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Successful Thrombectomy of the Posterior Cerebral Artery P2 Segment in a 61-Year-Old Man with Acute Ischaemic Stroke: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Male, 61-year-old
Final Diagnosis: Acute ischemic stroke
Symptoms: Amnesia • aphasia • right-sided hemianopia • dizziness and acute memory deficits
Clinical Procedure: —
Specialty: Neurology

Objective: Unusual setting of medical care

Background: Acute ischemic stroke in the posterior cerebral artery (PCA) territory can lead to persistent disabling deficits. The PCA is divided into 4 segments. The P2 segment begins at the posterior communicating artery and curves around the midbrain and above the tentorium cerebelli. This report is of a 61-year-old man with acute ischemic stroke involving the left hippocampus treated with direct thrombectomy of the P2 segment of the PCA.

Case Report: A 61-year-old white man presented with transient amnesia, aphasia, right-sided hemianopia, dizziness, and persistent acute memory deficits. Magnetic resonance imaging (MRI) showed a left hippocampal acute ischemic stroke with left PCA occlusion in the P2 segment. Despite a low National Institutes of Health Stroke Scale (NIHSS) score and the already-formed lesion in the hippocampus, successful stent retriever thrombectomy was performed due to a considerable perfusion–diffusion mismatch and a persistent potentially disabling neurocognitive deficit. Due to partial thrombus dislocation, occlusion of the common origin of the right posterior inferior cerebellar artery (PICA) and anterior inferior cerebellar artery (AICA) occurred and was immediately treated by thrombectomy to prevent severe cerebellar infarction. His clinical symptoms completely resolved and a neuropsychological exam showed no residual deficits.

Conclusions: Thrombectomy of the P2 segment of the PCA is feasible and can be considered to treat patients with acute occlusion at risk for persistent disabling deficits, based on clinical estimation of the impact of such deficits and the presence of potentially salvageable brain tissue. Potential procedural complications should be sought out and immediately treated, if technically feasible.

Keywords: Brain Infarction • Case Reports • Infarction, Posterior Cerebral Artery • Thrombectomy

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/941441

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Background

Posterior cerebral artery (PCA) occlusions can restrict the blood supply of multiple brain regions, including the occipital lobe, the inferomedial temporal lobe, a large portion of the thalamus, and the upper brainstem [1]. The PCA is divided into 4 segments: 2 proximal or deep (P1 and P2) and 2 distal or superficial (P3 and P4). The P1 segment lies between the end of the basilar artery and the posterior communicating artery and supplies the paramedian parts of the upper midbrain and thalamus through the thalamo-perforating arteries. The branches of the P2 segment include the thalamo-geniculate arteries, which supply the ventrolateral part of the thalamus, and the posterior choroidal arteries, which supply the lateral geniculate body, pulvinar, posterior thalamus, hippocampus, and parahippocampal gyrus. The P3 segment is the quadrigeminal segment and leads to the anterior and posterior inferior temporal arteries. The P4 segment is the cortical segment within the calcarine fissure and becomes the calcarine artery. Other branches of the distal part of the PCA are the occipitotemporal and occipitoparietal arteries [1].

Acute ischemic stroke in the PCA territory accounts for 5-10% of all ischemic strokes and can be caused by different mechanisms, including large-vessel atherosclerosis, cardioembolism, and small-vessel disease [1]. It can lead to disabling deficits, including visual field deficits and cognitive and behavioral dysfunction [1,2]. However, evidence about the efficacy and safety of acute reperfusion therapies in patients with acute occlusion of the PCA is limited. Intravenous thrombolysis appears to have similar efficacy for both the posterior and anterior circulation, with lower hemorrhage risk in posterior circulation stroke [3]. Regarding endovascular treatment, retrospective analyses have shown the feasibility and safety of mechanical thrombectomy [4-6]. The treatment decision is even more challenging in patients with PCA occlusion and low National Institutes of Health Stroke Scale (NIHSS) score.

We report a case of a 61-year-old man with acute cognitive impairment, aphasia, and right-sided hemianopia due to acute ischemic stroke involving the left hippocampus, with perfusion–diffusion mismatch on magnetic resonance imaging (MRI), who was treated by direct thrombectomy of the P2 segment of the posterior cerebral artery (PCA).

