

Case Report

Early Marked Behavioral Symptoms in Bilateral Posterior Cerebral Artery Stroke: A Disguised Presentation

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ABSTRACT

Clinical signs and symptoms of posterior cerebral artery (PCA) stroke are varied and can be challenging to diagnose at early stage. A case of bilateral PCA infarct presenting with marked behavioral symptoms and minimal neurological symptoms is presented here. A 34 years old female had presented with marked behavioral symptoms, blurring of vision and tingling sensation in left half of body. Though the latter complaints resolved following day, her behavioral complaints persisted. Magnetic Resonance Imaging (MRI) of brain revealed acute non-hemorrhagic infarct in bilateral PCA territory. Psychotropics were beneficial for her behavioral symptoms. Isolated behavioral symptoms in PCA stroke led to speculate anatomical substrate for those symptoms. We discussed possible anatomical substrates for behavioral symptoms. Our case adds to the existing literature on a range of disguising presentations in PCA stroke and also emphasizes those distinctions between 'neurological' or 'psychological' or 'psychiatric' disorders are often sketchy.

Key words: Behavioral, cerebrovascular accident, posterior cerebral artery, stroke

INTRODUCTION


A recent review has documented several neurological signs and symptoms of posterior cerebral artery (PCA) stroke with variable focal neurodeficits.^[1] Although clinical features of PCA stroke has not been studied as extensively as in other vascular territories, it includes more clinical signs including frequent sensory, slight motor, and neuropsychological deficits than typical visual field deficits.^[2] Researchers have documented a

few other behavioral symptoms in PCA stroke, which pose a diagnostic challenge to clinicians. Bilateral PCA stroke has led to total dream loss in a 73-year-old man.^[3] Furthermore, changes of personality and emotional disturbances have been noted.^[4] Deranged circulation in occipital region has been associated with fetish behavior.^[5] Aggression, too, has been found rarely in a prospective observation in patients with acute PCA stroke.^[6] Added with this, agitated

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delirium was observed in medial temporo-occipital infarction.^[7] Occipital hypoperfusion has led to pathological laughter.^[8] We here report a case of bilateral PCA infarct presenting with marked behavioral symptoms and minimal neurological symptoms at our clinic.

CASE REPORT

Index patient, a 34-year-old female, hailing from a middle socioeconomic status, was brought to the clinic with marked behavioral symptoms. History revealed that about 10 days back, she had an abrupt onset of blurring of vision, with tingling sensation on the left half of her body. This subsequently improved within 12 h after being given benzodiazepines. Prominent behavioral changes followed this. She became exceedingly talkative, started repeating the same phrases many times, and would often ask for food after eating. She was unable to remember the particulars of her son like which class he was studying in. Reportedly, she was unaware of date and day, which started improving after about 7 days of onset of her illness. Biological functions were within physiological limit. She was unable to perform her household activities during the period, though she was taking care of her basic personal care under supervision. There was no history of psychoactive substance use. History of past medical or surgical or psychiatric illness revealed hypertension and occasional headache but without any psychiatric illness. Her medication history revealed she was on oral contraceptive pills and antihypertensive. There was no past history of any mental illness. Family history revealed dementia in her paternal grandfather. On physical examination, no focal neurodeficit was observed. She was cooperative toward the examiner, yet appeared anxious, restless and had stereotyped repetition of certain phrases. Speech output was increased in volume, and she was answering the queries of the examiner in an occasional irrelevant manner. No psychotic symptom was elicited. Judgment and insight was maintained. Her hematological and biochemical profile on the day of visit was unremarkable [Table 1]. She was started on divalproex sodium 500 mg at night. Considering her acute onset blurring of vision, a neurological opinion was sought. She was advised for neuroimaging and multiplanar magnetic resonance images of brain through T2-weighted and fluid-attenuated inversion recovery sequences revealed acute nonhemorrhagic infarct in bilateral temporo-occipital region (PCA territory) [Figure 1]. At follow-up after 3 weeks, she was still more talkative and irritable, and there had been occasional aggressive outbursts without any new physical complaints. She was complaining of headache at times and was sleeping less than usual.

