ORIGINAL RESEARCH PAPER

Psychiatry

TUBERCULOMA LEADING TO CATATONIA: ORGANIC CATATONIA: CASE REPORT

KEY WORDS:

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ABSTRACT

Catatonia has been described as a disorder of diminished (stupor) or increased (excitement) psychomotor activity associated with catatonic symptoms. A number of neurological and metabolic disorders can present with symptoms of catatonia (Taylor, 1990). Here we report a case of 28-year-old male brought by his wife with chief complaints of right sided unilateral, headache, loss of left sided field of vision since 10 days after 3 days patient experienced 5-6 episodes of convulsions after that the patient became completely silent, was not able to eat, was not able to recognise his family members, maintained a position for long time, not following commands & was giving a blank stare look, this continued for 3-4 days then patient was brought to the hospital.On CNS examination multiple cranial nerves & motor system was involved. On Psychiatric Examination he was mute, showed catalepsy and maintained odd postures for a long time. He was actively negativistic and refused to open his mouth to take meals, maintained a constant staring look and showed extreme hypo activity, immobility & minimally responsive to stimuli. NCCT Head was S/O large area of hypo dense involving right occipital & posterior parietal region with midline shift towards contralateral side likely to be subacute infarct. MRI brain showed tubercular focal lesions in right posterior parietal lobe & MR spectroscopy reveals lipid lactate peak S/O tuberculosis.

INTRODUCTION

Catatonia labels a cluster of striking motor symptoms and signs that co-exist with idiopathic psychoses (affective disorder, schizophrenia), intrinsic coarse brain disease, metabolic disorders that affect brain function or drug induced syndromes (most commonly the Neuroleptic Malignant Syndrome) (Taylor, 1990). Catatonia was first described by Kahlbaum in a monograph in 1874. Subsequently catatonia was incorporated as a subtype of schizophrenia (Fink & Taylor, 1991). In ICD-9, DSM-III and DSM-III-R, catatonia was categorized only as a subtype of schizophrenia, there has been a gradual recognition of the fact that catatonia can be observed in a variety of organic disorders, as well as in affective disorders (Gelenburg, 1976). The ICD-10 Classification of Mental and Behavioral Disorders (World Health Organization, 1992) has included a category of organic catatonia under F 06 'Other mental disorders due to brain damage and dysfunction and to physical disease'. It has been described as a disorder of diminished (stupor) or increased (excitement) psychomotor activity associated with catatonic symptoms. A number of neurological and metabolic disorders can present with symptoms of catatonia (Taylor, 1990).

Deep medial lesions of the frontal lobe, particularly those involving the SMA (supplementary motor area), may give rise to emotional blunting, decreased speech output or mutism, perseveration, severe apathy, posturing and catatonic-like symptoms (like gegenhalten and waxy flexibility) (Joseph, 1996). Lesions of parietal lobe too can lead to organic catatonia, like biparietal infarct. lesions affecting the limbic system (especially the cingulated gyrus and its connections to the frontal lobe) and the temporal lobe have been reported to cause catatonia. It is interesting to note that amygdala, under conditions of extreme fear and arousal, can produce catatonic-like frozen states, including waxy flexibility, presumably by acting on the stratum and SMA (supplementary motor area). Similarly, massive bilateral lesions of caudate and anterior putamen have been known to produce catatonic features. Many times, catatonia can occur due to diffuse brain pathology, such as closed head injury and diffuse brain trauma (Sutter et al ,1959), dementia (Valenstein et al ,1985), diffuse cerebral atrophy, and multiple sclerosis (Mathews, 1979; Pine et al.,1995; Mendez.1999). Bacillar aneurysms, AVM of posterior cerebral vessels and ACA aneurysms, may also present with catatonia. Catatonic signs can also appear due to metabolic & endocrinological disorders along with nutritional deficiencies. It may also occur both during intoxication as well as withdrawal from drugs of dependence.

Case Presentation

The patient is a 28-year-old male brought by his wife & other family members to the outpatient unit of department of psychiatry in CIMS Bilaspur [C.G] with chief complaints of right sided unilateral, excruciating headache, intolerant to light and sound , was not able to sleep with loss of left sided field of vision since 10 days later on after 3 days patient experienced 5-6 episodes of convulsions which manifested as up rolling of eyes, deviation of mouth, drooling of saliva & complete stiffness in the body. Each convulsion lasted for 2-3mins with no loss of consciousness, involuntary defecation or urination. The very next day the patient became completely silent, was not able to eat, was not able to recognise his family members, maintained a position for long time, not following commands & was giving a blank stare look, this continued for 3-4 days as patient was being taken to local quacks for the resolution of his problems. As the symptoms continued finally they decided for the doctor's opinion & brought him to the hospital. As per the informant the patient used to smoke 10-12 bidis every day since 12yrs and use to drink a quarter of local alcohol 3-4 times weekly since 5-6 yrs. On applying the BFCRS –Catatonic scale the score was found to be 24/69.

