ORGANIC CATATONIA DUE TO CENTRAL PONTINE MYELINOLYSIS- A CASE REPORT

RACHEL PACKIASEELI CHELLADURAI
Department of Neurology,
TIRUNELVELI MEDICAL COLLEGE

Abstract:
Catatonia is a syndrome reported in psychiatric patients. Rarely medical and neurological conditions contribute to the catatonic state. We report a case of catatonia due to central pontine myelinolysis. A 54 year old man, chronic alcoholic presented with acute onset of hypokinesia, generalised rigidity with no weakness. The limbs were kept in the same position during passive movements for prolonged period eventhough the position and posture seemed uncomfortable and bizarre. Patient also had dysphagia to liquids, dysarthria and urinary incontinence. Deep tendon reflexes were normal. There was no significant past medical illness. Complete haemogram, basic biochemical tests and other relevant blood tests were normal. MRI brain showed symmetrical T2 hyperintense signals in the centre of pons representing Central Pontine Myelinolysis. Patient recovered completely with supportive line of management.

Keyword: Catatonia, central pontine myelinolysis(CPM), alcoholism, hyponatremia

INTRODUCTION:
It is customary to consider catatonia as a purely psychiatric disorder, despite many case reports demonstrating a wide range of medical and neurologic disorders associated with catatonic symptomatology (Barnes et al 1986, Clark & Richards 1999 a). The Central Pontine Myelinolysis(CPM) presenting as catatonia is an unusual clinical presentation(9,10). There are only very few case reports of CPM presenting as catatonia especially in this subcontinent.

Key words: Catatonia, central pontine myelinolysis(CPM), alcoholism, hyponatremia

CASE REPORT:
A 64 year old man, chronic alcoholic for 25 years, presented with acute onset, progressive slowness of all activities of 2 weeks duration. He had dysphagia to liquids and dysarthria. He also had urinary incontinence. He had generalised stiffness of all four limbs with no weakness and involuntary movements. The limbs were kept in the same position for a prolonged period even...
when the position and posture seemed uncomfortable and bizarre. There was no altered level of consciousness. He was admitted in psychiatry ward one month ago with a history of excess and irrelevant talk with loss of sleep for one week. He was diagnosed to have Substance Induced Psychosis and treated with Risperidone, Trihexyphenidyl, and Diazepam. He discontinued the antipsychotic medications after discharge. He was not under any medications including antipsychotic drugs for the next 3 weeks. There was no preceeding history of fever or loose stools. There was no past history of hypertension, diabetes, CVA, hepatitis or any other significant medical illness. On examination, vitals were stable. Patient was conscious and oriented with a MMSE 28/30. There was generalised rigidity and catatonia was present. There was waxy flexibility, mask like facies and hypokinesia. Speech was slow and monotonous. There was no weakness of limbs. DTR- were normal. Plantar-bilateral flexor. Urinary incontinence was present. Sensory system and Cerebellum - Normal.

INVESTIGATIONS:

Complete haemogram and basic biochemical investigations were normal. $T_2$ wt MRI Brain showing hyper intensity in pons LFT and Thyroid Function Tests were normal. Serological tests for HIV and Hepatitis were negative. CSF analysis did not reveal any abnormality. EEG was normal.

Neuroimaging with 1.5 Tesla MR scan showed symmetric high signal intensity in transverse $T_2$ wt images which is not suppressed by flair present in the centre of pons without mass effect on the fourth ventricle or brain stem representing myelinolysis of the base of pons spreading centrifugally from the medial raphe characteristically leaving the outer rim of pons unaffected.

DISCUSSION:
The core clinical feature in this case was Catatonia associated with Central Pontine myelinolysis. The diagnosis of organic catatonia was made in this case as per DSM-IV which states “Catatonia disorder due to general medical condition includes the presence of catatonia (as manifested by motoric immobility or excessive motor activity that is apparently purposeless and not influenced by external stimuli, extreme negativism or mutism, peculiarities.
of voluntary movement or echolalia or echopraxia”. The criteria requires evidence from history, physical examination (or) laboratory findings that the disturbance is a direct physiological consequence of a general medical condition and the disturbance is not better accounted by another mental disorder. The disturbance should not occur exclusively during the course of delirium.

