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CASE REPORT
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Case Report: Thalamic Stroke Patient with Neuropsychiatric Symptoms

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Introduction

The thalamus is a complex structure acting as a relay for various tracts to convey information and mediate action. The reticular activating system also forms a part of the thalamus and, affects arousal attention and mediates voluntary movement. Thalamic strokes, although rare, constitute 3% of all CNS strokes with a telling symptom of excessive daytime somnolence and impaired arousal.[1] It can present with vegetative symptoms and can present as geriatric depression and sometimes even with delerium.[2] This case report highlights the clinical presentation of thalamic stroke in the form of avolition and neurological negative emotionalism with a peculiar quality of depression with themes of identity loss, that were not typical of a simple mood episode with negative symptoms. We recorded a case of thalamic stroke, which presented with severe depression and delirium and was detected and treated early due to an early psychiatric referral.

Case Report

Mr. J. D., an 81-year-old retired male previously working as a medical practitioner, presented with the following complaints of cognitive and emotional changes that were acute in onset and with progressive decline in functioning, including prominent forgetfulness and naming difficulty. The patient and relative complained of excess daytime drowsiness with a total sleep duration amounting to 21 hours. The patient reported of severe lack of energy and sadness of mood experienced for most of the day for most days in the last 3 months. Relatives reported poor self care, with the patient needing assistance for all activities of daily living. The patient voiced repeated passive death wishes with a constant theme of loss of sense of meaningful existence. On tracking the progress of the illness, we uncovered a previous similar episode with the deterioration that could be graphed as a progressive step ladder pattern of deteriorating illness over the last 4 years. The patient was a known case of chronic dermatological illness of psoriasis. The patient lived with his wife and had no history of substance use. Patient had a past psychiatric history of Moderate depressive episodes without

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psychotic symptoms, which occurred 6 years ago, and lasted for 9 months but Treatment history was not available.

Mr. J.D. presented to the neurology clinic with complaints of cognitive decline and emotional disturbances following a recent stroke. He reported the sudden onset of symptoms approximately six weeks ago, characterized by weakness on his left side and difficulty speaking. The patient needed assistance with all activities of daily living. On clinical assessment there was evidence of Right trigeminal nerve dermatome with herpes lesions. Magnetic resonance imaging (MRI) of the brain revealed an acute infarct in the right medial and inferior thalamus region and lacunar infarcts in the periventricular region. Patient's neurological examination revealed Mr. J.D. presented with left-sided hemiparesis and mild dysarthria. Cranial nerve examination was within normal limits except for the right facial nerve, with Ramsey Hunt syndrome evident. Sensory examination showed decreased sensation to pinprick and light touch on the left side. Reflexes were brisk on the right side, with an extensor plantar response. The cognitive assessment revealed deficits in attention, executive function, and memory, and hemispatial neglect on the clock drawing test. Mood assessment demonstrated symptoms of depression, including low mood, anhedonia, and feelings of worthlessness, along with severe apathy and avolition.

Diagnostic evaluation was done as follows, with an MRI brain showing an acute infarct involving the right thalamus, consistent with a lacunar stroke. Laboratory tests were routine blood tests, including complete blood count, electrolytes, renal function, and thyroid function tests, which were within normal limits.

Neuropsychological evaluation

Formal neuropsychological testing revealed deficits in attention, executive function, and verbal memory consistent with thalamic involvement. Clock drawing test showed visuospatial impairment and hemineglect on the left side with crowding of numbers on the right side.

Diagnosis

As per ICD 11code 6D81, dementia due to cerebrovascular disease and 6E62 Secondary mood syndrome- boundary with dementia. [Mr. J.D. was diagnosed with a thalamic stroke resulting in cognitive impairment and neuropsychiatric symptoms, including depression. (vascular dementia, post-stroke depression)]

Management was as follows: with medical management for Mr. J.D., he was started on antiplatelet therapy (aspirin) and statin therapy to reduce the risk of further cardiovascular events. His blood pressure and lipid profile were optimized. Dermatology referral and management of herpes. For physical therapy, he was referred to a rehabilitation program for physical and occupational therapy to improve motor function and activities of daily living. For neuropsychiatric treatment, A multidisciplinary approach involving neurology and psychiatry was employed. Mr. J.D. was started on an antidepressant (selective serotonin reuptake inhibitor) for the treatment of depression. Transient delirium was managed with low-dose antipsychotics along with non-pharmacological management with daily schedule and activity planning. Multivitamin injections with age-appropriate nutrition management were done. Psychoeducation and supportive therapy were provided to address emotional adjustment to stroke-related disability. For cognitive rehabilitation, strategies focusing on attention, memory, and executive function were implemented to help mitigate cognitive deficits. While admitted for the duration of one week, Mr. J.D. was scheduled for regular follow-up appointments with neurology, psychiatry, and rehabilitation services. He demonstrated gradual improvement in motor function and cognitive symptoms over the subsequent week. His mood symptoms also showed significant improvement with pharmacotherapy and supportive therapy.

