

Case Reports

Arm Posturing in a Patient Following Stroke: Dystonia, Levitation, Synkinesis, or Spasticity?

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Abstract

Background: Post-stroke movement disorders occur in up to 4% of stroke patients. The movements can be complex and difficult to classify, which presents challenges when attempting to understand the clinical phenomenology and provide appropriate treatment.

Case Report: We present a 64-year-old male with an unusual movement in the arm contralateral to his ischemic stroke. The primary feature of the movement was an involuntary elevation of the arm, occurring only when he was walking.

Discussion: The differential diagnosis includes dystonia, spontaneous arm levitation, synkinesis, and spasticity. We discuss each of these diagnostic possibilities in detail.

Keywords: Post-stroke movement, dystonia, levitation, synkinesis, spasticity

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Introduction

Abnormal movements develop as residual complications in up to 4% of patients with strokes.^{1,2} The movements are usually focal or unilateral, and contralateral to the injury. Occasionally, they may be bilateral or segmental.¹ The types of movement are numerous; hemi-ballism/hemi-chorea, dystonia, tremor, and myoclonus have all been reported following stroke.^{3,4} Here, we present a patient who exhibited an unusual involuntary elevation of his arm while walking. The clinical phenomenology was difficult to classify, as the movement resembled a number of different entities. This presented a challenge when considering the appropriate treatment. Given the challenges in assigning a diagnosis, we thought a detailed delineation and discussion of the differential diagnosis would be both instructive and educational to a broader community.

Case report

The patient is a 64-year-old right-handed male with a history of diabetes mellitus; he presented to an outside hospital in January 2013 after collapsing in his bathroom. At that point, he was found to have

marked left face, arm, and leg weakness due to a right middle cerebral artery (MCA) territory stroke. The etiology of this stroke was not known. As he had been on 81 mg/day acetylsalicylic acid, he was switched to clopidogrel, 75 mg/day. He was recovering strength and function well with the help of extensive outpatient rehabilitation. In early 2014, approximately 1 year after his stroke, he first noticed mild shaking of the left thumb. In January 2015, his left hand shaking worsened and he also began to notice some stiffness and pain in the left arm. When he presented to the Yale Movement Disorders Clinic in May 2015, he was taking clopidogrel (75 mg/day), baclofen (for stiffness, 20 mg/day), quetiapine (for post-stroke behavioral problems, 50 mg/day), and escitalopram (for depression, 40 mg/day). Brain magnetic resonance imaging (MRI) showed an old right MCA territory infarct with significant encephalomalacia and Wallerian degeneration extending all the way down to the cerebral peduncles and pons (Figure 1). On our initial examination in May 2015, he had a normal mental status. An upper motor neuron-type facial droop on the

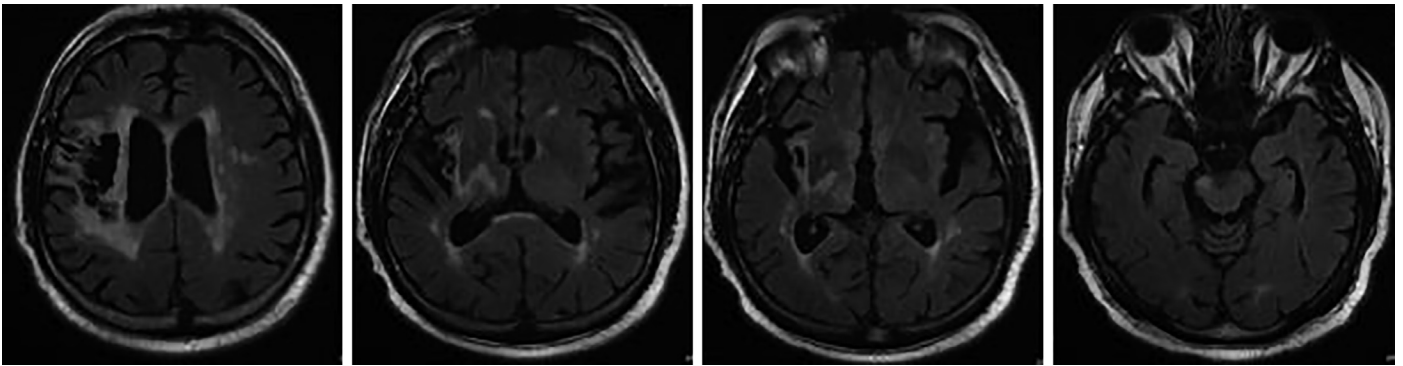


Figure 1. T2-weighted Magnetic Resonance Imaging. Sequences show degeneration in the right frontoparietal cortex, deep gray structures, and the cerebral peduncle.

