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LETTER TO THE EDITOR

Focal localized enhanced physiological tremor after physical insult

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Sirs,

Enhanced physiological tremor (EPT) is a strengthening of physiological tremor to more visible levels, and it is associated with 8 to 12 Hz central neurogenic oscillators, which are enhanced by reversible conditions such as anxiety, fatigue, hyperthyroidism, and drugs [1, 2]. This normally manifests as bilateral upper-limb action tremor, which, by definition, encompasses postural and isometric tremor and sometimes resting tremor [2, 3]. EPT can be diagnosed retrospectively, when the tremor has been normalized after appropriate management [2].

Peripheral trauma may induce tremor even without overt tissue and/or nerve damage [4]. Some argue that movement disorders after trauma are mere coincidence and would be confounded by concurrent psychogenic origin. However, the causal relationships between movement disorders and trauma are increasingly advocated by others [4–6].

Herein, we report a rare experience of localized EPT that was timely and anatomically locked to the site of a physical insult.

A 16-year-old girl complained of tremulousness of her right hand for 1 month. The tremor began 2 days after she accidentally hit a wall with her clenched right hand. The pain was transient though severe. She did not have any problems manipulating her hand thereafter, nor did the tremor hinder her normal activities. Her hand was examined by an orthopedic doctor and was found to be free of bone fracture or muscle and tendon damage. Thus, a splint was not applied. Her past medical, perinatal, and developmental history was unremarkable, and she had no psychosocial diseases. There was no family history of movement or psychiatric disorders.

On neurological examination, a fine, low amplitude with high frequency (about 10–12 Hz) tremor of her right hand was observed during resting and posturing. The tremulousness scarcely extended above her wrist. Dystonic posture was not noticed, and null point reduced the tremor. When she outstretched her arms, shakiness commenced without latency, and the character of the tremor did not alter (video segment 1). Isometric tremor was also found, but action tremor was not observed during writing. Alleviation by distraction or entrainment of the tremor was not observed. Co-activation sign of antagonist muscles was not detected by palpation. The tremor disappeared during passive movements of the affected limb. Tremor was not detected in any other body parts, including the contralateral arm, leg, and head. No trophic changes, abnormal sudomotor, or vasomotor activities were noted. Craniocervical examination did not uncover any peculiar findings. Motor weakness or sensory impairments, such as allodynia, hypesthesia, hypoalgesia, and hyperpathia, were not found. Range of hand motion was not limited or fixed to a certain posture. Pathologic reflexes were not identified.

Blood tests, including thyroid function and copper and ceruloplasmin levels, were normal. Ophthalmologic evaluation did not disclose any abnormalities. Brain magnetic resonance imaging (MRI) did not unveil any structural abnormality related to tremorgenic areas (Fig. 1a). Nerve conduction studies of the median and ulnar nerves did not show any damage, and somatosensory evoked potential (SEP) evaluation of the same nerves did not disclose enlarged cortical amplitudes (Fig. 1b).

She was not prescribed any medications because she did not complain of functional disability. Her isolated, focal, unilateral tremor persisted for 3 months without progression, and then it spontaneously regressed without further occurrence or any other abnormal movements.

This is an unusual report of focal EPT after physical insult to the affected body part. EPT has many causes, such as drugs, thyrotoxicosis, and emotional lability [2, 3], but no localized form after the trauma has been reported. Tremor frequency, time, and location associations with the traumatic event and its

