Neurogenic Pain and Abnormal Movements Contralateral to an Anterior Parietal Artery Stroke

Andrea O. Rossetti, MD; Joseph A. Ghika, MD; François Vingerhoets, MD; Jan Novy, MD; Julien Bogousslavsky, MD

Background: Unlike delayed pain syndrome, acute central pain is a very rare symptom in acute stroke. In addition, the incidence of hemiballismus in acute cerebrovascular diseases is less than 1%. To our knowledge, the association of these 2 clinical conditions has not been previously described.

Patient and Methods: After observing one patient with hemiballismus accompanied by ipsilateral acute limb pain at stroke onset, we retrospectively examined more than 4000 patients in the Lausanne Stroke Registry for hemiballismus-hemichorea occurring together with acute ipsilateral pain.

Results: Of the 29 subjects with hemiballismus-hemichorea, the observed patient was the only one to have acute pain at the onset of stroke. Magnetic resonance imaging showed acute infarction in the territory of the right anterior parietal artery, whereas the basal ganglia, thalamus, and subthalamic region were intact.

Conclusions: The syndrome of acute limb pain associated with hemiballismus may result from disconnection of the parietal lobe from deeper structures. In contrast with isolated hemiballismus, we suggest that the simultaneous occurrence of this movement disorder with ipsilateral pain is specific for an anterior parietal artery stroke.

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anticoagulants, not regarding the chronic subdural hematoma as a contraindication. He was then referred to another hospital for rehabilitation.

STROKE REGISTRY REVIEW

After observing the patient, we retrospectively reviewed the history of more than 4000 patients in the Lausanne Stroke Registry, looking for hemiballismus-hemichorea associated with acute pain symptoms. The registry considers this movement disorder when abnormal movements are involuntary, brisk, fast, and unpredictable, with some element of rotation, occurring mainly in the proximal segment of the limbs, and when these movements are increased by attempts to move a limb. It is differentiated from other movement disorders that occur during acute stroke, such as pseudochoreoathetosis, dystonia, tremor, asterixis, and myoclonus. The radiologic investigations consisted of computed tomographic scans and/or magnetic resonance imaging (after 1989) of the brain.

RESULTS

Only 29 patients with hemiballismus-hemichorea were identified and, of these, only 2 showed an association of abnormal movements with pain. A female patient described delayed painful paresthesias in her affected hand; unfortunately, the computed tomographic scan performed during the acute phase (1981) failed to show any lesion. The patient whom we described in the “Methods” section was the only patient with a hemispheric lesion and contralateral acute pain associated with hemiballismus.

COMMENT

Our patient had the unusual association of acute pain and hemiballismus secondary to an anterior parietal infarction. Since, to the best of our knowledge, movement disorders have not been described following ipsilateral subdural hematoma, it is unlikely that the latter accounted for the clinical signs and symptoms in our patient. Furthermore, the possibility of embolic involvement of the basal ganglia was ruled out by magnetic resonance imaging.

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 spinothalamoparietal 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 deafferentiated parietal neurons, especially in the secondary somatosensory area, leading to a spontaneous painful sensation as a result of their disconnection. A functional study underscored the importance of the ipsilateral insular and parietal areas, although this work focused on delayed allodynic pain. In a review study, all 6 patients...
with spontaneous pain following a hemispheric stroke had right lesions, mainly in the parietal lobe, suggesting a role of the “minor” hemisphere. Our patient showed ischemia involving the right thalamoparietal fibers as well as the primary somatosensory cortex. The secondary somatosensory cortex was left intact, and thus disconnected from deeper structures. The pain was described as burning, as in previous reported instances of acute pain,5.7.9,10 and lasted for only a short time (2 days), which was in agreement with some of the reports.5.9

Hemiballismus or, since the clinical signs and symptoms may overlap in some instances, hemiballismus-hemichorea, is an extremely rare manifestation in acute stroke (Lausanne Stroke Registry prevalence, 0.4%). Its differential diagnosis primarily involves the more common pseudochoreoathetosis, which results from severe impairment of proprioceptive sensation and appears as slow, snake-like, involuntary movements principally confined to the distal extremity.10,17 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.18 Lesions that disrupt this balance, which are classically located in the subthalamic nucleus19 but have also been described in the subthalamic-pallidal fibers, striatopallidal connections, basal ganglia, thalamus,19-21 corona radiata,22 and the frontal23 or parietal23,24 lobe, may induce hemiballismus. More recently, a report stressed the occurrence of this movement disorder after lesions of the parietal cortex or adjacent white matter pointed to the role of the interruption of the parietotraitional pathways,22 and this was corroborated by the finding of reduced perfusion of the radiologically unaffected striatum in some of these cases.23,24 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. Compared with conditions that result from destruction of the subthalamic nucleus,4,25 our patient’s hemiballismus was milder and easier to treat with neuroleptics. This feature may reflect a slightly different pathophysiologic mechanism.

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 (Figure 1C) 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.

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Corresponding author: Andrea O. Rossetti, MD, Service de Neurologie, BH 07, Centre Hospitaux Universitaire Vaudois, 1011-Lausanne, Switzerland (e-mail: andrea.rosetti@chuv.hospvd.ch).

REFERENCES