Case Report

A 61-year-old white man with a past medical history of melanoma excision, no known vascular risk factors or ongoing treatment, and unremarkable family history, presented with amnesia, aphasia, right-sided hemianopia, dizziness. The exact time of symptom onset was unknown as he awaked with stroke symptoms that were not present prior to falling asleep (i.e. wake-up stroke). In addition, he did not remember the names of his children and had difficulties finding words. His symptoms spontaneously improved and at hospital admission his neurological exam was unremarkable (NIHSS 0) except for a 5-minute delayed recall task, in which he was unable to remember 3 out of 5 words. Brain MR imaging and angiography (MRI and MRA) with dynamic susceptibility contrast (DSC) perfusion imaging showed left hippocampal diffusion restriction (**Figure 1A**), hyperintense in fluid-attenuated inversion recovery (FLAIR), with occlusion of the left posterior cerebral artery

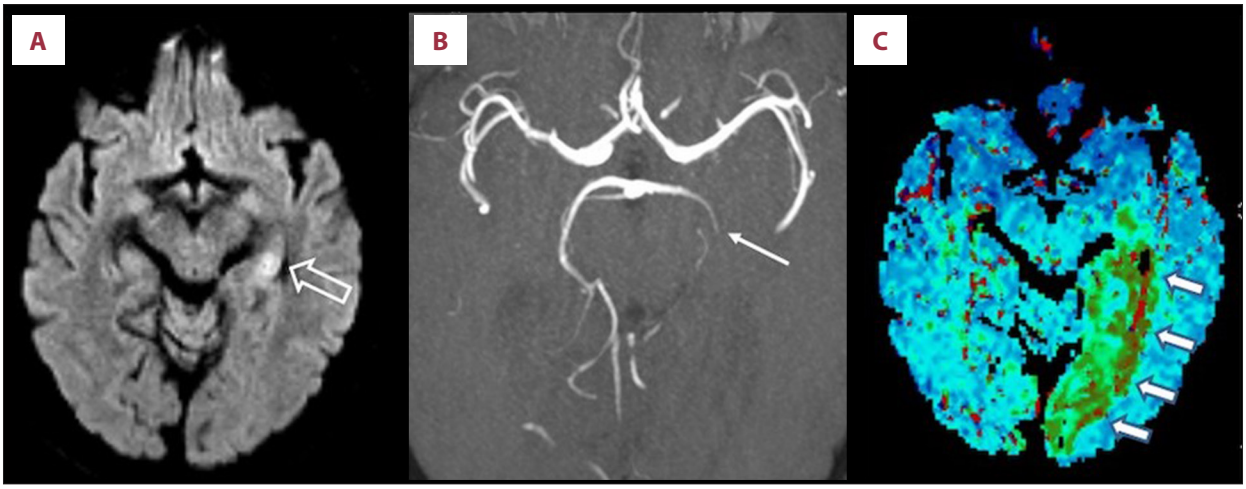

Figure 1. Brain Magnetic Resonance Imaging of a Patient with Left Hippocampal Acute Infarction due to an Occlusion of the Left Posterior Cerebral Artery (PCA) in the P2 segment. Axial diffusion weighted imaging with b=1000 value (**A**) showing left hippocampal diffusion restriction (thick arrow) consistent with acute ischemic stroke in the PCA territory. Transversal maximum intensity projection time-of-flight image showing occlusion (thin arrow) of the left PCA in the P2 segment (**B**) with corresponding extensive perfusion deficit (multiple arrows) in the time to peak map (**C**).

