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CASE REPORT

Post-stroke Bipolar Affective Disorder: A Case Report

Kaushal K. Singh[1]*, Zareen Akhtar[1], Shashank S. Sinha[2], Prerak Kumar[2], Babli Kumari[3], Mohit Jain[3], Amit Singh[3]

[1]*Faculty of Medicine, King George's Medical University, Lucknow, Uttar Pradesh, India*
[2]*Department of Geriatric Mental Health, King George's Medical University, Lucknow, Uttar Pradesh, India*
[3]*Department of Psychiatry, King George's Medical University, Lucknow, Uttar Pradesh, India*

Abstract

Post-stroke patients are at substantial risk of developing a spectrum of neuropsychiatric syndromes due to permanent damage to brain parenchyma. Existing literature suggest that depression, dementia, and anxiety are amongst the common organic mental disorders in post-stroke patients. However, little literature exists that report cases of bipolar affective disorder after stroke. Here we have mentioned the case of a 58-year-old person, suffered a cerebral hematoma, then developed depressive symptoms, and subsequently developed manic symptoms.

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***Correspondence:**
Kaushal Kishor Singh
kaushal2018@kgmcindia.edu
King George's Medical University, Lucknow, Uttar Pradesh, India

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Introduction

Stroke is defined as "a sudden loss of blood supply to the brain leading to permanent tissue damage caused by thrombotic, embolic, or hemorrhagic events."[1] Brain stroke is the second leading cause of death globally with an annual mortality rate of about 5.5 million.[2] Moreover, patients who survive after stroke are at substantial risk of developing a spectrum of neuropsychiatric syndromes due to permanent damage to brain parenchyma. The occurrence of neuropsychiatric disorders such as depression, anxiety disorder, apathy, mania, pathological laughing, and crying as sequelae of vascular injury to the brain has long been known.[1] These disorders are essential to be understood, diagnosed and managed as they directly affect the response to rehabilitation, recovery, and quality of life of post-stroke patients and are a major burden to mental health caregivers.

Post-stroke depression (PSD) has been found to be the most common out of the spectrum of disorders reported to occur in post-stroke patients, with a cross-sectional prevalence of 18 to 33% according to various meta-analyses.[3] Despite the known strong association, the pathogenesis of PSD is partially understood however, the majority of the literature suggests that both neurobiological and psycho-social factors play a role.[4] Symptoms of PSD are similar to other forms of depression. However, PSD patients experience greater degrees of sleep disturbances, vegetative symptoms, and social withdrawal.[5]

The diagnosis of PSD according to the DSM-5 relies on five criteria:

- presence of depressed mood or anhedonia,
- symptoms are pathophysiologically related to the stroke,
- symptoms are not better explained by other psychiatric disorders,
- disturbance does not occur exclusively in the presence of delirium,
- symptoms cause significant distress or impairment.[3]

Mania, though a rarely reported sequela of stroke with a prevalence of less than 1%, has an evident impact on the patient's social functioning.[6] The pathogenesis of post-stroke mania has been related to genetic factors, pre-existing subcortical brain atrophy, and damage to the right corticolimbic pathway.[7]

Bipolar affective disorder (evidence of both depressive and manic episodes) following stroke is a rare sequel further. Very few reported cases of post-stroke bipolar disorder exist in literature which makes its diagnosis and management challenging in the clinical practice.[8-10] We here report a case of post-stroke depression, followed by mania, in a hypertensive, diabetic male patient who suffered a stroke one year two months back.

Case Presentation

A 58 years old elderly male with a history of diabetes mellitus, hypertension, and hypothyroidism had an episode of stroke 1-year and 2 months back. After recovering from a stroke, the family members reported that the patient was remaining withdrawn to himself. He also reported low mood, decreased interest in work and interaction. He mostly confined himself to his house and had disturbed sleep. He believed that nothing would be alright and was feeling hopeless. The consulting neurologist prescribed him escitalopram 10 mg/day and clonazepam 0.5 mg/day as an add-on to the treatment going on for diabetes mellitus, hypertension, and hypothyroidism. The patient's depressive symptoms improved with this treatment, and he became symptom-free in approximately four months; however, he continued to take the antidepressant medications regularly.

However, after 6 months, without any apparent stressor, it was noticed that the patient was talking more than usual and becoming irritable on trivial issues. He was sleeping less than usual and roaming here and there throughout the day. The patient developed increased goal-directed activity in the form of planning to set up a new business and buying a new house despite a lack of financial and logistic resources. He would claim that he had a good fortune for his business plans. Initially, he would even meet strangers and share his plans with them. Gradually, his symptoms worsened over a few weeks to the extent that the patient started picking up things from garbage, buying old items from shops, and hoarding them at home. Irritability has been replaced by anger outbursts on trivial issues, and he argued a lot with strangers stating that they are envious of his plans. Arguments were sometimes followed by physical quarrel with others.

