

Case Report

Acute hemichorea associated with ipsilateral chronic subdural hematoma

Tarun Kumar Ralot¹, Umesh Chahar²*, Jainendra Kumar Sharma², Chinmay Vishwanath Hegde², Raghavendra G.²

¹Department of Neurology, RNT Medical College, Udaipur, Rajasthan, India
²Department of General Medicine, RNT Medical College, Udaipur, Rajasthan, India

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*Correspondence:
Dr. Umesh Chahar,
E-mail: drumeshchahar@gmail.com

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ABSTRACT

Here we are describing left-sided hemichorea in a 71-year-old female which developed within 3 days without any history of weakness, unconsciousness, fever, headache, vomiting. She had a history of head trauma 5 year back. No abnormality was detected in routine blood investigations. Computed tomography revealed a left chronic subdural hematoma. Neurosurgical intervention in form of left temporal “burr hole” drainage was performed and the patient’s involuntary movements improved in the postoperative period.

Keywords: Hemichorea, Subdural hematoma, Burr hole, Ipsilateral

INTRODUCTION

Hemichorea is mostly because of structural lesion in brain but the causes vary with age, like stroke is common in older age groups and rheumatic chorea is still common in children’s in developing countries. Post-traumatic chorea can present acutely or with significant delay from the time of initial injury, with the most effective treatment being intervention by surgery. The etiology of chorea varies widely but chorea associated with chronic subdural hematoma (SDH) is rare and very few cases have been reported so far.¹⁻⁴ Here we are reporting a case with acute hemichoreiform movements associated with ipsilateral chronic SDH.

CASE REPORT

A 71-year elderly female presented with complaint of involuntary curling like movements in left upper and lower extremity which started suddenly 3 days ago.

These movements were lasting 25-28 seconds and recurring in every 40-50 seconds. No involuntary movements were present in right extremity & face. Movements were causing significant interference with her routine life but could be suppressed partially. She didn’t complaint of any weakness or sensory disturbance in extremity and trunk. She had no history of headache, vomiting, vertigo, unconscious. No complaint of bowel, bladder involvement. No history of any prior vaccination. She had history of head trauma 5 years ago in the form of fall from bed but there was no loss of consciousness or weakness at that time and they did not take any medical advice. She denied any history of alcohol use or any drug treatment history.

Family history was negative for any similar illness. Her examination revealed blood pressure 150/78 mm of Hg, pulse rate 76 bpm and was conscious, orientated, and cooperative. Neurological examination revealed involuntary, irregular, semi-purposeful and non-rhythmic
choreiform movements of the left upper and lower extremities. No involuntary movements were detected in right upper and lower extremities and face. Her muscle strength was 5/5 at all joints. Deep tendon reflexes were normal. Plantar reflex were flexor in both extremities. There was no sensory disturbance in any extremity, trunk or face. Cerebellar sign examination was normal. There was no neck stiffness and cranial nerves were intact. Routine blood test like hemoglobin, total blood count, erythrocyte sedimentation rate and platelets were normal. Other test like thyroid function test blood sugar level, creatinine, electrolytes prothrombin time, total protein was within normal limits.

Figure 1: Non contrast CT scan of head revealed extra axial crescent shape hypodensity is seen in left fronto parieto-occipital region, (maximum thickness12mm marked with red arrow) suggestive of chronic sub dural hemorrhage.

Figure 2: MRI T2W image of brain revealed csf intensity subdural hygroma is seen in left cerebral convexity measuring about 11.5 mm in thickness but no lesion in the basal ganglia, thalamus, or brainstem.

Test’s antinuclear antibody test, HIV, HbsAg and anti-HCV did not reveal any abnormality. Based on clinical history, neurologic examination and neuroimaging a diagnosis of chronic SDH induced hemichorea due to trauma was considered and burr hole drainage was done by the neurosurgeon. Approximately 60 ml of hematoma was evacuated through a single burr hole in left parietal bone. The choreiform movements became markedly less after hematoma evacuation. She was discharged ambulatory 5 days after the operation. Resolved completely during follow up.

DISCUSSION

Chorea refers to involuntary arrhythmic movements of a forcible, rapid, jerky type. Although the movements are purposeless, the patient may incorporate them into a deliberate act. When superimposed on voluntary actions, they may assume an exaggerated and bizarre character. Usually the movements are discrete. Hemichorea may be limited to one side of the body (hemichorea). When the involuntary movements involve proximal limb muscles and are of wide range and flinging in nature, the condition is called hemiballismus. In most cases, the onset of chorea is slow and insidious. An abrupt or subacute onset is more typical of many of the symptomatic causes of chorea, such as Sydenham chorea, hyperthyroidism, cerebral infarcts, and neuroleptic drugs, systemic lupus erythematosus (SLE) and other autoimmune choreas. Symptoms isolated to one side of the body suggest a structural lesion in the contralateral basal ganglia.

The precise anatomic basis of chorea is often uncertain or at least inconsistent. In Huntington chorea, there are obvious lesions in the caudate nucleus and putamen. The localization of lesions in Sydenham chorea and other chorea diseases has not been determined beyond a generalized disturbance in the striatum, which is evident on some imaging studies. Hemichorea is a relatively uncommon movement disorder that usually follows an ischemic or hemorrhagic lesion in the contralateral caudate nucleus or putamen, corona radiata, Lesions outside these structures may also be associated with hemichorea. Among the reported cases of choreiform movements associated with chronic SDH there were three bilateral and five unilateral hematoma cases. Generalized choreiform movements were observed in three bilateral and in three unilateral cases. Ipsilateral hemichorea was observed in two cases of unilateral SDH. In above mentioned cases, choreiform movements subsided immediately, or were markedly reduced and resolved completely after evacuation of hematoma. Our patient has a small amount of left sided SDH that probably caused dysfunction of the right cerebral hemisphere without manifesting localizing sign in left cerebral hemisphere. Other postulated mechanism of chorea in chronic SDH are 1) the mean hemispheric cerebral blood flow decreased on both side with hematoma 2) the cerebral blood flow reduction was always greater in the putamen and thalamus than in the cortex 3) reduced cerebral blood flow occurred even in patient with only headache and minimal or no brain shift on CT scan (hematoma volume 20-70 ml).
CONCLUSION

This case shows that SDH can produce hemichorea without any localizing sign in basal ganglia or any pre-existing lesion. Further studies are needed to exactly describe the mechanism of these presentation.

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