left, and slight dysarthria, were observed. No sensory deficits were observed on examination. On motor examination, he had mild 4 to 5–/5 weakness in the left arm and leg, and full strength in the right arm and leg. He had increased tone in the left arm at the elbow, with normal tone on the right side. Reflexes were asymmetric and increased on the left. Fine finger movements such as finger taps were somewhat clumsy and abnormal on the left and were assigned Unified Parkinson's Disease Rating Scale scores of 1.⁵ As he rested his left arm in his lap, there was mild dystonic extension of both the thumb and the first finger. A tremor of the left thumb was noted, which resembled a rest tremor; however, the thumb was not fully at rest because it was mildly dystonic. In this manner, the tremor was reminiscent of those observed in many patients with Scans Without Evidence of Dopaminergic Deficit,⁶ indicating that the patient likely did not have underlying Parkinson's disease but rather had dystonia. He had mild difficulty walking, with a tendency to circumduct his left leg. As he walked, his left arm began to slowly elevate, with abduction of the arm at the shoulder joint as well as flexion and elevation of the elbow. When he stopped walking, his left arm returned to his side, and there was slight dystonic posturing (fisting) in the left hand (Video 1). There was no arm elevation while he was seated. The tremor in his left thumb continued as he walked. He was asked to taper and then stop quetiapine. When re-examined 3 and 9 months after stopping quetiapine, his tremors had not stopped and the remainder of his examination remained unchanged.

Discussion

We present a 64-year-old male with an involuntary movement in the arm contralateral to his ischemic stroke. The most striking feature of the movement was an elevation of the arm, occurring only when he was walking. This movement is unusual and interesting, and the diagnosis not immediately obvious. Given the nature of the movement, a number of diagnostic possibilities should be considered: dystonia, spontaneous arm levitation (SAL), synkinesis, and spasticity.

The patient's abnormal finger posture at rest and arm elevation while walking may be dystonic. Dystonia is characterized by intermittent or sustained muscle contractions, causing abnormal

movements or postures.⁷ Dystonia is often initiated or worsened by voluntary muscle action, and associated with overflow muscle activation. Post-stroke dystonia has been reported in the setting of lesions of the lentiform nuclei as well as the frontoparietal cortex, the thalamus, and the brainstem.⁴ Such dystonias usually occur with the recovery of motor function and strength.⁸ They can manifest in a variety of ways, and may be accompanied by other movements, such as tremor, or athetosis.^{9–15} Our patient's arm elevation could have been dystonic. The presence of mild dystonic posturing and dystonic tremor in two of the fingers of the left hand, and the fact that the involuntary arm elevation was task specific (i.e., aligned with walking) further support this diagnosis. Of interest is that dystonic posturing of the arm, with internal rotation of the shoulder and flexion of the elbow (termed "handbagging"), has been reported in Huntington's disease patients during the act of walking.¹⁶ Although the precise posture seen in that setting is not the same as that seen in our patient, the phenomenology is very reminiscent.

SAL is an alternative diagnosis to consider, and often presents as part of alien hand syndrome (AHS) due to parietal lobe involvement in degenerative diseases such as corticobasal ganglionic degeneration, Creutzfeldt–Jakob disease (CJD), and progressive supranuclear palsy.^{17–19} The predominant movement in our patient was an elevation of the arm. Although this was involuntary, he did not endorse any foreign-ness of the arm, intermanual conflict or spontaneous exploratory behavior, which are typical of AHS. SAL has also been observed following strokes, and particularly those affecting the corpus callosum or frontoparietal regions, due to disruption of the corticothalamic inhibitory pathways.^{20–24} The arm elevation occurred slowly in our patient, which favors SAL, as abnormal postures often occur with less slowness in dystonia. He was also not aware of any sensory tricks to control these movements, as may be seen in patients with dystonia. However, the fact that the elevated posturing was linked to one specific task (i.e., walking) and did not occur while seated or standing, argue against SAL. Furthermore, the observed flexion at the elbow, rather than straight arm elevation, which is more characteristically seen in SAL, further argues against SAL.



