

Arm levitation sign in acute right frontoparietal infarct

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ABSTRACT

نستعرض في هذا المقال حالة مريضة تبلغ من العمر 80 عاماً وتشكو من جلطة حادة في الفص الجبهي الجداري الأيمن من المخ (right fronto-parietal stroke)، بالإضافة إلى ظهور علامة مهمة في الفحص السريري العصبي ألا وهي استرفاع الذراع (arm levitation). وسوف نناقش علاقة هذه العلامة بنتائج تصوير المخ وسنقوم بافتراض المسببات الوظيفية الممكنة لظهور هذه العلامة. كما أننا سنناقش إمكانية ظهور هذه العلامة نتيجة خلل في التوصيلات العصبية إضافة إلى المشاكل التي قد تواجه عملية إعادة تأهيل المرضى.

We present the case of an 80-year-old female with acute right fronto-parietal stroke and an interesting neurological sign on clinical examination; the arm levitation sign. We discuss the imaging correlates of this sign and hypothesize on the possible functional etiology of the sign. We also discuss in brief, the possibility of neuronal misconnections causing the sign and the resultant problems with rehabilitation when patients have this sign.

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Alien hands syndrome (AHS) is described as involuntary complex motor activity of a limb along with denial of ownership or feeling of foreignness. Typically, AHS is seen with lesions involving the anterior corpus callosum or medial frontal lobe. Involuntary spontaneous arm levitation (SAL) has been described as a posterior variant of AHS in patients with a nondominant parietal lobe lesion.¹ In this report, we described a patient with an acute right fronto-parietal stroke and involuntary arm

levitation, but lacking spontaneity of movement and feeling of strangeness, the arm levitation sign.

Case Report. An 80-year-old right-handed woman with a history of hypertension presented to the emergency department with sudden onset of left sided weakness. Neurological examination revealed left upper motor neuron facial weakness, left hemiplegia, left hemisensory loss, and right gaze preference. A noncontrast head CT scan (Figure 1) showed subtle hypodensity in the superior and posterior right frontal lobe. A CT angiogram showed occlusion of the distal right anterior cerebral artery, as well as occlusion of the right M2 and posterior division of the right M3 segments of the middle cerebral artery. She was given intravenous alteplase (rtPA) followed by intra-arterial rtPA within 2 hours of the onset of symptoms. A brain MRI revealed acute infarcts in the right fronto-parietal cortex (Figure 2). The next day, she demonstrated marked improvement in power in her left arm. Power in her left leg did not improve. Left hemisensory loss persisted. Her left arm was noticed to involuntarily levitate in supination with elbow flexion and finger extension with dystonic posturing of the fingers. This sign occurred only when she tried to elevate the weak left leg, but not spontaneously. There was no aphasia, frontal release signs, agnosia, apraxia, right-left disorientation, inter manual conflict, or foreign perception of the hand. Deep tendon reflexes were brisk on the left. The plantar response was equivocal bilaterally.

Discussion. Our patient had left arm levitation whenever she tried to lift the weak left leg without any other component of AHS. The SAL sign is recognized as a posterior variant of the AHS. This sign is typically seen in cortical-basal ganglionic degeneration. The SAL was reported in 4 pathologically proven cases of progressive supranuclear palsy (PSP). In addition to the classical features of PSP, neuropathological examination in this series showed areas of cortical degeneration in the motor and premotor cortex.² In another series, 60% of patients with pure parietal strokes were found to have laterodeviation or levitation of the arm when they were asked to keep both arms extended forwards with the

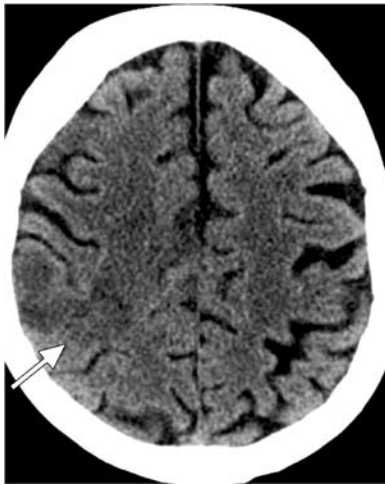


Figure 1 - A noncontrast axial CT scan demonstrates hypodensity in the superior and posterior right frontal lobe.

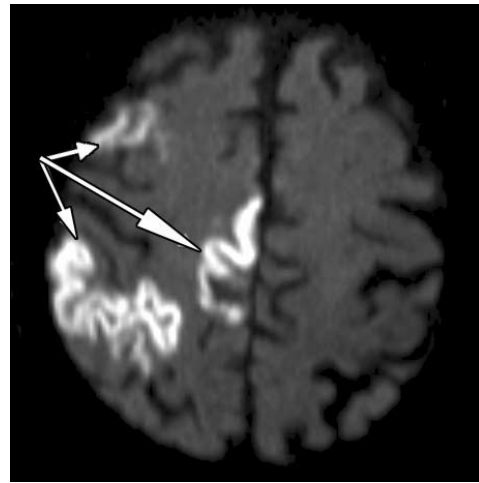


Figure 2 - Diffusion weighted MRI shows areas of restricted diffusion suggestive of acute infarction in the right frontal and parietal cortex.

palms up.³ Although cortical involvement is a common feature in the aforementioned diseases, arm levitation has also been reported in right thalamic hemorrhage,⁴ and left pontine hemorrhage.⁵ These cases have proprioceptive sensory loss in the affected arm, which may play a role in the pathophysiology of SAL.

The unique features in our patient are the absence of strangeness and inter manual conflict, thus suggesting that this is not AHS, and absence of spontaneity of the movement, thus suggesting that it is neither SAL. There were no other associated abnormal movements.

We hypothesize that involvement of the premotor and the sensorimotor cortex as well as the subcortical white matter tracts may have caused distortion and resultant misconnections in the cortico-striato-thalamic pathway. As a result of these misconnections, the arm motor cortex may have been activated instead of the leg motor cortex when she was instructed to move her left leg. Alternatively, the activated leg motor cortex may have been misconnected to the subcortical white matter fibres that originally serve the arm. Additional loss of sensory modalities of the left arm and leg resulted in unawareness of the position of the left arm when trying to lift the weak leg distracted her attention. Misconnections of the type we propose may impair rehabilitation, and novel techniques may be needed to help in recovery.⁶

Functional MRI and MR tractographic studies along with EEG and somatosensory evoked potentials may be needed to substantiate our hypothesis and show the functional correlates of this rare neurological sign.

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