In our case, the organic catatonic state was due to Central pontine myelinolysis which was due to chronic alcoholism. A similar case of catatonic stupor in pontine and extrapontine myelinolysis as described by Ruiz, Miyares(4), the patient presented with generalized catatonia, compulsory eating, confusion, pseudobulbar syndrome and CPM. The cause for CPM was hyponatremia (Serum Sodium 127 mmol/L). The patient had no past history of alcoholism. Our patient also had catatonia and pseudobulbar features. But serum sodium was normal.

Another case of catatonia due to CPM was described in a 64 years old woman with a past history of episodic depression and hyponatremia (serum sodium 105 mmol/L) by Julio Chalela(5). With rapid correction of hyponatremia, patient had developed tetraparesis with increased tone and bilateral extensor plantar response over next 2 days the reflexes normalized, but patient developed akinetic mutism with catatonia. Patient had gradual recovery over two weeks. But our patient did not develop pyramidal tract signs and he recovered over two weeks.

When catatonic state occurs in chronic alcoholism, the differential diagnosis should include Alcoholic Psychosis (or) Wernickes Encephalopathy. It was not difficult to distinguish our case from Wernickes Encephalopathy in that there was no oculomotor abnormality, nystagmus, ataxia or confusion with lack of signal changes in the mammillary bodies in periaqueductal area. It was also possible to distinguish our case from alcoholic psychosis in view of the absence of visual hallucinations, intact orientation and the unusual time course unlike alcoholic psychosis in which the symptoms occur within 2 – 4 days after alcohol withdrawal.

Apart from psychiatric illness, organic syndromes like stroke, demyelinating diseases, encephalitis, medications, medical disorders like autoimmunediseases, uraemia, hyperthyroidism, ketoadicidosis, porphyria and Cushing’s disease can cause catatonia.

Several authors have found that a significant percentage of (20 – 39%) of catatonic patients suffer from organic catatonia (Barners et al 1986, Wilcox1986, Bush et al 1996 a). The neurological cause of catatonia include a variety of lesions that may affect any level of the Central Nervous System from the brain stem to the cerebral hemisphere. Catatonia was a statephenomenon (Patterson 1986).

Organic Catatonic disorder occurs due to a variety of neurological disorders mostly affecting the basal ganglia, limbic system, frontal, temporal, parietal lobes and diencephalon. One of the recurring themes in the etiology of organic catatonia is the lesion in an around the III ventricle. These include traumatic haemorrhages in III ventricle (Newmann 1955), Ependymoid cyst in III ventricle (Cairms 1941), Thalamotomy and thalamic lesions (Kleist 1960), Subthalamic mesencephalic tumours (Newmann 1995); Globus pallidus lesions either bilateral (Mettler 1955) or focal (Kleist 1960), atherosclerotic parkinsonism (Brain 1962) can also be associated with catatonic features.

Catatonia has also been thought to be a manifestation of frontal lobe lesion (Kleist 1960, Taylor 1990), parietal lobe lesions (Trippins & Dunner 1981), temporal lobe lesions
(Malamud & Boyd 1929, Sours 1962, De Morsier 1968), Post encephelatic states (Timey & Howe 1920, Cheyette comings 1995) and AIDS dementia / HIV encephalopathy (Volkon et al 1987, Carroll et al 1994, Brough et al 2000). Our patient presented with catatonic state with feature of pseudobulbar palsy. Lobar functions were normal. Two principal forms of catatonia are described. 1. A hypokinetic retarded – stuporous (Parkinsonism) variety 2. A hyperkinetic excited- delirious variety. Neurochemical and neuroanatomical basis of catatonia remain unknown. It seems that several neurotransmitters are critical in the causation of catatonia. The Parkinsonism subtype may indicate dopaminergic disturbance in cortico-striatal-thalamo-cortical circuit (motor loop). GABAergic system too have been implicated. The dyskinetic subtype may signify GABAergic dysfunction. Benzodiazepines acting as GABA facilitation probably alleviate a dopaminergic blockade in the mesostriatal and mesolimbic systems thereby stimulating the motor systems and releasing the inhibited catatonic behaviour (Salam et al 1987). This finding is important keeping in mind the good response of ‘hypo kinetic catatonia’ to lorazepam and other benzodiazepines which act on GABA-A receptor. CPM associated with alcoholism has a better prognosis. (Mochizuki et al 2003). Our patient also improved with supportive management and benzodiazepines.

CONCLUSION:
Central pontine Myelinolysis presenting as catatonia is a very rare presentation. That too alcoholism as a cause for CPM is unusual as described above. This case is presented for the rare association of catatonia / CPM/alcoholism.

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