Commentary

Hemichorea-hemiballism is thought to be produced by imbalance in complex neuronal cortico-basal ganglia circuit(s), involving the striatum, globus pallidus and subthalamic nucleus, thalamus and cortex.[1] Vascular lesions (ischemic or hemorrhagic) are among the most common causes of hemichorea. Other causes are metabolic, including hyperglycemia, autoimmune, including antiphospholipid syndrome and Sydenham's chorea and space occupying or destructive lesions, including primary and metastatic tumors, vascular malformations and infections, including tuberculomas, toxoplasmosis, and cysticercosis. Traditionally, the subthalamic nucleus is considered the most common site of involvement; however, lesions in the striatum seem to be more common and they may also occur in the globus pallidus and thalamus.[2] Given the interaction of the basal ganglia with at least the prefrontal cortex, it would be expected that the cortex could be a site of involvement in some patients.

In this issue, the paper by Rana *et al*.[3] reminds us that (a) hemichorea may appear together with dystonia and (b) it can be triggered by space occupying lesions affecting the frontal lobe, making neuroimaging necessary in such patients. Direct (pressure) or indirect (vascular compromise) effects on basal ganglia could be the cause. However, an effect on the frontal cortex may offer an additional explanation.

Despite early reports about hemichorea after frontal and/or parietal stroke or subdural hematoma,[4-6] the concept of "cortical" hemichorea due to any cause (vascular, tumor) received adequate attention only recently and may account for up to 30% of hemichorea-hemiballism cases.[7] In the stroke series of Chung *et al*., cortical stroke was responsible for 22% of hemichorea cases and sites of involvement included frontal, parietal, insular, and temporal cortex.[2] It has been proposed that, in such patients, dysregulation of the cortico-striatal and cortico-subthalamic pathways affects the basal ganglia output, resulting in hemichorea.[8]

Regardless of the mechanism, imaging of the brain, preferably by magnetic resonance imaging (MRI) should be provided in hemichorea patients, in order to identify possible space occupying and potentially treatable lesions.

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