DOI:10.4274/Tnd.56823

Posterior Cerebral Artery Infarction with Cognitive Findings: More Than Hemianopia

Bilişsel Bulgularıyla Posterior Serebral Arter İnfarktı: Hemianopsiden Fazlası

Zehra Bozdoğan, Aslıhan Taşkıran Sağ, Zeynep Neşe Öztekin, Fikri Ak
Ankara Numune Training and Research Hospital, Neurology Clinic, Ankara, Turkey

Summary

Posterior cerebral artery infarctions constitute 5-10% of all ischemic strokes. The case of a 62 year-old man presenting with right hemiparesia and dysarthria is discussed in this article. The patient had acute onset demential symptoms and difficulty in reading, and an acute infarction in the territory of left posterior cerebral artery was detected in cranial magnetic resonance imaging. The patient who had acute ischemic lesions in left parahippocampus, splenium of corpus callosum, left fusiform gyrus and left thalamus was evaluated with detailed neuropsychological tests. Cognitive deficits severe enough to affect daily living were detected. Pure alexia, anterograde amnesia and anomia, being often neglected manifestations of unilateral posterior cerebral artery infarction, are going to be reviewed with relevant radiological findings. *(Turkish Journal of Neurology 2013; 19:139-142)*

Özet

Arteria cerebri posterior infarktları tüm iskemik inmelerin %5-10'unu oluşturur. Bu yazıda sağ hemiparezi ve konuşma bozukluğu yakınmasıyla başvuran 62 yaşında bir erkek olgu sunulacaktır. Hastada bu olayla birlikte başlayan demans bulguları ve okuma bozukluğu gözlenmiş olup beyin manyetik rezonans görüntülemesinde sol posterior serebral arter sulama alanına denk gelen akut infarkt tespit edilmiştir. Sol parahipokampüste, korpus kallozumun splenium kesiminde, sol fusiform girusta ve sol talamusta akut iskemik lezyonları olan hastaya erken dönemde detaylı nöropsikolojik testler uygulanmış ve günlük yaşantısını etkileyecek boyutta bilişsel bozukluklarının ortaya çıktığı tespit edilmiştir. Tek taraflı posterior serebral arter infarktının günlük uygulamada çoğunlukla ihmal ettiğimiz bulguları olan saf aleksi, anterograd amnezi ve anomi radyolojik bulgular eşliğinde tartışılacaktır. *(Türk Nöroloji Dergisi 2013; 19:139-142)*

Anahtar Kelimeler: Aleksi, anomi, amnezi, posterior serebral arter

Introduction

Posterior cerebral artery (PCA) infarctions constitute 5-10% of all ischemic strokes (1). It commonly presents with hemianopia and somatosensory findings. Clinical manifestations may vary depending on the involvement of superficial or deep segments. Certain critical brain structures that PCA supplies to, such as splenium, thalamus, hippocampus and parahippocampal gyrus, may create distinct cognitive disorders during PCA infarctions.

In this report, a case of hemiparesis in a 62-year-old male is presented and his cognitive deficits started at the early post-stroke stage will be discussed in the light of his neuropsychological evaluation results and their anatomical correlates.

Case

A 62-year-old male patient came to the emergency service after a transitory weakness on the upper and lower extremities that lasted for 30 minutes and difficulty in speaking. In the neurological examination, his blood pressure was 195/90 mmHg, he was fully conscious, fully cooperative and oriented with isochoric pupils. Direct and indirect light responses were intact bilaterally. There was homonymous hemianopia on the right side. Eye movements were unrestricted in every direction. There was no visible facial asymmetry but the labial dysarthria during speaking was noticeable. His lower right extremity was at 5-/5 according to Medical Research Council (MRC) scale during the motor examination. There was right-sided hemihypesthesia that also included the face.

