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Case Report
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Intracranial Space-Occupying Lesion Presenting as Catatonia in a Patient of Affective Disorder with a History of Recurrent Catatonic Episodes: A Case Report

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ABSTRACT

Intracranial space-occupying lesions (ICSOLs) like hematomas, neoplasms, granulomas, or brain abscesses can present with diverse neuropsychiatric manifestations. Subtle cognitive deficits, anxiety, affective symptoms—especially depression, personality changes, and psychosis, most commonly hallucinations, have been associated with organic etiology. Catatonia has seen a paradigmatic shift in its etiological basis and space-occupying lesion is a rare yet important cause to be investigated for, as it poses a challenge in terms of searching for early clinical indicators of cerebral pathology and difference in treatment outcome due to etiology. Here we present a case of catatonia due to ICSOL in a patient of affective disorder with a history of recurrent catatonic episodes, highlighting the importance of approaching each catatonic presentation as a syndrome with diverse possible etiology.

Keywords: Catatonia, intracranial space-occupying lesion, affective disorder

INTRODUCTION

Intracranial space-occupying lesions (ICSOLs) can present with a multitude of neuropsychiatric manifestations. Hematomas, neoplasms, granulomas and abscesses are broadly grouped under space-occupying lesions. They can present with new onset psychiatric symptoms, color the presentation of preexisting psychiatric disorder, or even get masked by presence of a psychiatric comorbidity. Keschner et al reported 78% of diagnosed brain tumor patients had psychiatric symptoms and 18% of these patients presented initially with psychiatric symptoms.[1] Psychiatric manifestations depend on various factors like histological type of the tumor, its location, pressure effects and even premorbid factors.[2] Subtle cognitive deficits,[3] anxiety, and affective symptoms—especially depression, apathy, personality changes, and psychotic symptoms—have been associated with organic etiology.[4] Over the years, catatonia has seen a paradigmatic shift in its etiological conceptualization from initial observations of Kahlbaum describing it as a part of various psychiatric illnesses to its current understanding as a syndrome with a multitude of etiological factors, including brain masses. Literature on intracranial space-occupying lesions presenting as catatonia is limited, mostly to lesions of diencephalon, limbic system, including hydatid and epidermoid cysts, pinealoma and craniopharyngioma, arachnoid cysts

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of parietal lobe, glioblastoma of corpus callosum and brain abscess.[5] Emergence of such association poses a challenge for the psychiatrist to search exhaustively for focal neurological signs and seek aid of neuroimaging liberally as organic causes alter the treatment outcome and overall prognosis markedly.[6] Here we present a case of catatonia suspected due to ICSOL in a patient with a history of multiple previously diagnosed episodes of catatonia in background of affective disorder. We aim to demonstrate a clinical scenario where initial features presented a case for psychiatric etiology but evolution of symptoms and appropriate investigations revealed an underlying structural etiology. The literature on such atypical presentations is scant and we hope the longitudinal course of symptoms described here would aid clinicians in early diagnosis and better management. Written informed consent was obtained from the patient for anonymous use of clinical information and material for purpose of academic sharing and publishing.

CASE PRESENTATION

A 37-year-old male presented with complaints of forgetfulness, inattention, and reduced talks for past 11 days, not taking food, not talking at all, and maintaining odd postures for past 6 days.

Past Psychiatric History

• At age 23 years, developed odd posturing, wandering behavior, paranoid ideation, absconded from home 1 month into illness, returned 5 months later by self and was at premorbid functioning.

• At age 25 years, depressive episode with catatonic signs subsiding with 7 sessions of electroconvulsive therapy (ECT) and subsequently maintained on olanzapine (10 mg) and fluoxetine (20 mg).

• At age 30 years, 12 days after stopping medications, patient developed catatonia. Subsided completely with 7 sessions of ECT.

• At age 32 years, developed catatonic signs over 1 month, showed good response with escitalopram (10 mg) and haloperidol (15 mg) initially, but eventually showed switch to manic symptoms. Maintained well on carbamazepine (1200 mg) and quetiapine (200 mg).

• At age 37 years, while on treatment, developed manic signs, which subsided in 15 days on substituting quetiapine with risperidone (4 mg).

Neuroimaging or immunological testing were never carried out during the entire course. He had no comorbid major illness.

Family History

Cerebrovascular accident at age of 45 years in the elder brother.