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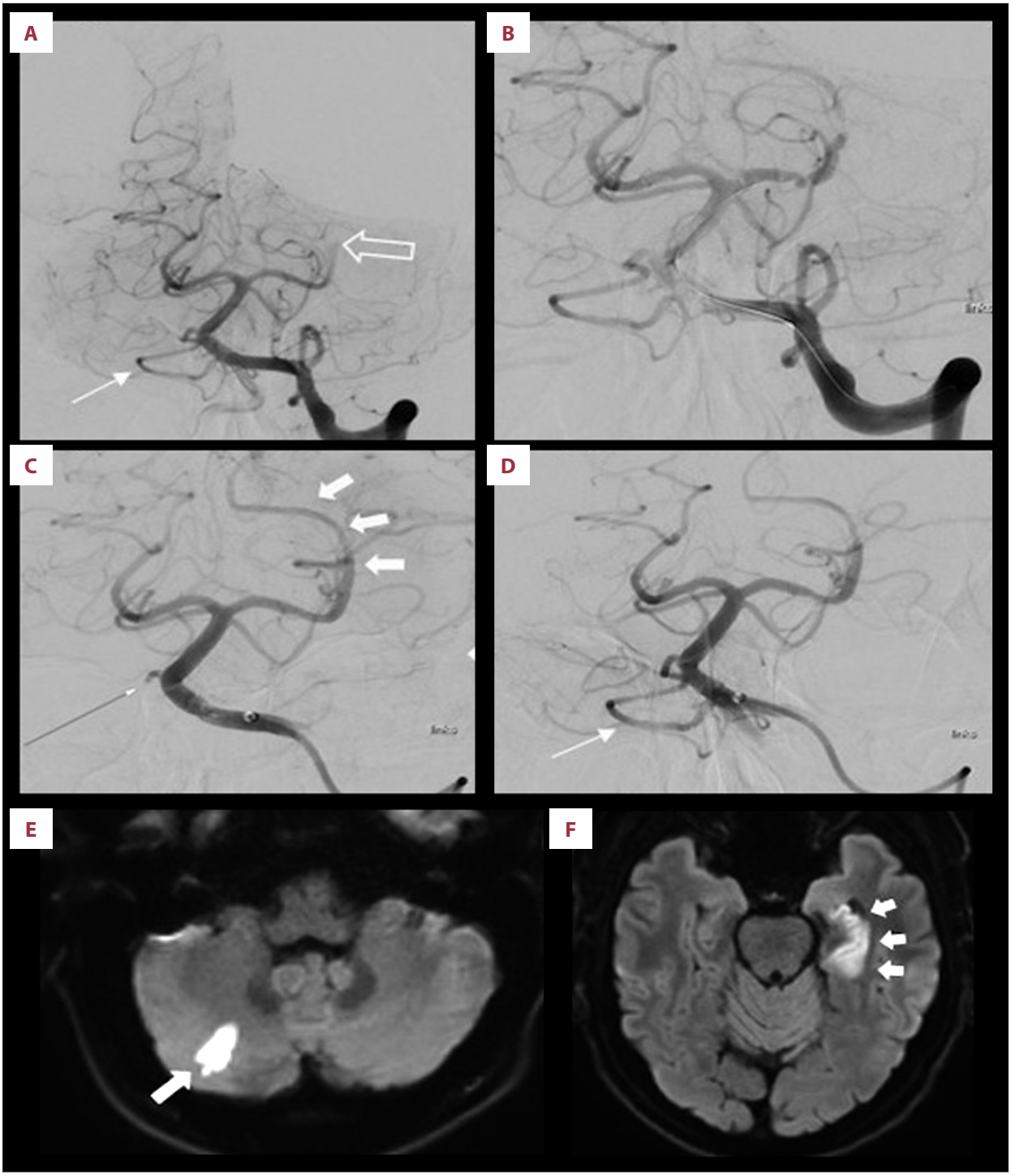

Figure 2. Digital Subtraction Angiography (DSA) and 24-hour control MRI in the Same Patient. DSA showing occlusion of the left posterior cerebral artery (PCA) in the P2 segment (empty arrow) with patent right posterior inferior cerebellar artery (PICA) (thin arrow) (**A**), with positioned stent retriever from the left P1 to the P2 segment (**B**). DSA showing complete recanalization of the PCA (thick arrows) and occlusion of the common origin of the right PICA and anterior inferior cerebellar artery (AICA) (thin arrow) due to partial thrombus dislocation (**C**). Post-procedural DSA showing complete reperfusion of the PICA and AICA (arrow) (**D**). 24-hour diffusion weighted imaging (DWI) showing a new subacute small cerebellar ischemia (arrow) in the right PICA territory (**E**) and 24-hour fluid-attenuated inversion recover (FLAIR) images showing the known left hippocampal infarction (arrows) (**F**).

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(PCA) in the P2 segment (**Figure 1B**), and perfusion–diffusion mismatch (**Figure 1C**). After interdisciplinary discussion, we decided not to thrombolyse intravenously due to the unknown time of symptom onset and FLAIR positivity, with a potential risk of hemorrhagic transformation. However, despite having mild symptoms with low NIHSS score and the already-formed ischemic lesion in the hippocampus consistent with the memory deficit, we decided to perform endovascular treatment due to a considerable perfusion–diffusion mismatch with an occlusion of the P2 segment, and the suspicion that the persistent and potentially disabling neurocognitive deficit might be in part related to peri-hippocampal hypoperfusion, on the top of already infarcted head of the hippocampus. We therefore immediately performed a successful stent retriever thrombectomy with local anesthesia, with a dedicated device for small vessels (Catch Mini, Balt, Montmorency, France) in combination with a distal access catheter (Catalyst 5F, Stryker, Cork, Ireland) (**Figure 2A, 2B**). The P2 segment completely recanalized (**Figure 2C**). Due to partial thrombus dislocation, occlusion of the common origin of the right posterior inferior cerebellar artery (PICA) and anterior inferior cerebellar artery (AICA) occurred as embolus in a new territory (ENT) despite utilization of a distal access catheter (**Figure 2C**). To prevent severe cerebellar infarction with associated complications, successful recanalization with the same stent retriever 5-10 minutes after occlusion was performed [7]. Post-procedural angiography showed complete reperfusion with a Thrombolysis In Cerebral Infarction (TICI) score of 3 (**Figure 2D**). The patient was strictly monitored to immediately detect any clinical, hemodynamic, or metabolic changes. Due to his stable clinical condition and hemodynamic/metabolic parameters, no periprocedural medication was administered. The 24-hour control MRI and MRA showed a new subacute small ischemia in the right PICA territory (**Figure 2E**) without any significant extension of the known left PCA territory infarction (**Figure 2F**), hemorrhagic transformation, or arterial reocclusion. No new infarctions were detected on further control MRIs. While hospitalized, the patient showed isolated attention and memory deficits. At discharge on day 8, the patient could directly remember 6 out of 8 words on the word list recall test, and only 3 out of 8 after 10 minutes. At the formal neuropsychological evaluation on day 24, his cognitive performance profile was unremarkable compared to the age- and education-adjusted normative data. The patient continued to have increased fatigue, which completely resolved until clinical follow-up at 3 months, for which the modified Rankin score was 0. In the extensive etiological workup, including 3 ambulatory 7-day ECG monitorings, an atrial septal defect was detected, which was closed at 7 months by successful transcatheter closure. The patient had no clinical or biological parameters suggestive of a deep venous thrombosis or a prothrombotic status, including cancer. Vascular risk factors, including mild arterial hypertension, hypertriglyceridemia, and overweight, were also detected and treated.

