://pubmed.ncbi.nlm.nih.gov/24809669/.
13. Tomoo Inoue. Postpartum dissecting aneurysm of the posterior cerebral artery. J Clin Neurosci, 2007 Jun;14(6):576-81. doi: 10.1016/j.jocn.2006.04.005 https://pubmed.ncbi.nlm.nih.gov/17430781/.
14. S Kawaguchi. Management of dissecting aneurysms of the posterior circulation. Acta Neurochir (Wien).1994;131(1-2):26-31.doi:10.1007/BF01401451. https://pubmed.ncbi.nlm.nih.gov/7709782/.