Address for Correspondence/Yazışma Adresi: Aslıhan Taşkıran Sağ MD, Ankara Numune Training and Research Hospital, Neurology Clinic, Ankara, Turkey
Phone: +90 312 508 4510 E-mail: aslihantaskiran@gmail.com
Received/Geliş Tarihi: 25.10.2012 **Accepted/Kabul Tarihi:** 31.03.2013

He was bilaterally proficient in the cerebellar tests and did not show any pathological reflex. There were no pathological findings in his hemogram and blood biochemical evaluation. He did not show signs for acute infarction in his computerized tomography (CT). His history did not include chronic cerebrovascular risk factors such as hypertension, atherosclerotic heart disease, diabetes mellitus or obesity. He smoked a pack of cigarettes every day for the past 30 years. The patient who was employed as a taxi driver did not have signs of dementia prior to the event. After the event, however, he reported certain cognitive deficits that would interfere with his daily activities such as forgetting names of places or people, impaired navigation skills and difficulty reading.

The patient was started on a secondary prevention with 300 mg acetylsalicylic acid. To stabilize the increased blood pressure following this treatment, dual antihypertensive treatment was used for maintenance. Lipid profile showed LDL:11 mg/dl and thyroid function tests, B12 vitamin and folate levels were within normal ranges. Echocardiographic surveys did not show intracardiac thrombus, valvular dysfunction or stenosis. There was no atrial fibrillation in the Holter recording.

In the diffusion-weighted volumes and apparent diffusion coefficient (ADC) maps of magnetic resonance imaging (MRG), signal changes forming patchy, distributed acute ischemic regions resembling restricted diffusion patterns were observed on left parahippocampal, fusiform, occipital and cingulate gyri, left part of corpus callosum splenium and left thalamus (Figure 1). On the right side, there were 2 hyperintense areas on the millimetric scale but there were no corresponding signs on the ADC.

Neck and brain CT angiography were taken to inspect the state of the vascular bed. Bilateral arteria vertebralis, arteria basilaris, bilateral arteria cerebelli inferior posterior, arteria cerebelli superior and right PCA were normal. The distal part of the left PCA P1 segment was found to be in narrow calibration. P2 segment was normal. In the bilateral carotis communis bifurcation, calcified and non-calcified plaques towards proximal part of carotis interna were found. At the bilateral carotis interna supraklinoid levels, the plaques were still seen but there was no narrowness. Bilateral arteria cerebri media and right arteria cerebri arterior (ACA) A1 segment were normal. Left ACA A1 segment had more narrow calibration compared to the right. Coincidentally, saccular aneurysm of 10.2x6.2x8.3 mm was found on the arteria communicans anterior right ACA A2 segment.

The patient completed a battery of neuropsychological tests including standardized mini mental test (SMMSE), clock drawing test, enhanced cue recall memory test (ECRM), trail making test, semantic and phonemic fluency tests.

The patient scored 16 in SMMSE but failed in working memory, attention and arithmetic, naming, reading and copying figures. His repetition and comprehension was normal. He could write sentences.

He showed visual neglect in clock drawing test and failed the task (Figure 2).

Enhanced cue recall memory test showed working memory impairment (Table 1).

In the naming task, he named 6 out of 10 items with delay. His color naming was severely impaired.

Discussion

The patient who consulted in our clinic with symptoms of motor and sensory loss underwent a comprehensive neurological examination and showed anterograde amnesia, pure alexia, anomic aphasia, acalculia, executive function deficit, and degradation in his topographic, visuospatial skills. These cognitive symptoms are discussed in the light of the anatomical findings.

Splenium of corpus callosum is composed of a commissural bundle of fibers connecting the two hemispheres from the posterior. According to the classical view, splenium lesions cause alexia without agraphia, or "pure alexia", by disconnecting the visual areas on the right hemisphere and language centers in the left hemisphere, specifically angular gyrus (3). Angular gyrus is an association center mediating different modalities during reading (4). Alternatively, some researchers argued that pure alexia is due to impairment in the coding of alphanumerical symbols and suggested the deafferentation of visual word form area in

