

Reversible parkinsonism secondary to chronic subdural hematoma

Wajid Nazir Wani, Kuljeet Singh Anand

Department of Neurosurgery, Post Graduate Institute of Medical Education and Research and Dr. Ram Manohar Lohia Hospital, New Delhi, India

ABSTRACT

Secondary parkinsonism is attributable to a wide variety of causes including supratentorial mass lesions. While tumors are known to present with parkinsonism, chronic subdural hematoma is rarely seen presenting as rapidly deteriorating parkinsonian features with complete disappearance following evacuation of hematoma. The authors present two such patients—70- and 78-year-old males who presented with sudden onset of parkinsonism features. Both failed to recollect any significant head injury. Imaging diagnosed the presence of chronic subdural hematomas, being unilateral in one and bilateral in other. Surgical evacuation resulted in complete resolution of parkinsonian symptoms. These cases reinforce earlier studies for chronic subdural hematoma to be one of the causes of reversible parkinsonism apparently from distortion of basal ganglia mechanically and bringing changes in dopaminergic function, harming the susceptible aging brain.

Key words: Craniocerebral trauma, hematoma, parkinson, surgery

INTRODUCTION

The common causes of secondary parkinsonism are drug-induced parkinsonism and parkinsonism-related with structural lesions on the basal ganglia circuits such as brain tumor or infarct.^[1] New onset parkinsonism and worsening of pre-existing Parkinson's disease are both uncommon but recognized presenting features of chronic subdural hematoma.^[2] Parkinsonism suggested by extrapyramidal rigidity, bradykinesia, and resting tremor are uncommon but recognized presenting features of chronic subdural hematoma.^[3]

Secondary parkinsonism is attributable to a wide variety of causes including intracranial mass lesions. While tumors are known to present with parkinsonism, chronic subdural hematoma is rarely seen presenting with rapidly worsening parkinsonian features and complete disappearance after evacuation.

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CASE REPORTS

Case 1

A 78-year-old man presented with slowness in performing activities of daily living with right-sided limbs since 15 days and stiffness of right lower limb for 7 days. There was no history of tremulousness of limbs or falls and no preceding history of cranial trauma, transient ischemic attacks, or stroke.

General physical and systemic examination was unremarkable. Neurological examination revealed normal higher mental functions and cranial nerves. Motor system revealed no tremors with normal power. Bradykinesia was present in right upper limb on finger-nose test, rapid alternating movements and on finger tapping. Mild rigidity was present in right-sided limbs. On gait examination, decreased arm swing was seen. Deep tendon reflexes were brisk and planters were flexor.

Routine hematological and biochemical investigations were normal. MRI brain [Figure 1] showed a large extra-axial concavo-convex collection in left frontotemporal region of altered signal intensity appearing predominantly hyperintense on both T1WI and T2WI with hypointense linear medial margin and few interspersed hypointense areas anteriorly suggestive of chronic subdural hematoma. The hematoma measured 3 cm in thickness and caused

Address for correspondence: Dr. Wajid Nazir Wani, Department of Neurosurgery, Room No 403, Doctors Quarter, Dr. Ram Manohar Lohia Hospital Campus, Baba Khadak Singh Marg, New Delhi - 110 001, India. E-mail: dr_wajidnazir@rediffmail.com

mass effect upon underlying brain parenchyma with effacement of ipsilateral ventricle. Subfalcine herniation and gross midline shift to right side were also seen.

Emergency left frontal and parietal burr holes were made under local anesthesia and evacuation of chronic subdural hematoma was done which resulted in reversal of right hemi-parkinsonism within hospital stay of 7 days.

Case 2

A 70-year-old nondiabetic and normotensive man developed gait disturbances gradually since 1 month in the form of postural instability with tendency to fall forward which progressed to bradykinesia with festinant gait for 1 week. There was no history of trauma.