On the day of admission, the mental state examination (MSE) of the patient revealed increased psychomotor activity, tone, rate of speech, and pressured speech. His mood was cheerful with irritable affect, grandiose delusion of power and possession resulting in persecutory delusions were present. Referential ideas were also present. However, perceptual abnormality was absent. Judgment and insight were poor. At the time of admission, the Young Mania Rating Scale (YMRS) score was 34. His past and family histories were not contributory. The patient had a history of tobacco use in dependence pattern for the past 15 years.

Detailed physical examination did not reveal any abnormality. Blood investigations were within normal limits. Magnetic resonance imaging (MRI) of the brain at the time of stroke revealed acute hematoma in the left frontal lobe extending medially up to the left lateral ventricle leading to moderate surrounding edema and midline shift to the right side (Figure 1). A diagnosis of bipolar and related disorder due to another medical condition (organic bipolar affective disorder) with tobacco use disorder with type 2 diabetes mellitus (T2DM) with Hypertension and Hypothyroidism was made. Considering multiple co-morbidities, the patient was prescribed Sodium Valproate 1000 mg/day, Aripiprazole 10 mg/day, and Lorazepam 3 mg/day,

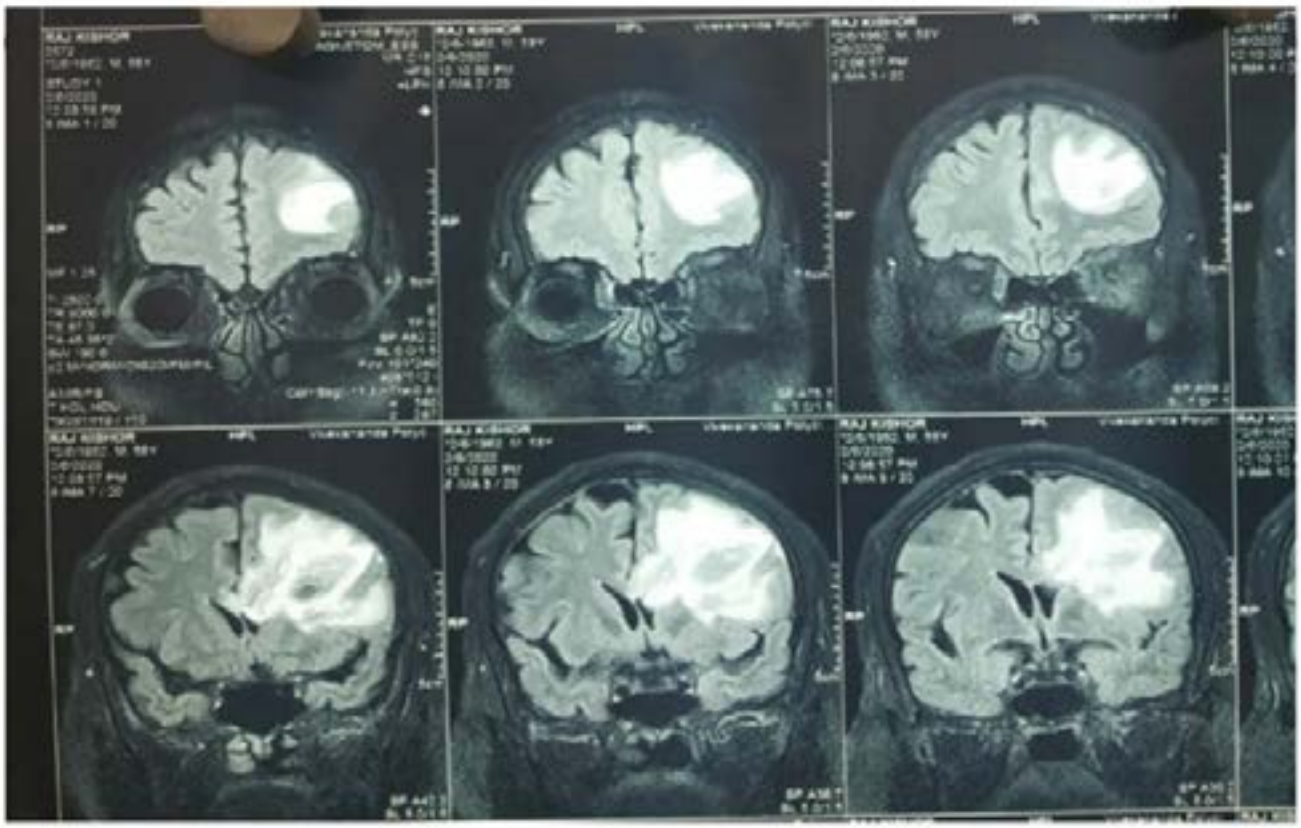

Figure 1: MRI of the brain showing acute hematoma in the left frontal lobe extending medially up to the left lateral ventricle leading to moderate surrounding edema and midline shift to the right side.

oral hypoglycemic agents, antihypertensives and thyroxine supplements were continued.

