Case Report

Cerebellar Stroke-manifesting as Mania

Venkatesan Jagadesan, Kannapiran R. Thiruvengadam, Rengarajalu Muralidharan

ABSTRACT

Secondary mania resulting from cerebral Cortex are described commonly. But secondary mania produced by cerebellar lesions are relatively uncommon. This case report describes a patient who developed cerebellar stoke and manic features simultaneously. 28 years old male developed giddiness and projectile vomiting. Then he would lie down for about an hour only to find that he could not walk. He became quarrelsome. His Psycho motor activities and speech were increased. He was euphoric and was expressing grandiose ideas. Bender Gestalt Test showed signs of organicity. Score in Young mania relating scale was 32; productivity was low in Rorschach. Neurological examination revealed left cerebellar signs like ataxia and slurring of speech. Computed tomography of brain showed left cerebellar infarct. Relationship between Psychiatric manifestations and cerebellar lesion are discussed.

Key words: *Cerebellar lesions, cerebellar stroke, mania*

INTRODUCTION

Cerebellum accounts for over half the brain's population of neurons. The dominant motoric view of cerebellum has now changed to include its role in all cognitive functions and behavior as for the cerebral cortex. Lesions of the cerebral cortex producing secondary mania are commonly described, but cerebellar lesion producing mania is relatively uncommon. Present report deals with a patient who developed left cerebellar stroke. He had simultaneous onset and persistence of both cerebellar and manic features.

CASE REPORT

This was a case report of a 28-year-old unmarried man from a rural low middle-income group, farmer by occupation presented to our out-patient department (OPD) on 05.05.2008 with acute excitement and inability to walk. Although working in the fields the previous day, he had a feeling of giddiness and had projectile vomiting. He was feeling dazed and uneasy and lied down for next 60 min. Subsequently, he noticed that he could not walk. He noticed a change in his speech. He started talking excessively and was quarrelling with others. He could not sleep on the day of onset of his complaints. Throughout the night he was disturbing others and demanding things. He was brought to the OPD next morning.

On examination, he was noted to be asthenic built. His psychomotor activity was accelerated. He was talking spontaneously and excessively to everyone as if they were familiar to him. His mood was euphoric with irritability at times. He was expressing grandiose ideas that he has a lot of power and can even beat 500 men. Hallucinations could not be elicited. He was distracted by events happening around him. He was well oriented. His recent memory was normal and he described his onset of illness very lucidly. He lacked insight to his mental illness, but accepted his walking difficulty and said he wants medical attention.

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Department of Psychiatry, Thanjavur Medical College, Thanjavur, Tamil Nadu, India

Address for correspondence: Dr. Kannapiran R. Thiruvengadam
Department of Psychiatry, Government Thiruvarur Medical College, Thiruvarur, Tamil Nadu, India. E-mail: rtkannapiran@yahoo.com

Neurological evaluation showed left sided cerebellar signs. He was ataxic and could walk only with the support. Finger nose incoordination was present in left upper limb. There was mild slurring of speech, but the classical speech of cerebellar disorder was absent. Cranial nerves and fundii were normal. No nystagmus was noted. Sensory system and other motor functions were normal. Cortical lobar functions were normal. His blood pressure, pulse and other systems were normal. There was no Kayser-Fleischer ring.

He is born to non-consanguineous parents. He is the first of three siblings. There was no positive past or family history of mental illness or neurological complaints. There was no history of fever, convulsions, incontinence, swallowing or visual difficulty. He has studied up to 5th standard. He has not consumed alcohol in the past.

INVESTIGATIONS

Urine examination, blood examination, including electrolytes, X-ray chest and skull were within the normal limits. Electroencephalography (EEG) was within the normal limits. Psychological testing was carried out 3 days after the onset of illness. He scored 26 in the mini-mental state examination. His memory quotient was 79. IQ was 88. Bender-Gestalt Test showed signs of organicity [Figure 1]. In Young Mania Rating scale, he scored 32. Rorschach showed that he was very productive.

He was seen by neurologist twice and was diagnosed of having left cerebellar stroke. Computed tomography (CT) brain was done twice, which showed a hypodense lesion in left cerebellar hemisphere suggestive of vascular infarct [Figure 2]. Magnetic resonance imaging (MRI) could not be done due to economic factors. He fulfilled the criteria to diagnose organic manic disorder – F06.30 as per ICD 10.

Patient was followed-up in the next 1 month. He gradually started walking even without support but is still ataxic. Psychiatric symptoms have receded. He was given tablet sodium valproate 200 mg 2 bd and tablet olanzapine 5 mg 2 bd and tablet diazepam 5 mg 2 h.