Discussion

We present a case of acute ischemic stroke in the PCA territory treated by mechanical thrombectomy of the P2 segment. Our patient presented with severe symptoms, which spontaneously improved. However, due to persistent potentially disabling cognitive deficits and the presence of potentially salvageable brain tissue at on perfusion–diffusion MRI, the decision was made to perform a mechanical thrombectomy.

Evidence about the benefit of acute reperfusion therapies in patients with occlusion of the PCA and low NIHSS score is limited since this patient group has likely been underrepresented or in some cases excluded from randomized clinical trials on intravenous thrombolysis, and the pivotal trials on thrombectomy have included no patients with acute occlusion of the PCA and only some patients with low NIHSS [8]. However, strokes in the PCA territory can lead to deficits, which while not causing high NIHSS scores can impair functional independence and quality of life, entailing neuropsychological deficits and visual symptoms [1,2]. Potential higher efficacy of endovascular treatment over medical treatment, with trends for better prognosis in terms of cognition, visual field, and global disability outcomes, was found in a retrospective analysis of 106 patients with acute ischemic stroke and isolated occlusion of the PCA [4]. Similarly, a retrospective multicenter analysis demonstrated the feasibility and safety of thrombectomy for isolated PCA occlusion [5]. In a recently published multicenter case-control study conducted on 184 patients treated for primary distal occlusion of the PCA, significant treatment effects of mechanical thrombectomy were observed in the subgroup of patients with NIHSS scores on admission of 10 points or higher and in the subgroup of patients without intravenous thrombolysis [9]. No differences in symptomatic intracranial hemorrhage occurrence were found [9]. Our patient's procedural and clinical evolution was comparable to that of patients reported in previous publications [4-6,9]. However, there is a need for randomized clinical trials including such patients with acute PCA occlusion, potentially disabling deficits independently on NIHSS, and presence of potentially salvageable brain tissue. In addition, while waiting for more scientific evidence, an individualized decision should be taken based on clinical estimation of the functional impact of such deficits and the presence of hypoperfused, still viable brain tissue. For instance, we believe that our patient's favorable clinical evolution, including complete regression of mnestic deficits, was at least partially related to the recanalization of the P2 segment of the PCA, leading to timely reperfusion of the peri-hippocampal region.

As in our case, if the decision is taken to perform an endovascular treatment in a distal vessel, a dedicated device for small vessels needs to be used to minimize the risk of hemorrhagic complications [10].

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Furthermore, in our patient, an ENT was immediately detected and treated. In fact, although the exact prevalence of posterior circulation stroke is unclear, ENTs generally occur in about one-third of patients treated by thrombectomy and they are usually angiographically occult [11]. They are more frequent in patients treated by direct endovascular treatment (ie, without pretreatment with intravenous tissue plasminogen activator), without protection during stent-retrieval (Thrombectomy with Balloon Guiding Catheter or Distal Aspiration Catheter), and in cardio-embolic stroke [11]. ENTs are associated with poor outcome with increasing size [11].

The main limitation of this case report is that it is based on a single specific experience, which limits the generalizability of our findings.

Conclusions

Thrombectomy of the P2 segment of the PCA seems to be feasible and might be considered to treat patients with acute occlusion at risk for persistent disabling deficits. While waiting for strong scientific evidence from randomized clinical trials, an individualized decision should be considered for such patients based on clinical estimation of the impact of such deficits and the presence of potentially salvageable brain tissue. Potential procedural complications should be detected and treated immediately if technically feasible.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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