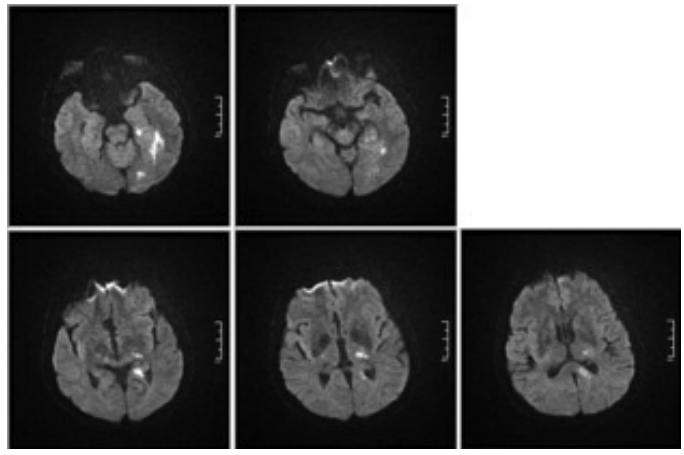

Figure 1. Magnetic resonance imaging evaluation of the patient.

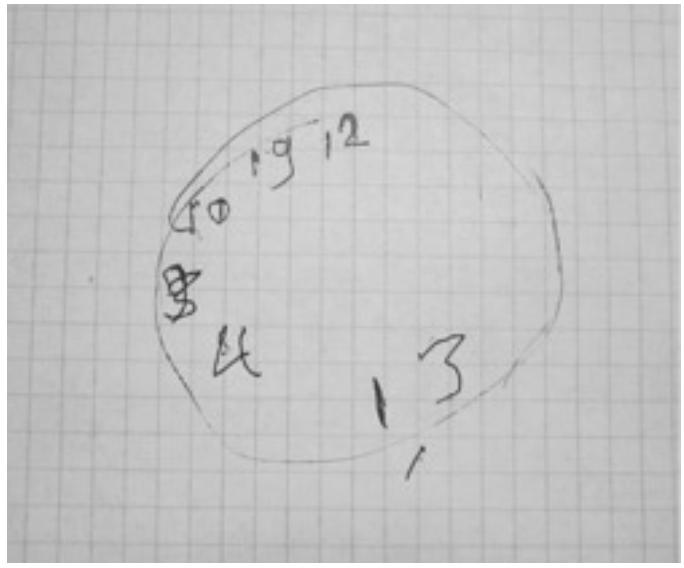

Figure 2. Patient's clock-drawing performance.

Table 1. Results of Neuropsychological Evaluation

Test	Performance
SMMSE	16/30
Attention	
Number range – forwards	4
Language and related functions	
Word/phonemic fluency	4/min
Category/semantic fluency	10/min
Comprehension	5/5
Repetition	15/15
Naming	
Object	6/10 (with delay)
Color	1/6
Reading	Impaired
Writing	Intact
Left – right orientation	Intact
Digit naming	Impaired
Topographical and visual skills	
Intersecting pentagons	Failed
Cube copying	Failed
Clock drawing	1/4 points*
Route description	Impaired
Agnosia	
Visual agnosia	None
Apraxis	
Ideational apraxia	None
Ideomotor apraxia	None
Limb-kinetic apraxia	None
Memory	
ECRT	20/48
Executive functions	
Trail-making test	Failed
Number range – backwards	2
Calculation	Failed
Pairwise similarity test	0/2

SMMSE: Standardized Mini Mental State Examination, Enhanced Cue Recall Test * (2)

the fusiform gyrus as a potential cause (5,6). Our patient also reported difficulty reading. His writing skill was mostly intact yet he was unable to read his own writing, recognize words, letters or numbers. With this set of symptoms, the patient fits into the alexia without agraphia description first made by Dejerine in 1892 (3). The discriminating property of pure alexia is that the reading difficulty and duration of reading increases as a function of word length. In the reading examination, the patient's performance was 3 words in one minute with letter-by-letter reading and individual fixations on every letter. While he could read shorter words more easily, he had more difficult time as the words got longer. The cases were left posterior fusiform or inferior temporal gyri or occipitotemporal sulcus is damaged or patients who read slower than 50 word/minute with an effort increasing as a function of word length should be evaluated for alexia without agraphia (7).

