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Case report

Bilaterally impaired hand dexterity with posterior cortical atrophy

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ABSTRACT

A 79-year- old man presented with bilaterally impaired hand movements pertaining to handling of objects although hand movements without the use of objects were preserved, findings consistent with tactile apraxia. His hand and finger movements were slow and clumsy. He had an isolated optic ataxia, a component of Balint's syndrome. The computed tomography scan showed enlargement of the posterior horns of the lateral ventricles. He had recurrent falls probably owing to visual attentional deficits, which may be present in patients with posterior cortical atrophy. The findings can be deemed to fall within the posterior cortical atrophy spectrum. The underlying mechanisms are discussed.

1. Introduction

Klein[1] first used the term “tactile apraxia” to define the loss of purposive movements of the hand. Tactile apraxia is characterized by an isolated disturbance of hand movements for use of and interaction with objects (transitive), whereas intransitive movements are preserved.[2] It could impair hand and arm movements to the same extent as motor impairment resulting from damage to the motor cortex.[3] In patients with tactile apraxia, the lesion involves mainly the posterior parietal cortex, and posterior parietal lesions can give rise to severe disturbances of purposive behavior of the hand during explorative finger movements and manipulation of objects.[3] Posterior cerebral dysfunction may be the result of a variety of causes such as primary degenerative cerebral diseases, vascular causes, tumors, or metabolic factors.

Posterior cortical atrophy (PCA) is a clinicoradiologic syndrome.[4] It is a progressive neurodegenerative disorder characterized by an array of manifestations that include higher visual dysfunction with subsequent development of alexia, agraphia, visual agnosia components of the Balint's and Gerstmann's syndromes, and transcortical sensory aphasia attributed to involvement of the parietal and occipital cortices.[5] Among patients with PCA, several symptom clusters or subsyndromes emerged that have been recognized to have neurological correlates, for instance, Gerstmann's syndrome is attributable to left angular gyrus dysfunction and Balint's syndrome is attributable to bilateral parieto-occipital dysfunction. Memory and cognition are spared until late in the course of the disease.[5] Frontal lobe involvement becomes evident as the disease progresses. We describe a man with bilaterally impaired hand movements, severe ataxia, and recurrent falls associated with PCA.

2. Case report

A 79-year-old right-handed man was seen with a history of unsteady gait and recurrent falls, the duration of which was unclear but could be for 1 year or more. When seen he was able to stand on his own and could walk alone but unsteadily. He had mild hypertension and was on enalapril (Renitec; 5 mg daily). Examination of the nervous system revealed no weakness of the limbs. He was seen a few months later when his condition had worsened. His wife volunteered the information that she had to buy him several teacups with different sized handles as he was unable to maneuver his fingers of either hand to hold the cup by the handle. The cranial nerves were intact. Visual fields were normal by direct

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Table 1
Neurological findings.

Motor	
Power	Normal
Movements of hands and fingers[a]	
5 finger movements	Reduced, poor sequencing
Alternate, repetitive movements	Slow, clumsy
Paper crumbling, one finger tapping	Ineffective
Explorative pattern	Meaningless, ineffective
Reciprocal coordination	Not possible
Bimanual tasks—buttoning	Slow
Object bound hand movement with visual guidance	Not possible
Finger identification	Partially possible

[a] Bilaterally.

confrontation, pupillary reflexes were normal, and the external ocular movements were omnidirectional. Muscle strength and bulk were equal and normal in all four limbs. The tone was slightly increased in the limbs. The reflexes were normal and the plantars were flexor. Sensations for touch pain, deep pressure pain, and discriminative sensations such as joint sense, topographical localization, and direct simultaneous stimulation were normal. Stereognosis for size and shape was possible for large objects but he had difficulty with small objects because of decreased manipulative hand movements. His memory and cognition were mildly impaired. He has had no formal (school) education. He had never learned to read, write, or spell but could add and subtract single digits. Hence, the following items from the Mini Mental State Examination were not included: to spell, W O R L D and in reverse (5 points), write a sentence (1 point), and read "Close your eyes" (1 point), and his final score was 19 out of 23.