General physical and systemic examination was unremarkable. Neurological examination revealed normal higher mental functions and cranial nerves. Motor system revealed marked bradykinesia of both sides with mild rigidity of upper limbs and no tremors. Patient had stooped posture and festinant gait with initial freezing and instability on pull test. Deep tendon reflexes were brisk and planters were flexor. Routine hematological and biochemical investigations were within normal range. Computed tomography scan of head [Figure 2] was done which revealed bilateral frontoparietal chronic subdural hematomas with marked compression of cerebral hemispheres. Bilateral parietal burr holes were made and evacuation of hematomas was done which resulted in complete disappearance of hypokinetic parkinsonism syndrome.

DISCUSSION

The first report of a subdural hematoma producing hemiparkinsonism was by Sandyk in 1968 as an occasional finding during a stereotactic surgery.^[4] Most of the cases have been detected in the elderly in the age groups of 60-80 years.^[5] Majority of cases are due to unilateral subdural hematoma though bilateral and interhemispheric chronic subdural hematomas have also been reported. Deterioration of pre-existing parkinsonism has also been reported as a result of development of subdural hematoma.^[6] The parkinsonian features seen in these cases are possibly due to mechanical compression and distortion of the basal ganglia regions with associated changes in dopaminergic function harming the susceptible aging brain.^[7] Subdural hematomas can present with a wide variety of symptoms. An atypical presentation can be movement disorders. The key feature is that the history of onset is more rapid than with neurological conditions such as Parkinson's disease.^[8] Most elderly patients with CSH, who subacutely developed parkinsonism, recovered partially or completely following spontaneous resolution of the hematomas.^[9]

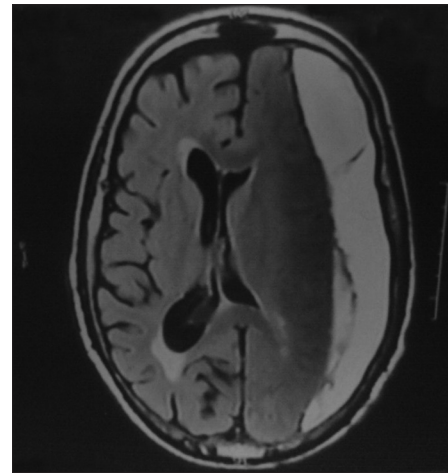


Figure 1: MRI brain showing a large extra-axial concavo-convex collection in left frontotemporal region of altered signal intensity appearing predominantly hyperintense on both T1WI and T2WI

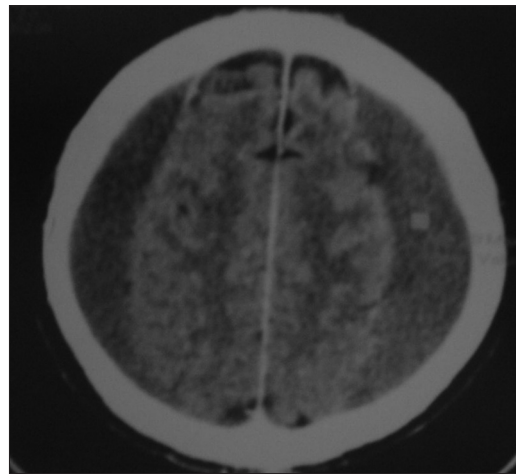


Figure 2: CT scan of head revealing bilateral frontoparietal chronic subdural hematomas with marked compression of cerebral hemispheres

CONCLUSION

The mechanism leading to parkinsonism in patients with a subdural hematoma is not well understood. It has been suggested that direct compression on the basal ganglia by space-occupying lesions can cause the decreased number of dopaminergic receptors in the striatum which explains parkinsonism secondary to brain tumor.^[10] Furthermore, the mass effect that compresses the midbrain and thus interferes with nigro-striatal dopaminergic transmission may also induce parkinsonism. In those mechanisms, the levodopa-responsiveness may be depending on the region of compression and the involvement in the midbrain tend to be more responsive rather than basal ganglia compression.^[11] Surgical evacuation of hematoma results in prompt resolution of parkinsonism suggesting a cause and effect relation.^[7] Elderly patients presenting with akinetic rigid parkinsonism without tremors with or without history of trivial head trauma should be

subjected to neuroimaging to exclude chronic subdural hematoma.

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