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Postural Tremor after Thalamic Infarction

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A variety of movement disorders (chorea, dystonia and asterixis) might be produced by cerebral infarction [1]; however, isolated, distal postural tremor after thalamic infarction is rarely reported [2–4]. Mechanism and treatment of this type of tremor remain controversial [4, 5]. We report a patient who developed such a tremor after thalamic infarction that responded to propanolol and primidone.

A 77-year-old woman, with a history of high blood pressure and hypercholesterolemia abruptly developed left hemiparesis. CT scan showed a lacunar infarction of the right lateral thalamus (fig. 1). MRI showed a similar image (fig. 2). This lesion impinged to several degrees upon the posterolateral and ventrolateral thalamus nuclei, and may be squeezing the posterior limb of the internal capsule. This syndrome probably results from infarction in the territorial supply of branches arising from thalamogeniculate arteries.

Three months later, the patient developed a progressive, incapacitating postural and intention tremor of the left upper limb that enhanced because of anxiety. This tremor was intermittent, its oscillation varied from 5 to 7 Hz, and showed variable amplitude.

The first-choice drug, propanolol (a total of 120 mg/day, until dose-related side effects intervened), showed to be transitorily effective (4 months) in reducing the tremor amplitude. However, the patient is experiencing long-term dramatic tremor improvement with a combination of propanolol and primidone (250 mg/day).

Recent advances in surgery of movement disorders and new knowledge about the functional organization of the basal ganglia might be used to formulate hypotheses concerning the mechanisms of the movement abnormalities seen in basal ganglia lesions.

It is known that primary disturbances in the putamen or in its pathways produce dystonia; chorea is due to lesions in both the caudate and subthalamic nuclei; bilateral pallidus damage causes parkinsonism, and brain stem lesions could produce blepharospasm and other cranial dystonias. However, the mechanism of tremor is poorly understood. It could be that posterolateral thalamus lesions give rise to deafferented neurons (from cerebellar, mesencephalon and caudate pathways). These deafferented neurons could begin pulsing at a stable frequency [2, 4].

Thalamic infarcts cause several types of tremor, whether rest, or postural tremors. However, some patients may display both types of tremors simultaneously. The frequency of these tremors varies from 8 to 12 Hz, persists, and is intensified during movement and in states



Fig. 1. Cranial CT showing an infarction in the right thalamus having an effect on the internal capsule.

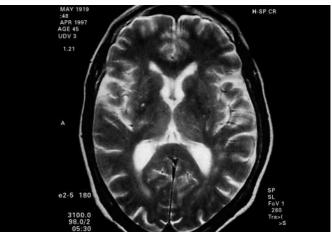


Fig. 2. MRI showing an ischemic lesion in the posterolateral thalamus.

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Accessible online at: http://BioMedNet.com/karger of anxiety. The tremor does not usually manifest itself as early as other components of the syndrome, but starts from a few weeks to some years after the stroke.

In agreement with most reported cases, our patient showed a symptom-free period after stroke for 3 months before the onset of tremor [2–6]. The underlying mechanism producing the delayed neurological outcome (tremor) should be determined by spatial and temporal dynamic mechanisms (i.e. intact neurons, alternative pathways, receptor hypersensitivity, structural and functional integrity, recovery of the injured thalamic cells, remodeling of neural circuits with partial reinnervation, and so on) [2, 4, 5, 7].

Many patients with intrusive tremor after thalamic stroke cannot be treated satisfactorily, and there is no agreement on pharmacological management. Clinical drug trials have concentrated solely on relieving low-frequency rest tremor, which responds unpredictably to levodopa [7]. Some patients with postural tremor have been treated with ceruletide [8]. As yet, most reported cases do not respond satisfactorily to medication, or such conclusions are left out. In our patient, propanolol and primidone have additive therapeutic effects and produce significant improvement in terms of activities of daily living.

Anticholinergics had a small overall effect on tremor of Parkinson's disease. Nevertheless, they can be used in patients with tremor after thalamic stroke whether the rest tremor predominates or is associated with dystonia [3]. Propanolol, or a combination of primidone and propanolol, might have greater benefit in case of intention or postural tremor prevalence.

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