Discussion

Thalamic strokes can result in a wide range of cognitive and neuropsychiatric symptoms due to the thalamus's role in relaying sensory and motor signals and its involvement in cognitive and emotional processing.[1,2] Cognitive deficits commonly include attention, memory, and executive dysfunction, while neuropsychiatric symptoms such as depression can significantly impact functional outcomes.

Multidisciplinary management involving neurology, psychiatry, rehabilitation, and supportive services is essential for optimizing outcomes in patients with thalamic stroke. The patient showed 50% improvement at the time of discharge.

The thalamus consists mostly of grey matter but also has white matter structures composed of external and internal laminate, which cover the lateral surface of the thalamus while dividing the thalamic nuclear into many divisions.[3] Functional division of the thalamus includes reticular and intralaminar nuclei (arousal and pain), sensory nuclei, effector nuclei, associative nuclei (cognition), and limbic nuclei (mood and motivation). The circuit of the reticular activating system is responsible for arousal, attention, sleep-wake transitions, and circadian rhythm. Within the RAS: The cortical–hippocampal–cortical circuit is responsible for sleepy-dependent memory consolidation, while the basal forebrain circuit is for REM sleep, cortical activation, and attention. The prefrontal-amygdala circuit for sleep-related emotional reactivity, and attention. It was clinically evident in our patient that the excessive daytime sleepiness amounting to over 20 hours of sleep and with a score of 22 on the Epworth sleepiness scale, with the above symptoms that we were looking at geriatric depression, which was actually a result of CVA, probably affecting thalamus and was masking the vascular event. During wakefulness, the RAS originates in the brainstem and activates the thalamus and cortex via well defined "Bottom-up "pathway; however, another "Top-down" pathway includes projections for the sapience network of the neocortex, amygdala, and hypothalamus.[4] Hypersomnolence is a typical feature seen in 80% of anterior strokes and 20% of posterior thalamic strokes.

Sleep and Neuroplasticity

Deep sleep, characterized by sleep spindles, is important for neuroplasticity, and changes in the sleep micro architecture following thalamic stroke could underline cognitive deficits like thalamic amnesia and dementia. Anterior and paramedian thalamic strokes typically cause thalamic dementia.[5,6] Characterised by temporal and spatial disorientation, behavioural changes: Apathy or agitation, Impaired executive functions: working memory, cognitive flexibility, abstract thinking, Attention deficits. Association of depression and anxiety is higher in anterior thalamic lesions; behavioral disturbances and delirium are associated with a paramedian stroke of the thalamus.[7]

Thalamic stroke has shown to have not complete recovery along with reduced neuroplasticity related to changed sleep. Patients with incomplete recovery need a high amount of sleep immediately and even after one year of the thalamic stroke.[8] This is most pronounced in anterior strokes. We learn from this that along with the psychiatric assessment and neurological assessment that guided the treatment, it is important to pay as much importance to the descriptive quality of the emotion that the patient experiences. Assessment t of states of arousal was thoroughly done, which led us toward the clinical diagnosis of a thalamic stroke and prompted apt treatment. However, the need for better rehabilitation services was felt with an urgency in the tribal region of Palghar. Although the patient recovered symptomatically after a week of indoor admission, the stigma of the depressive illness led to the discontinuation of treatment one week after discharge.

In conclusion, this case highlights the complex interplay between neurological and psychiatric symptoms following a thalamic stroke. It cannot be stressed enough that early psychiatric referral and treatment initiation can change the treatment outcome and improve the quality of life of the patient significantly. A comprehensive approach addressing medical, rehabilitative, and neuropsychiatric needs is crucial for maximizing functional recovery and quality of life in these patients. Long-term monitoring and support are essential to address ongoing cognitive and emotional challenges.

Declaration of Funding

Nil.

Conflict of Interest

Nil

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