He was unable to direct his hands to targets under visual guidance, for example, when asked to touch a number of targets on a picture he was unable to do so, pointing erratically, nor was he able to trace the outline of the map (optic ataxia). He was able to gaze when desired (no apraxia of gaze) and was able to pick up almost all the objects on a tray (no simultanagnosia). He was unable to perform rapid finger movements with or without visual guidance in both hands. Sequential movement of the fingers—touching the tip of the thumb with the palmar surface of the other four fingers—was not possible in both hands with eyes closed and open. Right–left discrimination was possible, and finger identification was partial. There was no apraxia for intransitive movements such as to wave goodbye, stop, go, and salute, but not to transitive movements such as opening the door with the key, using a scissors, and using a screwdriver. The neurological findings are summarized in Table 1.

The carotid Doppler revealed mildly echogenic plaques in both carotid bulbs at the origin of both internal carotid arteries. The right carotid showed 15–49% stenosis. The left carotid was normal. The computed tomography (CT) scan of the brain showed marked enlargement of the occipital horns of the lateral ventricles and widened sylvian fissures together with prominent sulci over the parietal and occipital regions bilaterally. There were hypodense areas in relation to the anterior horns and in the paraventricular regions. There was no evidence of recent or old cerebral infarction (Fig. 1).

3. Discussion

There are several subsets in PCA consisting of variable components of Balint's and Gerstmann's syndromes, but the most

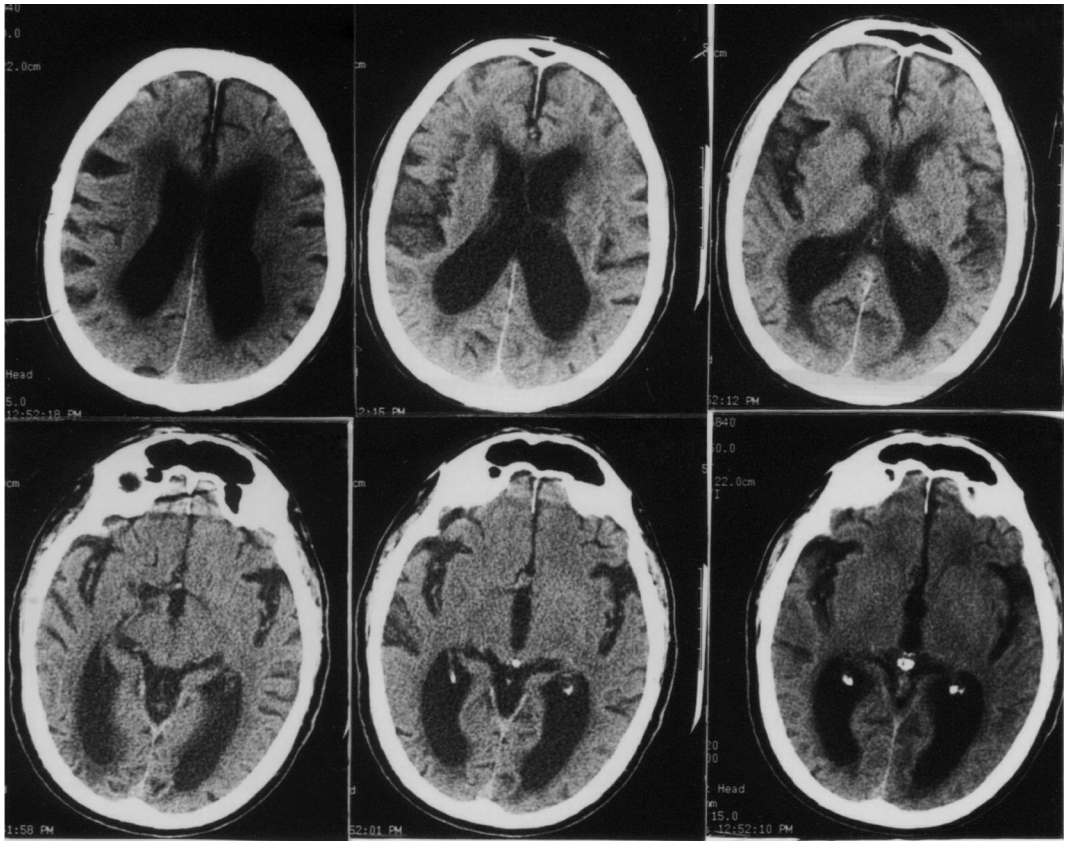

Fig. 1. Computed tomography (CT) scan showing bilateral symmetrical striking enlargement of the posterior horns of the lateral ventricles with prominent sulci in the parietal and occipital regions and widening of the sylvian fissures bilaterally.