

HEMICHOREA – A RARE PRESENTATION OF ACUTE STROKE

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Abstract : A rare case of hemichorea occurring in an adult male after acute stroke is presented. The incidence and precise lesion location of post stroke hemichorea remains unclear. A 32 year old male presented with slurring of speech, involuntary movement of right upper limb. CT scan of brain showed infarct in left insular cortex and left external capsule. Hyperkinetic movement disorders like hemichorea after stroke are uncommon, with a prevalence of less than 1% in previous hospital based study.

INTRODUCTION

Stroke which was previously considered a “curse” and was associated with “hopelessness”, has entered in to the era of hope and prevention is the key; thanks to the recent advances in stroke management. Early recognition and reporting of such rarest presentation, are crucial.

Hyperkinetic movement disorders like hemichorea after stroke are uncommon, with a prevalence of less than 1% in previous hospital based study¹. We here report this rare presentation of stroke produced by infarct in left insular cortex and left external capsule, though hemichorea is most often due to lenticular lesion followed by subthalamic and cortical lesion².

CASE REPORT

A 32 year old male presented to the stroke unit of the hospital with slurring of speech, involuntary movement of right upper limb. On examination he was conscious, oriented, but anxious. He had mild shoulder pain due to movement of that limb. General examination was within normal limits. He had no history of fever, any drug intake like antiemetics or antipsychotics. He was normotensive and nondiabetic. His blood sugar random was 120 mg/dl. His blood pressure at the time of admission was 120/70 mmhg in left upper limb supine position. His thyroid profile was normal. On CNS examination apart from slurring of speech and abnormal movement of right upper limb, all were unremarkable. Motor examination demonstrated choreiform movements in right upper limb, primarily visible during motor tasks such as eating, writing (unable to hold a pen) etc. Coordination was affected by hemichorea on the right side but was normal on left. He had no weakness. Sensory examination results were normal. Gait was normal. Abnormal movement of the limb was just like typical hemichorea. We kept the possibilities of drug induced chorea, although there was no history of any drug intake.

Patient underwent plain and post contrast CT head. CT scan showed acute infarct in left insular cortex and left external capsule (Fig.)

On the basis of CT scan findings and clinical presentation, a diagnosis of hemichorea after stroke was made. We treated this patient with haloperidol and aspirin 300 mg. Patient showed improvement. Patient was doing well on a follow up visits after discharge from the hospital.

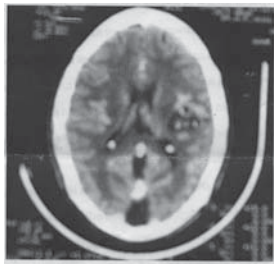


Fig. : C.T. Brain

DISCUSSION

Hemichorea is defined as involuntary, hyperkinetic disorder consisting of sudden irregular, purposeless, and random movements involving one half of the body. Although the movements are purposeless, the patients may incorporate them in to a deliberate act, as if to make them less noticeable. When superimposed on voluntary actions they may assume an exaggerated and bizarre character. In moments when the involuntary movements are absent or briefly held in abeyance, volitional movements end; may be excessively quick and poorly sustained⁷. It is the rarest presentation after acute stroke. The largest published study showed the incidence of post stroke hemichorea as 0.54%². Although hemichorea has been suggested to be the most common form of post stroke involuntary movements¹, the incidence was lower than that of post stroke asterixis (1.9%)³. In this patient hemichorea developed on the same day of stroke. That means post stroke hemichorea results from the stroke induced acute functional deregulation of the basal ganglia motor circuit rather than from restorative neuroplasticity⁴. The sub-thalamus⁵ and striatum² has been recognized as a typical site responsible for hemichorea probably because stroke involves these regions more frequently. The pathogenic mechanism of hemichorea caused by contralateral striatum would be the interruption of GABA transmission from striatum to the globus pallidus externa (GPe), which may increase the GPe neuronal activity and inhibit the subthalamic nucleus⁶. The inhibition of the subthalamic nucleus⁶. The inhibition of the subthalamic nucleus would result in the loss of its control upon the globus pallidus interna (GPI) neurons, which ultimately leads to a dysinhibition of the motor thalamus⁷. In these patients infarcts are limited to insular cortex and external capsule. The pathogenic mechanism of hemichorea in this patient remains unexplained. Unfortunately, we only considered the relationship between clinical feature and radiological findings. Further studies using functional or perfusion images are warranted to elucidate the pathophysiology of hemichorea especially in those involving the uncommon area.

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