Hippocampus is affected in 21% of PCA infarctions (8). Its ventral portion is supplied by anterior coroidal artery while its dorsal 2/3rd depends on hippocampal artery which branches off PCA's P2 segment. In our patient, the acute ischemic lesions starting from the left hippocampal and posterior cingulate gyri and ending at dorsal hippocampus explain the anterograde amnesia in the ECRM test. Despite being a taxi driver, out patient could not determine a route between two destinations or estimate the distance to a given location. This finding (topographical disorientation) is explained by the involvement of allocentric spatial coding and mapping system and associated with the lesions in hippocampal, parahippocampal structures. In such tasks, right hippocampus is expected to play a bigger role but left hippocampal structures are also known to be activated (9). Similarly, left PCA infarctions may produce visuospatial deficits similar to right PCA lesions, but possibly with less severity (10).

Enhanced cue recall memory test (11), aims to find memory deficits independent of the educational level and it was previously validated and standardized in Turkish population. Its detection score of 41 out of 48 for Alzheimer's disease showed that the test has 100% sensitivity and 93.9% specificity (12). In the ECRM test, the patient was seen to recognize the objects on the cards in the first introduction and described their uses either by gestures or verbally but had trouble finding their names (anomia). The names of the objects were then taught to the patient. Despite this, his free and cue recall was found to be severely impaired in the later stages of the test even beyond anomic aphasia (ECRM point 20/48).

In order to name an object in a drawing, the object should first be recognized by the visual system. To differentiate the object among others with similar appearance, a semantic process takes place where following the semantic access to the object, the phonological mechanism for the articulation of the name is engaged and the motor program for the actual articulation is initiated (13). As seen from this pipeline, the sylvian and extrasylvian structures form a functional network for naming. A single structure that is responsible for anomia in its entirety is not described but much like our patient, left occipitotemporal (Brodmann area 37) infarctions were reported to cause anomic aphasia (14,15).

Patient's learning deficit can be explained both by left hippocampal lesion and left thalamic involvement. The thalamoperforate and thalamogeniculate arteries branching from P1 and P2 of PCA, posterior choroidal and the polar arteries supply blood to thalamus. In the PCA infarctions where thalamus is affected, cognitive deficits emerge either at clinical or subclinical levels. Neuropsychological deficits are observed especially in the anterior and paramedian infarctions of left thalamus. Lesions of mammillothalamic tract cause verbal learning and recall deficits while the dorsomedial nuclei involvement that is connected to Broca and Wernicke's areas give rise to various language deficits (16,17).

Our patient had both letter/word and digit recognition deficit. He was unable to complete the trail-making test because of these deficits. Due to his impaired ability to name colors, Stroop task was also not applicable. Among a number of executive function tests, Wisconsin card sorting task was thought to be suitable for this patient. He was able to group the cards based on their shapes but he failed the task altogether when the task was based on color and numbers and ended the test. Parahippocampal area and retrosplenial cortex have strong connections to frontal association areas like orbital and dorsolateral prefrontal cortices (18). In line

with the previous reports, the PCA infarction accompanied by parahippocampus and splenium caused a distinct loss of executive functions (19). Left thalamic lesion arguably also plays a role in this loss. Arithmetic calculations can be conceptualized as a multifactorial skill. Being a taxi driver, our patient had to rely on his arithmetic skills every day and but developed acalculia due to his condition. It can be seen that a loss of arithmetic abilities follow after a decay in linguistic, spatial and executive functions (20). The fact that he did not have lesions in the parietal lobe or either his angular and supramarginal gyri makes it hard to localize acalculia to a single structure.

We tried to form an association between the diffusion MRI and the early stage cognitive deficits. By doing so, we investigated the anatomical correlates of the acutely developed cognitive deficits that may disappear due to mental plasticity in the chronic stage.

A large portion of the cognitive tests, especially those targeting executive functions, is usable with patients with intact language abilities. With the application of proper neuropsychological tests in patients with reading or naming deficits, it is possible to differentiate the cognitive decline associated with PCA infarctions from hemianopia and sensorimotor loss and adopt an appropriate treatment approach.

