



A case of acute hemiballism and neurogenic pain associated with parietal lobe infarct.

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Abstract :

Acute involuntary movement like hemichorea, hemiballism and athetosis are extremely rare after cortical infarction. A 56-year-old, right-handed, hypertensive and diabetic man presented as acute onset of involuntary movements of his right arm. Neurological examination revealed, involuntary, brisk, high-amplitude, very fast, unpredictable movements of the proximal segment of the left upper extremity and mild apraxia of right hand, else everything was normal. He also had unbearable burning pain deep in the right arm. Involuntary movements settled with Haloperidol. MRI scan revealed acute infarct in the right anterior parietal cortex while basal ganglia structures were normal

Keyword :

acute hemiballism neurogenic pain parietal lobe infarct

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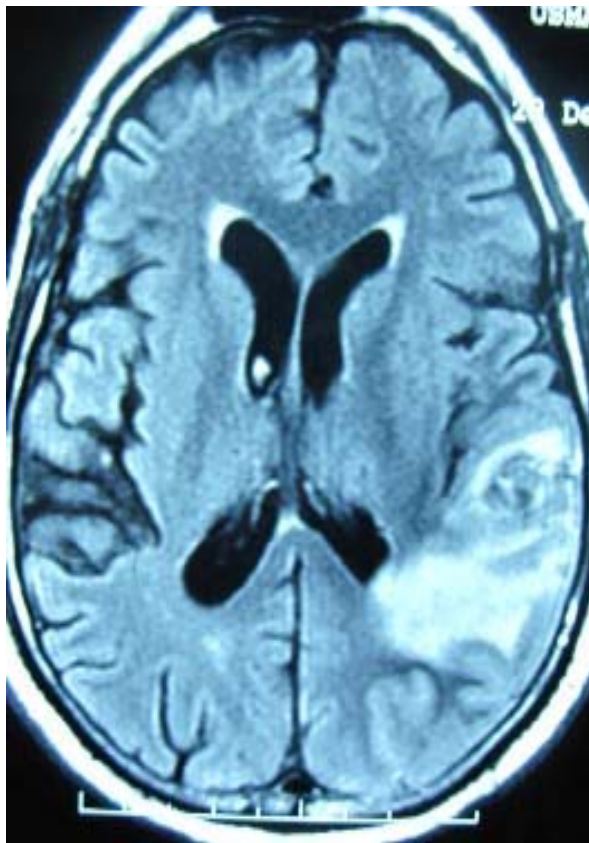
lism and neurogenic pain associated with parietal lobe infarct.
Acute involuntary movement like hemichorea, hemiballism and athetosis are usually observed in basal ganglia dysfunction. However they appear to be extremely rare after cortical infarction.

Case Report.

A 56-year-old, right-handed, hypertensive and diabetic man was presented to us as acute onset of involuntary movements of his right arm. On examination, his blood pressure was 160/100mmHg and the findings of general physical examination were normal. Neurological examination revealed, involuntary, brisk, high-amplitude, very fast, unpredictable movements of the proximal segment of the left upper extremity. The patient was alert and oriented, and there was mild apraxia of right hand. No motor weakness or sensory deficits were noted. On 2nd day he complained of an unbearable burning pain deep in the right arm. The abnormal movements subsided during sleep.

The painful symptoms disappeared spontaneously after 3 days, while the involuntary movements were favorably managed with haloperidol (2 mg, 2 times per day), and subsided after 3 weeks. Computed Tomography scan (Figure 1) performed on day 1 and Magnetic resonance imaging (Figure 2) performed 5 days after admission showed an acute infarction of the right anterior parietal cortex, extending to the upper posterior temporal lobe and the adjacent white matter and involving mainly the territory of the anterior parietal artery. The basal ganglia, thalamus, and subthalamic region were intact.





Hemiballismus is an extremely rare manifestation in acute stroke. Its differential diagnosis primarily involves the more common pseudochooreoathetosis, which results from severe impairment of proprioceptive sensation and appears as slow, snake-like, involuntary movements principally confined to the distal extremity. Our present understanding of basal ganglia function considers a balance between the direct pathway (cortex-caudate-internal pallidum, accounting for dyskinesias) and the indirect pathway (cortex-caudate-external pallidum-subthalamic nucleus-internal pallidum, accounting for akinesia), which modulate the glutamatergic activator thalamocortical retroactive pathway. Lesions that disrupt this balance, which are classically located in the subthalamic nucleus but have also been described in the subthalamic-pallidal fibers, striatopallidal connections, basal ganglia, thalamus, corona radiata, and the frontal

or parietal lobe, may induce hemiballismus. Under normal physiologic conditions, the cerebral cortex provides excitatory stimuli to the basal ganglia; thus, lesions involving corticostriatal fibers may disrupt the balance of basal ganglia circuits. Acute limb pain in stroke is a rare phenomenon and has been described in subjects with hemispheric, thalamic, and, less frequently, brainstem lesions. It must be differentiated from the more common acute paresthesias, which are not accompanied by definite and intense pain, from delayed central pain occurring weeks or months after a thalamic or parietal lesion, which is often seen after improvement of the sensory deficits, and from "acute hemiconcern," a right anterior parietotemporal syndrome without painful symptoms. The interruption of spinothalamic parietal projections is supposed to lead to spontaneous pain. Pathophysiologically, a disinhibition of the phylogenetically old pain pathway that passes through the intralaminar thalamic nuclei and projects to the anterior cingulate cortex and an imbalance of the putative modulatory action of the lemniscal system on the pain pathways have been postulated. Another theory points to the hyperactivity of the deafferented parietal neurons, especially in the secondary somatosensory area, leading to a spontaneous painful sensation as a result of their disconnection. In conclusion, our patient showed acute limb pain associated with ipsilateral hemiballismus following a contralateral hemispheric infarction. This syndrome may result from a diaschisis: the lesion interrupting the pathways between the hemisphere and thalamus/basal ganglia caused disconnection of the parietal cortex

from the thalamus, leading to acute central pain, and the striatum, generating the movement disorder. We suggest that, in contrast to isolated hemiballismus (which is not painful), the simultaneous occurrence of acute limb pain is specific to a lesion in the territory of the anterior parietal artery.

References

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