Table 1: Hematological and biochemical investigation reports

Test	Value	Reference range
Hemoglobin	12.4 g/dl	12.0-16.0 g/dl
Total leukocyte count	10,200/mm ³	3,500-12,000/mm ³
Neutrophils	73%	35-66 %
Lymphocyte	21%	24-44 %
Monocyte	2%	4-8 %
Eosinophil	4%	0-5 %
Basophil	0	0-1 %
Fasting blood sugar	66 mg/dL	65-110 mg/dl
Urea	18 mg/dL	7-20 mg/dl
Creatinine	1.0 mg/dL	0.8-1.4 mg/dl
SGPT	22 IU/L	5-40 IU/L
SGOT	16 IU/L	7-56 IU/L
Sodium	144 mEq/L	133-146 mEq/L
Potassium	3.9 mEq/L	3.8-5.4 mEq/L
C reactive protein	12 mg/L	Upto 6 mg/L
Triglyceride	175 mg/dL	Normal ≤170
Total cholesterol	156 mg/dL	130-240 mg/dl
HDL	39 mg/dl	40-70 mg/dl
LDL	82 mg/dl	Normal ≤150 mg/dl
VLDL	35 mg/dL	8-28 mg/dl
Serum TSH	4.37 mIU/L	0.35-4.94 mIU/L
Free T4	0.98 ng/dL	0.89-1.76 ng/dl
Serum antiphospholipid IgM	6.14 U/mL	Normal <10; elevated >10
ELISA for serum MPO-ANCA	0.37 RU/mL	Negative <20; positive ≥20
ELISA for serum PR3-ANCA	1.19 RU/mL	Negative <20; positive ≥21

HDL – High-density lipoprotein; LDL – Low-density lipoprotein; TSH – Thyroid stimulating hormone; MPO-ANCA – Myeloperoxidase antineutrophil cytoplasmic antibody; PR3-ANCA – Proteinase 3 antineutrophil cytoplasmic antibody; SGOT – Serum glutamic oxaloacetic transaminase; SGPT – Serum glutamic pyruvic transaminase; VLDL – Very-low-density lipoprotein; T4 – Thyroxine

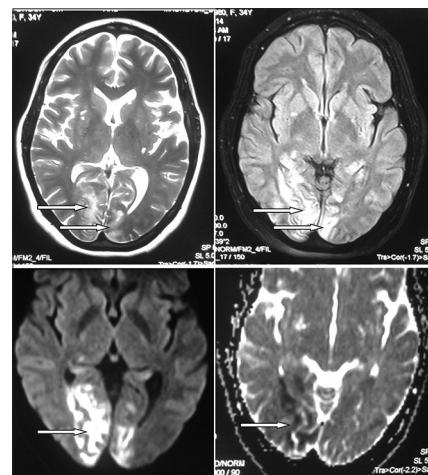


Figure 1: Top panel - T2 and fluid-attenuated inversion recovery images showing hyperintensities in bilateral posterior cerebral artery territory (right > left); bottom panel - diffusion weighted imaging and apparent diffusion coefficient images showing diffusion restriction (right > left)

DISCUSSION

Focal behavioral disturbance caused by cerebral ischemia can mimic “functional” disorder.^[9] Range of

behavioral symptoms in PCA stroke led to hypothesize about anatomical substrate for those symptoms and most of them remain speculative. Personality change and emotional disturbance in PCA stroke might be due to bilateral lesions within the medial inferior portions of the temporo-occipital lobe in the territory of the PCAs.^[4] DeJong *et al.* found in a similar case that the hippocampal formation, parahippocampal gyri, fusiform gyri, lingual cortex, and calcarine cortex were bilaterally involved. No impairment was observed in the thalamus, uncus, and amygdala.^[10] Decreased blood flow leading to decreased function of temporal lobe has led to fetish behavior.^[5,11] Release of the medial temporal region from other cortical areas or excitation of certain structures in the medial has been thought to produce agitated delirium in PCA stroke.^[7] Interruption of occipitotemporal connection has been blamed for aggressive behavior in PCA stroke.^[5] Interruption of cortico-pontine-cerebellar pathways including the ventral pontomedullary laughing center has been implicated for pathological laughter in PCA stroke.^[8,12,13] Memory impairment in our case might be due to involvement of lateral posterior choroidal arteries (branch of PCA), feeding inferomedial portion of temporal lobe, hippocampal gyrus, and hippocampus. Pain in the left half of body at earliest which improved later indicates thalamic involvement by thalamogeniculate branches.^[14] Bilateral lesions within the medial inferior portions of the temporo-occipital lobe in the territory due to the involvement of lateral occipital artery and/or lateral posterior choroidal artery might cause an emotional disturbance in our patient. An interruption in frontotemporal connection for speech might be speculated for her irrelevant talk and repetition of certain phrases.

CONCLUSION

Our case adds to the existing literature on a range of disguising presentations in PCA stroke. This case specifically highlights on subacute onset of behavioral disturbances with no/minimal neurological signs or symptom as a presentation of PCA stroke. Varied presentations of bilateral PCA stroke point toward its potential to be missed and diagnosed only at a relatively advanced stage at clinic. Clinicians should have high index of suspicion for PCA stroke and be aware of its range of probable clinical presentations which may mimic primary psychiatric and behavioral syndromes. This case also emphasizes that distinctions among

subcategories of brain disorders like “neurological” or “psychological” or “psychiatric” are often tenuous.

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Conflicts of interest

There are no conflicts of interest.

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