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LETTER TO THE EDITOR

Mirror movements and mirror dystonia in a patient with dystonic tremor due to stroke

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A 59-year-old male patient presented to the Diskapi Yildirim Beyazit Traning and Research Hospital, Department of Neurology, in February 2022 with complaints of involuntary tremulous movements in the left hand and contractions of the lower limb, which had started four years ago, soon after a hemorrhagic stroke of the pons in 2017. The patient had a history of a right thalamic stroke four months before the pontine stroke. The patient had suffered from mild left-sided paralysis after the initial thalamic stroke; however, it had recovered almost totally prior to the second stroke. The involuntary movements in the left side started a few weeks after the pontine hemorrhage and no marked progression occurred up to date. The neurological exam revealed slight left-sided hemiparesis (5-/5 muscle strength) and hemihypoesthesia. The extrapyramidal exams revealed both continuous or intermittent muscle contractions causing abnormal, irregular, repetitive movements in the left hand and tonic contractions in the lower limb that were compatible with dystonia. A written informed consent was obtained from the patient.

Remarkably, simultaneous contralateral, involuntary, and identical movements of the dystonic hand accompanied the voluntary movements of the unaffected hand (right hand), which was compatible with mirror movements (MMs; Video 1). Cranial magnetic resonance imaging showed the areas of encephalomalacia in the pons and dorsal portion of the right thalamus (Figure 1). The patient refused botulinum toxin therapy. However, the clonazepam dosage was increased from 2 to 4 mg, which provided a moderate improvement in the symptoms.

Mirror dystonia (MD) can be defined as a dystonic movement emerging in a dystonic limb when it is relaxed and the opposite limb is activated.[1] The increased incidence of MD in patients with hemidystonia and focal hand dystonia is an acknowledged issue, and it is accepted as an extremely useful diagnostic physical sign.[2] Mirror dystonia usually presents in the affected hand of patients who attempt to learn to write with their nondominant hand, suggesting an underlying dysfunctional neural plasticity.[3] Remarkably, the presence of MD and its clinical correlates have rather been studied in patients with idiopathic dystonia, particularly those with hand dystonia.[2,3] On the other hand, the clinical features of MD and its incidence in poststroke dystonia (or those secondary to structural lesions), where the mechanisms of neural plasticity are prominently involved, represent a subject that has not been studied. Therefore, the presentation of this patient and accompanying MM may be important for further deliberations. Mirror movement is a distinct entity from MD; however, the phenomenology of MD and MM resemble each other, and similar mechanisms might play a role in these two occurrences.[1,3] Mirror movements are involuntary movements of homologous muscles during voluntary movements of contralateral body regions. They are often observed after strokes and also in patients with old pyramidal tract

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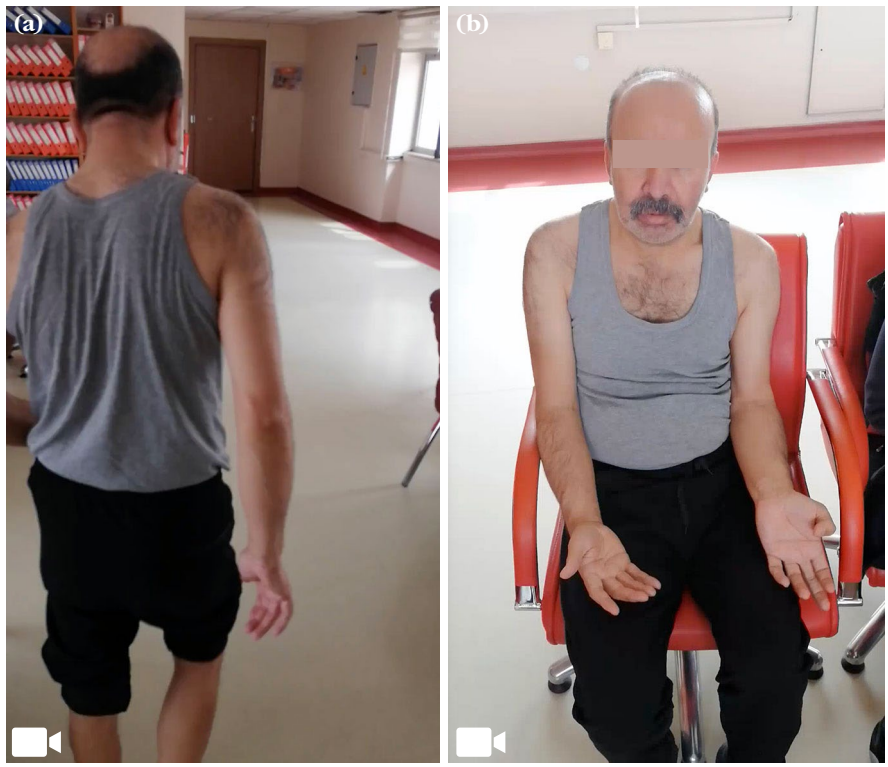

Video 1. The neurological exam of the dystonia in the left hand and gait of the patient. The extensor posture of the hand and the flexor posture of the forearm is observed during the interview. In addition to the sustained and intermittent muscle contractions of the left hand and forearm, **(a, b)** the often tremulous irregular dystonic movements are demonstrated. The overflow pattern of the dystonia, MD, and MMs are demonstrated. **(b)** In the last part of the video, the patient is instructed to perform voluntary movements with the right hand (unaffected), which leads to MMs.
MMs: Mirror movements.