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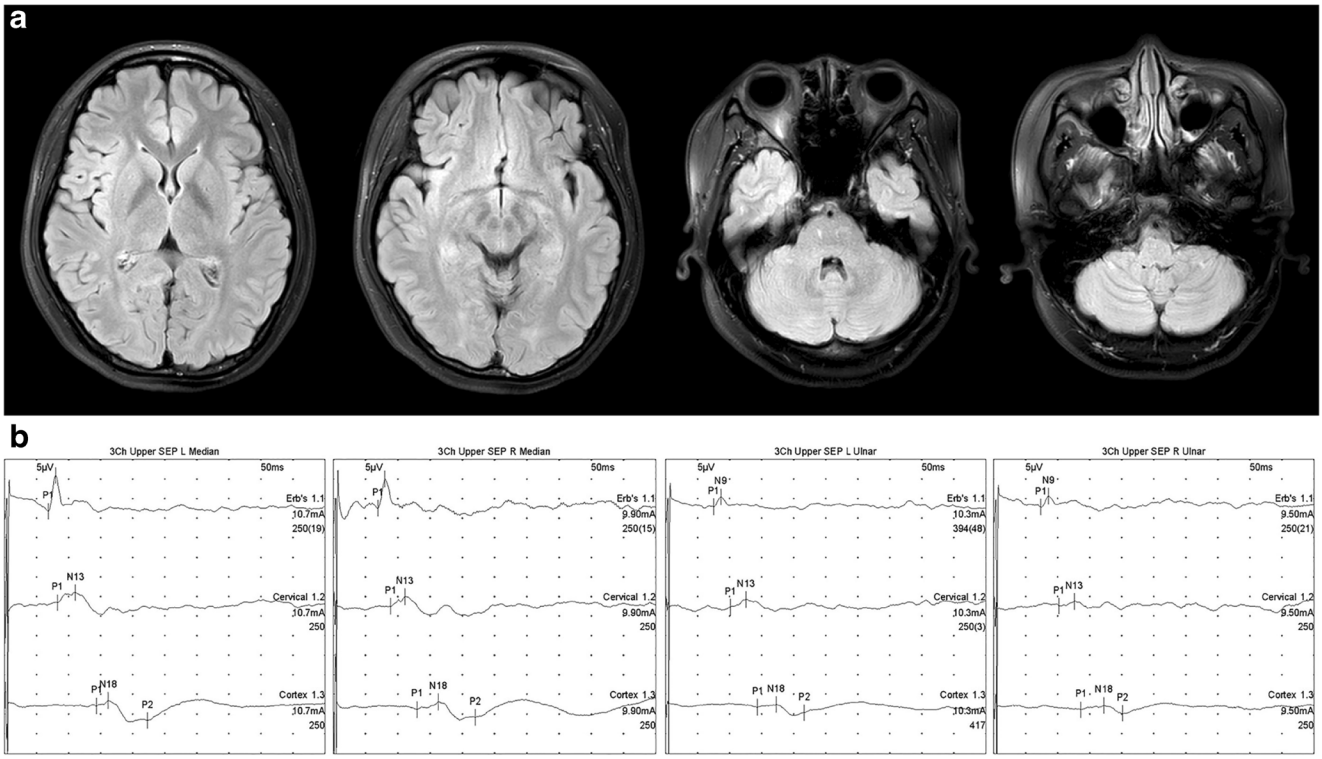

Fig. 1 **a** Fluid-attenuated inversion recovery imaging of her brain did not manifest any abnormalities in the globus pallidum, thalamus, midbrain, cortex, deep nuclei of the cerebellum, or inferior olivary nucleus. **b** Somatosensory evoked potential study did not reveal any giant cortical amplitude that reflects decreased intra-cortical inhibition or disrupted neuraxis

complete spontaneous resolution point toward a presumptive diagnosis of focal EPT. Although the tremor was not investigated electrophysiologically, its frequency visibly exceeded the usual pathologic frequency bandwidth, approximately oscillating above 10 Hz, and matching the 8 to 12 Hz central neurogenic oscillations of physiological tremor [1]. The tremor activation conditions of resting, postural without latency, and isometric contraction also conform to the recognized characteristics of EPT [3]. A recent study emphasized the regression of tremor when the cause is removed and successfully managed for confirmative diagnosis [2]. In this case, the patient was closely observed and serially followed after confirming intact neuraxis by MRI of her brain and neurophysiologic studies. Spontaneous resolution without any medication further substantiates the diagnosis of EPT. To establish a causal relationship, it is imperative that tremor must be temporally and anatomically related to the trauma [4, 5, 7]. In the present case, two facts (tremulousness occurred 2 days after trauma and was confined to the affected body part) strongly indicate a causal relationship between tremor and trauma.

In cases of movement disorders after trauma, psychogenic or functional tremor should be considered and carefully excluded. In this patient, co-activation sign, which is co-contraction of antagonists to cause clonus, was not observed and it can be an important clue in discriminating psychogenic tremor [1, 8]. However, once psychogenic tremor was initiated by a conscious co-contraction, it could persist without requiring much attention [1, 8], and functional tremor cannot be completely ruled out.

This case is distinctive from the acknowledged phenomena of peripherally induced movement disorder (PIMD). It is postulated that trauma alters sensory input and induces central reorganization, generating movement disorders such as tremor [5, 7]. This pathomechanism explains the persistence and spread of movement disorders to adjacent structures and resistance to treatment [4].

In this case, the symptom was short-lived, restricted to the original site, and spontaneously regressed. EPT could be induced by enhancement of the mechanical component by reflexes; upregulated muscle spindle sensitivity intensifies rhythmic afferent activity, leading to greater synchronization of the afferent and enhanced reflex activity to the central system [1]. Mechanical damage to the focal afferent system of the hand lowered the threshold that provoked tremor, but its signals did not prevail long enough to form neuroplasticity in the central oscillators. Therefore, the symptom was short-lived and spontaneously recovered; this change in neurophysiology was the main reason why the patient was

diagnosed with focal EPT rather than PIMD, despite its resemblance.

In conclusion, this case represents another example of post-traumatic focal EPT, although the pathophysiology and association with the traumatic event are still unverified.

Compliance with ethical standards

Ethics The Institutional Review Board at St. Mary's Hospital approved this case report.

Patient consent The patients consented to submission of the case report to the journal.

Conflict of interest The authors have no conflicts of interest or financial support to report.

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