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Tremor and Other Hyperkinetic Movements

Letters

Cortical Stroke and Hemichorea

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We congratulate the authors for their interesting case published in the 2016 August issue of the journal.[1] We read the article entitled ''Delayed Hemichorea Following Temporal–Occipital Lobe Infarction'' with interest and would like to make some observations and comments pertaining to the case.

Significance of neuroimaging

The authors briefly discussed the significance of neuroimaging in vascular hemichorea; however, the stroke mechanism remains unclear. Brain magnetic resonance imaging (MRI) performed during hospitalization demonstrated restricted diffusion in the right temporal lobe and a punctate focus of ischemia in the occipital lobe. Focal occlusion in the P2 segment of the right posterior cerebral artery (PCA) was identified on MR angiogram. It is well described that the thalamogeniculate branches of the PCA provide vascular supply to the thalamus and temporal lobes. Hemichorea and hemiballism are primarily disorders of the subthalamic nucleus (STN).[2] As mentioned by the authors, ischemia of the thalamus or STN could have been a cause for hemichorea in their patient; however, no further diagnostic testing was performed to corroborate this proposed mechanism.

In the modern era, neuroimaging has proven its mettle, especially in the stroke community.[3,4] Perfusion abnormalities depict hypoperfusion in ischemic territories that are at risk of infarction if revascularization is not restored. Computed tomography (CT) or MR-perfusion, including single-photon emission CT (SPECT) studies, could reveal hypoperfused or ischemic territories, such as the STN or thalamus in this case, which could result in extrapyramidal symptoms including chorea and dystonia. Vascular ischemia is a dynamic process that can lead to the delayed onset of movement disorders. We suggest that follow-up brain MRI would be fruitful in these cases to evaluate any extension or evolution of infarction.

Significance of pathophysiology

The authors briefly mentioned the interruption of intercortical or cortical-ganglionic connections as a possible mechanism for hemichorea in their patient. However, the theory of interneuronal connections by Hwang et al demonstrated intact perfusion status of subcortical gray matter on reviewing the MRI/SPECT neuroimaging patterns in patients with hemiballism/chorea.[5] They demonstrated perfusion abnormalities restricted to the parietal and especially premotor and supplementary motor areas of frontal cortices, while no perfusion abnormalities were noticed in the thalamus or basal ganglia. They suggested derangement of the parieto-frontal network as a likely cause for vascular hemichorea in their cohort. However, the mechanisms for hemichorea secondary to stroke located in temporal-occipital locations remain unclear. We suggest the inclusion of perfusion sequences and follow-up neuroimaging for thorough evaluation of these patients.

Significance of medical history and diagnostic workup

The clinical history and diagnostic workup remain crucial to accept or refute the myriad of differential diagnoses in complex cases. Various causes of acquired chorea include drug-induced, vascular, autoimmune, infectious, metabolic, paraneoplastic, and other miscellaneous etiologies. Vascular etiology is the second-most common cause of acquired chorea.[6] The vascular causes to be considered in the current

case of acquired chorea include ischemic stroke and the atypical variant of posterior reversible encephalopathy syndrome.[7] However, occlusion of the P2 segment on intracranial angiogram and an absence of fluid-attenuated inversion recovery changes on brain MRI confirm ischemic stroke as the likely etiology.

The patient had a remote history of sarcoidosis and was not on active immunosuppressive therapy. Autoimmune disorders are known to co-exist with delayed manifestations and tend to present with varied neurological symptoms including movement disorders. Choreiform movements are associated with various autoimmune disorders such as systemic lupus erythematosus, antiphospholipid antibody syndrome, Sjogren syndrome, and neurosarcoidosis.[8] Neurosarcoidosis infrequently involves subcortical gray matter mimicking different neuropathological disorders on imaging, and manifests with varied movement disorders including hemichoreoathetosis.[9,10] Interestingly, the presence of these autoimmune disorders tends to increase the risk for ischemic stroke. Thorough assessment of clinical history, specifically for prior vascular thromboembolic events and miscarriages, and specific laboratory testing including antinuclear antibodies, anti-double-stranded DNA, lupus anticoagulant, anticardiolipin antibody, and β2 glycoprotein antibodies should be evaluated. Detailed clinical investigation remains paramount in cases where clinical justification for the proposed diagnosis is lacking.

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