References

1. Brandt T, Steinke W, Thie A, Pessin MS, Caplan LR. Posterior cerebral artery territory infarcts: clinical features, infarct topography, causes and outcome. Multicenter results and a review of the literature. Cerebrovasc Dis 2000;10:170-182.
2. Cangöz B, Karakoç E, Selekler K, Saat çizme testinin 50 yaş ve üzeri Türk yetişkin ve yaşlı örneklemi üzerindeki norm belirleme ve geçerlik - güvenirlik çalışmaları, Turkish J Geriatrics 2006:9;136-142.
3. Déjérine J. Contribution a l'étude anatomo-pathologique et clinique des differentes variétés de cécité-verbale. Mém Soc Biol 1892;4:61-90.
4. Geschwind N. Disconnexion syndromes in animals and man. I. Brain 1965;88:237-294.
5. Cohen L, Martinaud O, Lemer C, Lehéricy S, Samson Y, Obadia M, Slachevsky A, Dehaene S. Visual word recognition in the left and right hemispheres: anatomical and functional correlates of peripheral alexias. Cereb Cortex 2003;13:1313-1333.
6. Henry C, Gaillard R, Volle E, Chiras J, Ferrieux S, Dehaene S, Cohen L. Brain activations during letter-by-letter reading: A follow-up study. Neuropsychologia 2005;43:1983-1989.
7. Pflugshaupt T, Gutbrod K, Wurtz P, von Wartburg R, Nyffeler T, de Haan B, Karnath HO, Mueri RM. About the role of visual field defects in pure alexia. Brain 2009;132:1907-1917.
8. Szabo K, Förster A, Jäger T, Kern R, Griebe M, Hennerici MG, Gass A. Hippocampal lesion patterns in acute posterior cerebral artery stroke: clinical and MRI findings. Stroke 2009;40:2042-2045.
9. Maguire EA, Frackowiak RS, Frith CD. Learning to find your way: a role for the human hippocampal formation. Proc Biol Sci 1996;263:1745-1750.
10. Park KC, Lee BH, Kim EJ, Shin MH, Choi KM, Yoon SS, Kwon SU, Chung CS, Lee KH, Heilman KM, Na DL. Deafferentation-disconnection neglect induced by posterior cerebral artery infarction. Neurology 2006;66:56-61.
11. Grober E, Buschke H, Crystal H, Bang S, Dresner R. Screening for dementia by memory testing. Neurology 1988;38:900-903.
12. Saka E, Mihci E, Topcuoglu MA, Balkan S. Enhanced cued recall has a high utility as a screening test in the diagnosis of Alzheimer's disease and mild cognitive impairment in Turkish people. Arch Clin Neuropsychol 2006;21:745-751.
13. Hillis AE, Kleinman JT, Newhart M, Heidler-Gary J, Gottesman R, Barker PB, Aldrich E, Llinas R, Wityk R, Chaudhry P. Restoring cerebral blood flow reveals neural regions critical for naming. J Neurosci 2006;26:8069-8073.
14. Raymer AM, Foundas AL, Maher LM, Greenwald ML, Morris M, Rothi LJ, Heilman KM. Cognitive neuropsychological analysis and neuroanatomic correlates in a case of acute anomia. Brain Lang 1997;58:137-156.
15. Foundas AL, Daniels SK, Vasterling JJ. Anomia: case studies with lesion localization. Neurocase 1998;4:35-43.
16. Szirmai I, Vastagh I, Szombathelyi E, Kamondi A. Strategic infarcts of the thalamus in vascular dementia. J Neurol Sci 2002;203-204:91-97.
17. Stuss DT, Guberman A, Nelson R, Larochelle S. The neuropsychology of paramedian thalamic infarction. Brain Cogn 1988;8:348-378.
18. Schmahmann JD, Pandya DN, Wang R, Dai G, D'Arceuil HE, de Crespigny AJ, Wedeen VJ. Association fibre pathways of the brain: parallel observations from diffusion spectrum imaging and autoradiography. Brain 2007;130:630-653.
19. Park KC, Yoon SS, Rhee HY. Executive dysfunction associated with stroke in the posterior cerebral artery territory. J Clin Neurosci 2011;18:203-208.
20. Ardila A, Rosselli M. Acalculia and dyscalculia. Neuropsychol Rev 2002;12:179-231.