Mental Status Examination

Patient had mutism, psychomotor retardation, and negativism (Bush–Francis Catatonia Rating Scale, BFCRS = 10). Voluntary postures were comfortable, gaze was fixed, and affect was mask-like.

Clinical Course

Upon admission for in-patient care, he showed response to parenteral lorazepam 6 mg and started oral intake within 24 hours (BFCRS = 3). Three days into admission, patient complained of bilateral headache. On oral lorazepam, 8 days into admission, patient developed generalized slowness, confused movements, maintaining postures and on examination had cogwheel rigidity, staring, and echolalia (BFCRS = 16). Twelve hours later, patient had an episode of complex partial seizure, manifesting as jaw movements, lasting for 30 seconds. Over the next 24 hours, patient showed improvement (BFCRS = 3) in taking meals and talking but had one episode of vomiting and complained of uneasiness and weakness and would prefer to lie down frequently. He developed an unsteady gait with reduced right leg swing and on examination had rigidity and autonomic instability. Lorazepam was stopped and patient kept on quetiapine (150 mg). Over the next 4 days, his food intake again reduced (BFCRS = 6) and on the fourth day, he had two episodes of projectile vomiting followed by an episode of generalized tonic clonic seizure. On examination, patient had weakness of adduction movement in left eye. Tomography of brain showed irregular multiloculated rim enhancing lesion of size approx. 46 mm × 36 mm in left parieto-occipital region with marked perilesional edema and midline shift (Figure 1). Differentials of abscess, metastatic lesion, and glioblastoma multiforme were considered. Over the next 24 hours, the patient showed improvement (BFCRS = 3) but on examination, had left eye adduction weakness, left eye mild ptosis and weakness in right lower limb. He was referred to a neurosurgical center, where he unfortunately succumbed before any intervention could be done.

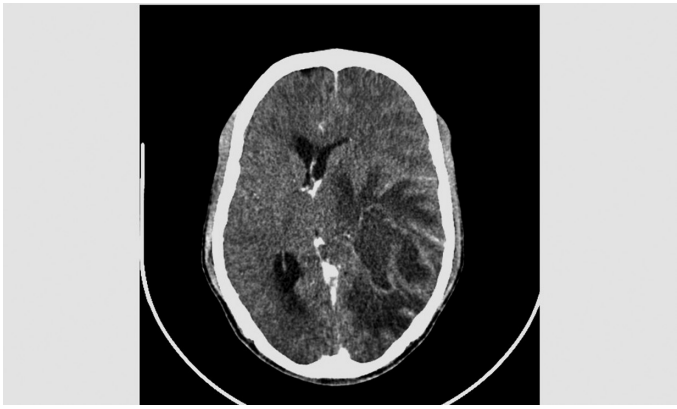

Figure 1. Computed tomography brain showing a space-occupying lesion in the left parieto-occipital region and midline shift.

DISCUSSION

Catatonia maybe of psychiatric or general medical condition origin, with systemic illnesses and central nervous system abnormalities like encephalitis, seizures, metabolic disturbances, or structural damage—contributing to the organic causes, responsible for up to 20%-25% of catatonic presentations.[5] In our patient, the development of bilateral headache, two seizure episodes, unsteady gait, and eventually left eye ptosis and reduction in right lower limb power were clinical signs of organicity. The absence of such signs in previous catatonic episodes and complete interepisodic recovery indicates a new onset brain lesion manifesting as a catatonic episode. A comprehensive evaluation of catatonic episodes have helped discriminating such comorbid conditions.[7] There is also a possibility of chronic cerebral lesion contributing to bipolar presentation over 15 years. Psychiatric symptoms have been shown to herald the diagnosis of brain tumors after months to years.[8] Slow progressive growth might not have led to focal neurological symptoms or signs. Coexistence of a silent organic lesion with bipolar illness, the pharmacological

management of which may have masked the emergence of symptoms specific to organic lesion is a theoretical possibility too.