DISCUSSION AND REVIEW OF LITERATURE

Cerebellum is no more considered to be a repository for motor functions only. Neuroimaging studies confirm that the cerebellum is activated milliseconds before cerebral activation in all aspects of cognitive function like memory and thinking as it is before motor movements. Cerebellum has rich to and fro connections not only to pyramidal areas but also to prefrontal lobe, temporal lobe, limbic area, cingulate gyrus, hypothalamus and thalamus.

The role of the cerebellum in cognition and behavior is gaining more and more importance now.[1] Faulty cerebellar circuits are implicated in the etiology of schizophrenia. Movement abnormalities in catatonia are again thought to be due to cerebellar and basal ganglia involvement.[2] Patients with cerebellar lesions manifesting as schizophrenia is reviewed by Turner and Schiavetto.[3] They beautifully illustrated a patient AA who presented with cerebellar signs as well as symptoms of schizophrenia. Andreason suggests the presence of cognitive dysmetria due to cerebellar involvement in schizophrenia which is analogous to motor dysmetria so far described in cerebellar patients.[4]

CEREBELLUM AND MANIA

Cerebral lesions producing Mania was described by many authors.[5] Yadalam *et al*. describes Mania in two

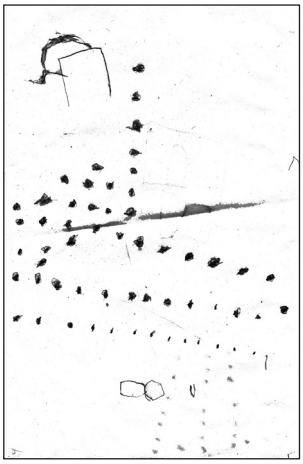

Figure 1: Bender-Gestalt Test drawing of the patient showing evidence of organicity like poor form level & poor relationship to one another and to the whole spatial background

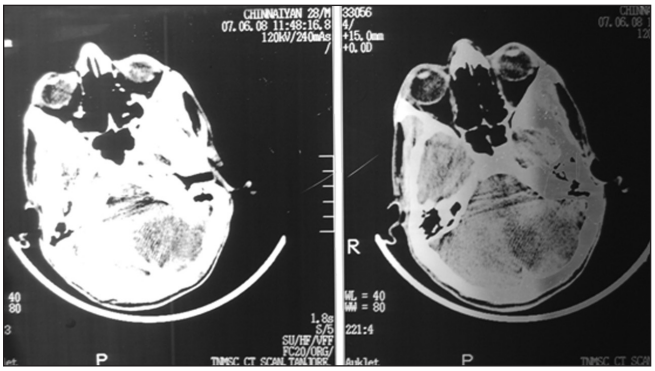

Figure 2: Computed Tomography of brain showing hypo dense lesion in left cerebellar hemisphere suggesting vascular infarct

sisters who have familial cerebellar disease. Both of them had marked vermian and cerebellar atrophy in CT scan.[6] Lauterbach analyzing 45 post-stroke patients noted mania in three patients who had lesions in left cerebellum or left cerebellar tracts to right caudate and thalamus.[7] Lauterbach records that 20% of patients with cerebellar lesions present as bipolar disorder.

EEG recordings in primate mania like aggression shows cerebellolimbic discharges.[8] Deep brain cerebellar stimulation has been noted to influence human mood. Acoustic neuroma presenting as mania has been reported in the literature. Unlike previous reports in literature, our patient is interesting in the sense that the cerebellar lesion has produced cerebellar and manic features simultaneously. Lauterbach patients developed mania in the post-stroke period. Our patient had mania as the acute manifestation of cerebellar stroke.

Schmahman and Sherman in his editorial in brain documents the evidence for the neuropsychological and behavioral involvement in cerebellar lesions.[9] He suggests that cerebellar lesions produce not only motor dysfunction but it produces a cerebellar cognitive affective syndrome. Its defining features are disturbances in executive function, spatial recognition, language and emotional regulation of behavior. He demonstrates these deficits in his bedside clinical neurological evaluation. Localization of lesion and behavioral function in the cerebellum is now reported. Posterior lobe lesions produce core cerebellar syndrome, whereas vermian lesions produce pronounced affective disturbance.

We have been thinking dementia to be of only cortical origin. Now we recognize subcortical dementia and obsessive-compulsive disorder is due to basal ganglia lesions. Similarly, by analogy we should give more attention to the cerebellum for cognition and behavior. Cerebellum is now implicated in the behavioral effects of dementia as well.[10]

CONCLUSIONS

Contribution of the cerebellum to cognition and emotion can no longer be ignored. From a clinical perspective, cerebellum has to be more closely looked for in CT and MRI pictures of our patients. We have to learn more about cerebellum and we should not confine it to only a minor motor role.

Frick mentions that the cerebellum may form a major neurological component of the ego, particularly sub serving the autonomous ego functions.[11] Before concluding I shall quote what Dow says about cerebellum in his book, "Just as cerebellum maintains motor balance it can as well balance other functions of brain of particular relevance to psychiatry."[8]

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