Physical examination: At initial evaluation the patients temperature, pulse and blood pressure were normal. Neck stiffness was not present. The abdomen, respiratory and cardiovascular systems were normal. C.N.S. examination showed rt sided semi dilated pupil, sluggishly reactive to light, involvement of 2^{nd}, motor inv. of 5^{th} & 7^{th} , 8^{th} & 12^{th} cranial nerves , in Superficial reflexes bilateral plantars were mute, in deep tendon reflexes biceps, triceps, supinator & knee reflexes were absent , sensory & cortical function were intact, no signs of meningeal irritation, Romberg test showed to & fro swaying movement of patient on eyes closed.

Psychiatric Examination: The patient opened his eyes spontaneously and looked around. He made no spontaneous movements and didn't obeyed simple commands. He made no spontaneous speech and did not respond to any questions. He was mute throughout the examination. He showed catalepsy and maintained odd postures for a long time. He

was actively negativistic and refused to open his mouth to take solids or Liquids. He maintained a constant staring look and showed extreme hypo activity, immobility & minimally responsive to stimuli.

Treatment & Course in Hospital: After admission the patient was started I/V Lorazepam 2mg/ml TID, the very next day all the catatonic features had disappeared and he began to speak in response to questions. He began to take feeds by mouth and was able to meet his toilet needs. Routine investigations were found to be normal. Later on NCCT Head was suggestive of large area of hypo dense involving right occipital & posterior parietal region with midline shift towards contralateral side likely to be subacute infarct. Further on medicine consultation appropriate treatment was started. Further on Ophthalmologic consultation the fundus examination showed bilateral papilloedema. MRI brain showed tubercular focal lesions in right posterior parietal lobe & MR spectroscopy reveals lipid lactate peak S/O tuberculosis. Later as the symptoms of patient showed no significant improvement the patient was referred to the neurology department for further evaluation & management.

DISCUSSION

According to Barnes et al. (1986), the definition of acute catatonic syndrome (ACS) requires that, over a period of days or weeks, the patient develops at least one motor sign (catalepsy, posturing or waxy flexibility) in combination with one or more behavioural signs such as: (a) negativism, mutism or stupor; (b) excitement; (c) bizarre repetition behaviour. More recent revised criteria from the Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5) published in 2013 also include agitation, mannerisms, stereotypies, grimacing, echolalia and echopraxia. According to this latter definition, three or more out of twelve established clinical features (among all characteristics already cited) are necessary to establish a diagnosis of catatonia. However, some specialists criticize these new DSM-5 criteria as it lists inaccurate motor and speech abnormalities which overlap with various neurological and psychiatric disorders, possibly causing confusion in determining catatonia as an entity in itself. Catatonia etiology can be structural or functional diseases of the central nervous system (CNS) as well as systemic disease causing neurological symptoms. Carroll et al. (1994) studied a group of catatonic patients and found that, of those cases with an apparent psychiatric etiology, around a third had a neurological condition as the presumed cause. The complete physiopathology of catatonic syndrome and its correlation with cerebral structures are not fully understood. Some studies suggest hypo metabolism in the basal ganglia, while others show hypo perfusion in frontal, temporal and parietal lobes. Functional imaging studies for instance have shown that catatonia is associated with altered activity in orbito-frontal, prefrontal, parietal, and motor cortical regions, where these cortical structures may play a role in the pathophysiology of catatonia. This is also reinforced by observations that GABA-A binding is reduced in cortical areas. There may also be dopaminergic involvement related to the diencephalon, brainstem and limbic regions, such as the cingulate gyrus.

CONCLUSION

If catatonia presents acutely, complete history with a thorough semiology analysis of patient symptoms and further complementary investigation are always necessary. The primary goal is to search for an epileptic event with no convulsive manifestation. We must change the misconception that catatonia is solely a psychiatric illness. In fact, it is a condition with many possible etiologies whose prognosis depends on correct differential diagnosis that allows the physician to provide early treatment of its underlying cause.

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