Considering non-response to the above medications, the dose of valproate was escalated to 1500mg/day, and later quetiapine was added up to 100mg/day due to persistent insomnia. With this treatment, the patient had improved significantly over six weeks. In the follow up the dose of benzodiazepine was tapered off. The patient reached the premorbid level of functioning over four months. Subsequently, the dose of quetiapine and valproate was also reduced due to an increased report of sedation.

Discussion

The "typical" patient of post-stroke mania is described as a male with no personal or family history of mood disorders, with at least one vascular risk factor developing a manic or hypomanic episode less than 2 years after a right hemisphere stroke.[7] In the reported case, the patient developed depression following recovery from a left frontal lobe stroke, followed by mania after 6 months.

A similar pattern was reported by Liu *et al.*[8] and Starkstein *et al.*[11] However, Melo *et al.* reported a reverse pattern, in which mania occurred first, 6 months following a stroke, followed by depressive symptoms developing after 1 year.[12]

Our case of a 58 years old diabetic and hypertensive male fits into the criteria largely but not completely. The patient developed mania 6 months after a left frontal lobe lesion not associated with any significant personal or familial history of mood disorders. Thus, the causal relationship between the patient's bipolar disorder and stroke is based on clinical evidence, especially in the absence of a family, personal history, or any other precipitating factor. Multiple studies have reported the occurrence of manic and bipolar episodes following vascular lesions in the absence of significant family or personal history.[13] However, Starksein *et al.* in their case series of 12 post-stroke mania cases, reported a case of recurrent episodes of depression and mania

1-month following a left frontal lobe stroke in a 25 years old male with a family history of psychiatric disorders.[11]

Several studies indicate that manic symptoms occur after a 'right' frontal, temporal, thalamic, or basal ganglia lesion. The detail about the higher prevalence of poststroke mania after right hemispheric lesions is based on the cerebral lateralization of emotion hypothesis. It says that the left prefrontal cortex is associated with positive emotions such as happiness and excitement, and the right prefrontal cortex is associated with negative emotions.[14] Consistent with this hypothesis, right-brain damage can manifest as a lack of negative emotions, with a predominance of positive emotions, and in extreme cases may encourage the development of manic episodes.[15] Also, Straksein *et al*. documented that patients with right cortical lesions developed unipolar mania and those with right subcortical lesions developed bipolar disorder.[11]

However, this does not match our case in which the left hemisphere is involved, and there is no subcortical lesion. Similar to our case, there are very few cases of post-stroke mania in people with left hemisphere involvement in literature. Notably, Liu *et al*.[8] reported a case of post-stroke bipolar disorder occurring after a left posterior temporal lobe lesion, and Turecki G. *et al*. reported a case of bipolar disorder following a left basal ganglia stroke.[16] An explanation for the occurrence of manic episodes after left-sided lesions was given by Sturm *et al*.[17] According to them, left-hemispheric lesions involving specific disruption of emotional regulation systems can cause disability of the left hemisphere to suppress the positive emotions leading to manic episodes. Additionally, in a case series of 9 patients with bipolar disorder following stroke, Berthier ML. *et al*. found that the average duration of manic swing was 8.6 ± 7 months (range 2–24) following the onset of stroke.[13] The average is slightly more than that reported in our patient who developed manic switch 6 months following the stroke.

Moreover, manic episode in patients with diagnosed bipolar disorder is reported to occur due to multiple stressors such as circadian rhythm disturbances, noncompliance to drugs, expressed emotion of caregivers, and personal achievement.[18] However, in our patient, no such factors were identified before the appearance of mania.

Conclusion

Post-stroke bipolar disorder, although a rare sequel of stroke, significantly affects health-related quality of life and is usually associated with poor outcomes in form of mortality or permanent disability in stroke survivors. Existing evidence reveals our limited knowledge of the diagnosis and management of post-stroke psychotic disorders. However, detailed history and mental status examination are crucial in diagnosing such patients. Further research is needed to better understand the correlation between bipolar disorder and vascular brain lesions and identify preventable progression and treatment measures.

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