lesions of other causes, suggesting a problem in the plasticity following stroke.[4] Mirror movements are generally in nonparetic limbs when patients move the paretic limb and mostly occur in the hands. A hypothesis to explain the MM after stroke is hyperactivation of the nonlesioned hemisphere after stroke.[4] Our observation of MD and MM in our patients with poststroke dystonia may point out the possible role of the ipsilateral motor area in the phenomenology. In an previous study, patients with hemidystonia due to structural lesions showed increased activity in both primary and accessory motor areas while performing tasks with the dystonic arm; similar but less marked changes were shown when the patients used their clinically unaffected arm.[5] However, the activation in the motor area was found to be decreased in patients with idiopathic dystonia.[5] The researchers hypothesized that the higher level of cortical activation in the group with acquired dystonia was a reflection of normal cortical response to subcortical pathology, whereas in the idiopathic group, the disease process might affect the primary sensorimotor cortex, leading to decreased activation in these regions.[5] In light of these data, the presentation of MD and MM in our patient with poststroke dystonia may be illustrative. We believe that the clarification of the presence of MD and MM, particularly in dystonia subjects secondary to structural lesions, may provide perspectives regarding the pathophysiology of dystonia. These results may give evidence about the contribution of the ipsilateral motor area of the brain in the dystonia pathophysiology.

Finally, another important aspect was the lesion site of the pontine hemorrhage that led to dystonia. The poststroke dystonia has been rather associated with the structurals lesion in the basal ganglia and thalamus.[6] However, there are several case reports illustrating dystonia subjects associated with pontine stoke.[6,7] Acute dysregulation of pallidal efferents

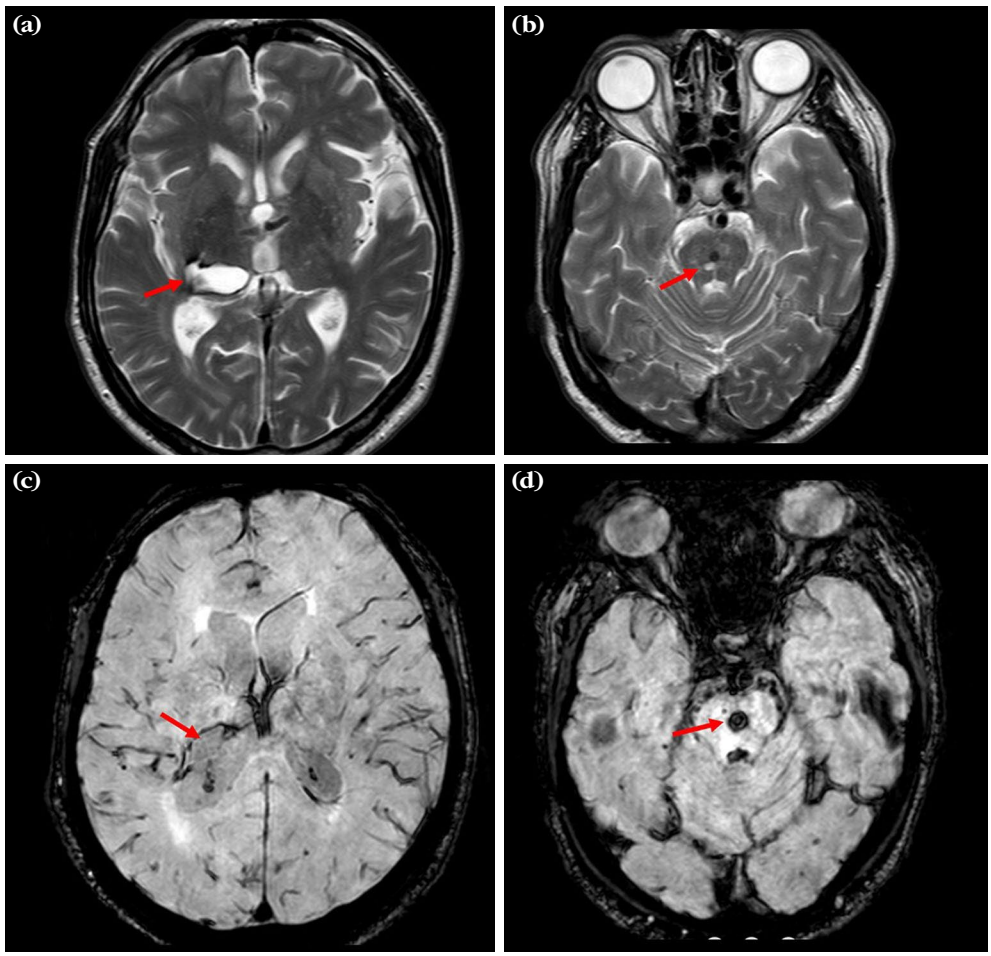

Figure 1. The cranial magnetic resonance imaging sequences showing the sequela lesion of the ischemic stroke in the ventral posterolateral and dorsal portions of the right thalamus **(a, c**; indicated by arrows on T2-weighted images) and sequela of the pontine hemorrhage **(b, d**; indicated by arrows on susceptibility-weighted imaging).

to the pedunculopontine or pontine afferents to the thalamus was hypothesized to lead to hemidystonia.[6] The documentation of our patient also contributes to the literature in this regard. However, although the association between the pontine hemorrhage and the occurrence of dystonic movements is clear in terms of temporal course, the contributory effect of the thalamic lesion in the phenomenology of dystonia and specific clinical features cannot be excluded.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Concept, design, supervision, analysis and/or Interpretation, critical review: H.O., S.C; Materials, data collection and/or processing, literature search, writing manuscript: H.O.

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