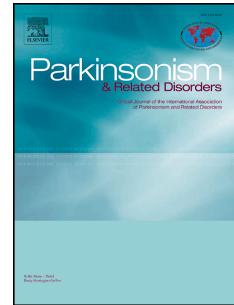


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“Video is part of the manuscript”

**Transient blepharospasm, apraxia of eyelid opening, and hemidyskinesia
following a right parietotemporal infarct**

Cheng-Yang Hsieh^{a,b}, Pi-Shan Sung^c, Wen-Juh Hwang^d

^aDepartment of Neurology, Sin Lau Hospital, Tainan, Taiwan

*^bInstitute of Biopharmaceutical Sciences, College of Medicine, National Cheng Kung
University, Tainan, Taiwan*

^cDepartment of Neurology, National Cheng Kung University Hospital, Tainan, Taiwan

*^dDepartment of Neurology, National Cheng Kung University Hospital, College of
Medicine, National Cheng Kung University, Tainan, Taiwan*

Corresponding author:

Wen-Juh Hwang, MD

Department of Neurology

National Cheng Kung University Hospital

College of Medicine, National Cheng Kung University

138 Sheng-Li Road, Tainan 704, Taiwan

Tel: +886-6-276-6187; Fax: +886-6-237-4285; e-mail: wjhwang@mail.ncku.edu.tw

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Blepharospasm and apraxia of eyelid opening have rarely been reported in acute nondominant hemispheric stroke [1,2]. It is also rare for hyperkinetic movement disorders to develop after acute hemispheric stroke [3]. We report a patient with an acute right parietotemporal infarct who presented with transient blepharospasm, apraxia of eyelid opening, and left hemidyskinesia.

A 71-year-old right-handed man was admitted with acute onset of involuntary movement in his left limbs and face, and difficulty opening his eyes. He had a medical history of hypertension, atrial fibrillation, rheumatic heart disease after mechanical mitral valve replacement, and congestive heart failure. He was treated with digoxin, furosemide, and warfarin. Neurological examinations showed normal orientation and normal speech, without aphasia or dysarthria. He had right-to-left disorientation, left hemineglect, left homonymous hemianopia, visuospatial impairment, and mild left hemiparesis, but no asomatognosia, astereognosia, or anosognosia. His pupils, ocular motility, somatosensory system (evaluated by pinprick and sense of passive movement), cerebellar system, and sphincters were normal.

The patient showed intermittent forceful contraction of the bilateral orbicularis oculi and corrugator muscles. He was also unable to open his eyes at will despite vigorous forceful contraction of the frontalis muscles and in the absence of orbicularis oculi contraction. There were frequent intermittent, irregular, fast, and more or less

patterned movements in his left limbs and face in the wakeful state (Video). His left hand was continually grasping. He did not display compulsive exploration of the environment or intermanual conflict. The patient did not show any avoidance posture or levitation in his left upper limb.

A brain computed tomography (not shown) and magnetic resonance imaging (Fig. 1) showed acute infarction in the right parietotemporal lobe. The basal ganglia, brainstem, and thalamus were normal. The dyskinetic movement responded well to intravenous diphenhydramine (30 mg): it completely disappeared in 10 minutes and recurred 4 hours later. He was then treated with haloperidol (15 mg/day). Both eyelid abnormalities and left hemidyskinesia disappeared in 4 days. The dose of haloperidol was gradually tapered and discontinued 10 days after its initiation, without a recurrence of the eyelid abnormalities or left hemidyskinesia.

The temporal lobe regulates memory, emotions, impulse control (sex, eating), hearing, smell, language, and learning. The amygdala in the temporal lobe plays a major role in processing unconscious emotional facial expression. Currently, there is no neurobiological evidence to support a link between the temporal lobe and the motor control of eyelids and limbs through the pyramidal tract [4]. Because the temporal lobe does not contribute to the pyramidal tract, the amygdala was not involved in our patient, and because the patient was conscious of his involuntary

movement, we proposed that the eyelid abnormality and hemidyskinesia were related to the parietal lobe lesion.

Parietal stroke rarely presents with hemiballism-hemichorea (HB-HC) [5], alien hand phenomenon, or action or delayed dystonia. The left hemidyskinesia in our patient is different from previous observations of patients with hemidystonia, which usually develops several months to years after an acute insult. Hemidystonia is usually refractory to treatment and persists after its appearance. The more or less patterned movement and a predominant involvement of the face and upper limb are different from HB-HC. "Pseudochoreoathetosis" is also unlikely, because our patient had no sensory abnormalities. The involvement of the face, the continual left hand grasping behavior, and the lack of avoidance response or levitation, did not favor posterior alien hand syndrome. We speculate that the parietal lobe lesion caused ignition failure for the voluntary eyelid opening (apraxia of eyelid opening) and disinhibition of basal ganglia and brainstem interneurons with the manifestations of blepharospasm and left hemidyskinesia.

Diphenhydramine is an anticholinergic antihistamine effective for treating acute akathisia and acute dystonic reaction. The sedative effect of antihistamine may suppress movement disorders. However, our patient remained clear and his hemidyskinesia disappeared after diphenhydramine treatment. *N*-methyl-D-aspartate

(NMDA) receptor antagonists such as amantadine alleviate dyskinesia in patients with Parkinson's disease. Because diphenhydramine, a histamine-1 receptor antagonist, has not been reported to affect NMDA receptors, the clinical effect is not likely due to an interaction with the glutaminergic pathway. Determining how diphenhydramine suppresses the dyskinesia in this patient is further complicated by the fact that the mechanism of hemidyskinesia is not fully understood. The paradoxical response of hyperkinetic movement disorders both to anti-cholinergic and to anti-dopaminergic agents was unclear.

This report has several limitations. First, we did not have simultaneous electromyographic recordings from the levator palpebrae superioris and orbicularis oculi muscles. Second, we did not have a blink reflex to suggest hyperexcitability of the brainstem interneurons. Third, we lacked perfusion imaging in the acute stage to exclude basal ganglia hypoperfusion.

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Legend to the Video

The video shows blepharospasm, apraxia of eyelid opening, and hemidyskinesia in the left limbs and face. The patient is asked to open and close his hands in succession: right hand first and then the left hand. He shows right-to-left disorientation when he is asked to hold up one finger of the left hand. When he is asked to use his left hand to touch his right ear, his left hand went to his left ear first without touching it, and then moved to touch his right ear. The left hemidyskinesia is not aggravated during the voluntary movements.

FIGURE LEGEND**Fig. 1.**

A diffusion-weighted magnetic resonance image (MRI) (left column) showing restricted diffusion in the right parietotemporal lobe. A T2-weighted MRI (right column) showing high signal intensity in the corresponding area. The midbrain, subthalamic nucleus, striatum, globus pallidus, and thalamus were normal in this study. The image shows acute infarction in the right parietotemporal lobe.