Video 1. Arm Elevation While Walking. There is intermittent tremor of the left thumb and first finger during the videotape. When he stops walking, the left arm is no longer elevated but there is some residual posturing (fisting) of the left hand. The patient provided signed, written consent to be videotaped.

Synkinesia is a rare disorder in which an involuntary movement occurs in coordination with a voluntary movement. It is postulated to be secondary to aberrant growth of regenerating neurons following injuries, and has been well documented in lesions of the facial nerve and brachial plexus.^{25–27} Synkinesias of the limbs can be homologous or heterologous. Homologous or mirror-movement synkinesias occur in the muscle group contralateral to the side involved in the primary motor actions. They are seen physiologically in children, as well as in

Parkinson's disease and CJD, where there is activation of the contralateral pre-motor cortex due to reduced inter-hemispheric inhibition.^{28,29} Heterologous synkinesias include ipsilateral hand-foot synkinesia due to increased activation of the supplementary motor cortex.³⁰ Elevation of the paretic arm while moving the ipsilateral leg during recovery from contralateral thalamic hemorrhage has been reported.³¹ However, in our patient, we did not notice any particular association of the elevation of the left arm with the movements of the other limbs. Pathologic associated movements in hemiplegic patients are a type of synkinetic movement characterized by involuntary activity in paretic groups of muscles that are stimulated by active innervation of other groups. They occur due to increased tone in the paretic and spastic arm resulting in abnormal postures, especially under physical exertion. The extent of these movements directly correlates with the degree of spasticity in the affected limb. Examples include the Strumpell's pronator sign, where active flexion of the paretic forearm can lead to arm pronation and flexion of the hand. However, in our patient, spasticity though present was mild. Second, his movements were mainly characterized by an abduction at the shoulder joint and flexion of the elbow, which does not fit with the typical pattern of associated movements described above.

Spasticity is a common complication following stroke and is defined as a velocity dependent increase in resistance during passive stretch resulting from hyper-excitability of the stretch reflex.³² It is associated with increased tone and hyper-reflexia. Spasticity of the arm is associated with increased activity of the flexor muscles at both the shoulder and the elbow, with extreme cases leading to contractures. In our patient we noted some spasticity, with mildly asymmetric reflexes, which were increased on the left. His arm posturing had some features that are similar to those seen in patients with spasticity, with flexion at the elbow joint being the most notable of these. However, with spasticity, it would be expected that the patient would similarly exhibit the posture at rest, which was not observed in our patient. Furthermore, significant weakness, an expected feature with spasticity, was also not seen in our patient. Therefore, although he had some features of spasticity in the left arm, which could have contributed to his phenomenology, we do not believe this was the primary or sole explanation for his arm posturing and elevation.

We also do not think his motor phenomenology was consistent with or due to underlying Parkinson's disease. While his fine finger movements on the left were clumsy and abnormal, we did not notice any decrement or pauses typical of Parkinson's disease. His fine finger movements on the right were normal. Similarly, tone was normal on the right and deemed to be spastic rather than rigid on the left. Furthermore, while his tremor in the left thumb resembled a rest tremor, it had a clear dystonic component.

In summary, our patient's arm elevation shared features with all of the above-discussed entities, although not to the same degree. We believe our patient's abnormal arm posturing is best explained as a form of dystonia. There was dystonic posturing of the fingers at rest, which supports this notion. Second, arm elevation occurred while walking, suggesting task specificity.

Arriving at the correct diagnosis is important in terms of planning the appropriate treatment. Given the nature of his movements, medications that reduce the severity of dystonia were considered to be the best first-line treatment. The patient was already taking oral baclofen, and was not eager to increase the dosage due to the potential for side effects. Additional medications, including trihexyphenidyl and botulinum toxin, were discussed with him. He is currently considering these options. He was also referred to a physiatrist and advised to follow up with the movement clinic to re-evaluate his symptoms.

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