The frontal lobe has been investigated for its involvement in catatonia, given its role in emotional control, motor regulation, and arousal. Functional neuroimaging studies have implicated the orbitofrontal cortex and dorsomedial prefrontal cortex in behavioral and motor symptoms of catatonia, respectively.[9] Frontal and parietal lobe connectivity alterations are very common in catatonic states.[10] Furthermore, the role of *N*-methyl-D-aspartate (NMDA) receptors has been unclear, though use of NMDA antagonist memantine in catatonia and high prevalence of catatonic presentation in anti-NMDA encephalitis[11] point toward a key role. Glial cells have regulatory role in NMDA transmission which can explain glial cell tumors presenting with catatonic symptoms. *N*-methyl-D-aspartate is known for its ability to induce long-term changes and our case in point, may predispose to further catatonic episodes with new-onset neurological lesions.

Poor response to benzodiazepines has been reported for organic causes[12] and was seen in this case. The role of electroconvulsive therapy is debatable as, although seizure episodes were followed with reduction in BFCRS scores, onset of raised intracranial tension is a relative contraindication. Also, cerebral pathology has been associated with poor response to ECT.[13] In lesions of diencephalon or temporal lobe,[14] resection of the tumor has led to resolution of catatonia.

CONCLUSION

The pathophysiology of catatonia remains unelucidated and further functional neuroimaging studies need to be carried out. It is of paramount importance to consider catatonia as a syndrome with diverse etiological factors and each presentation to be evaluated exhaustively, as our case indicates, despite the presence of past catatonic episodes.

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

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REFERENCES

1. Keschner M, Bender MB, Strauss I. Mental symptoms associated with brain tumor: A study of 530 verified cases. *J Am Med Assoc*. 1938;110(10):714-718. [CrossRef]
2. David AS, Fleminger S, Kopelman MD, Lovestone S, Mellers JDC. *Lishman's Organic Psychiatry - A Textbook of Neuropsychiatry*. 4th ed. Wiley-Blackwell; West Sussex (UK): 2009.
3. Cappa SF, Cipolotti L. 161Cognitive and behavioural disorders associated with space-occupying lesions. In: Cappa S, Abutalebi J, Demonet JF, Fletcher P, Garrard P, eds. *Cognitive Neurology: A Clinical Textbook*. Oxford: Oxford University Press; 2008. [CrossRef]
4. Madhusoodanan S, Ting MB, Farah T, Ugur U. Psychiatric aspects of brain tumors: a review. *World J Psychiatry*. 2015;5(3):273-285. [CrossRef]
5. Ahuja N. Organic catatonia: a review. *Indian J Psychiatry*. 2000;42(4):327-346.
6. Smith JH, Smith VD, Philbrick KL, Kumar N. Catatonic disorder due to a general medical or psychiatric condition. *J Neuropsychiatry Clin Neurosci*. 2012;24(2):198-207. [CrossRef]
7. Lyra A, Cordeiro Q, Shiozawa P. Catatonia after glioblastoma multiforme in a patient with schizophrenia: the importance of establishing etiology. *J Neuropsychiatry Clin Neurosci*. 2015;27(3):e216-e217. [CrossRef]
8. Ghandour F, Squassina A, Karaky R, Diab-Assaf M, Fadda P, Pisanu C. Presenting psychiatric and neurological symptoms and signs of brain tumors before diagnosis: A systematic review. *Brain Sci*. 2021;11(3):301. [CrossRef]
9. Northoff G, Steinke R, Nagel DCzerwenka C, et al. Right lower prefronto-parietal cortical dysfunction in akinetic catatonia: a combined study of neuropsychology and regional cerebral blood flow. *Psychol Med*. 2000;30(3):583-596. [CrossRef]
10. Walther S, Stegmayer K, Wilson JE, Heckers S. Structure and neural mechanisms of catatonia. *Lancet Psychiatry*. 2019;6(7):610-619. [CrossRef]
11. Rogers JP, Pollak TA, Blackman G, David AS. Catatonia and the immune system: a review. *Lancet Psychiatry*. 2019;6(7):620-630. [CrossRef]
12. Carroll B, Goforth H. Medical catatonia. In: *Catatonia: From Psychopathology to Neurobiology*. American Psychiatric Association; Ann Arbor, Michigan (USA): 2007.
13. Luchini F, Medda P, Mariani MG, Mauri M, Toni C, Perugi G. Electroconvulsive therapy in catatonic patients: efficacy and predictors of response. *World J Psychiatry*. 2015;5(2):182-192. [CrossRef]
14. Franssen A, Sienaert P. Temporal glioblastoma presenting as catatonia. *BMJ Case Rep*. 2019;12(3):e224017. [CrossRef]