

CASE REPORT

POST-STROKE MANIA – A CASE REPORT

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Abstract

Objective: Psychiatric disorders following cerebrovascular accidents are common. Post-stroke depression is the most common of these disorders, and post-stroke mania has been reported on rare occasions. **Methods:** We report a case of a 65 year-old elderly male who developed mania secondary to a left sided cerebral infarction in the territory of the middle cerebral artery. **Discussion:** It has been theorized that lesions in the cerebral hemisphere and limbic structures may produce symptoms suggestive of mania. Emotional and behavioral disorders after stroke negatively impact rehabilitation, cognition, and long-term recovery. **Conclusion:** Post-stroke mania is a rare medical condition that may occur after neurological deficits. *ASEAN Journal of Psychiatry, Vol. 15 (2): July – December 2014: 209-112.*

Keywords: Cerebral Hemisphere, Infarction, Mania, Stroke

Introduction

Mania is characterized by affective disturbances, flight of ideas, grandiose ideation, lack of insight and behavioral disturbances characterized by over activity and social disinhibition [1]. In 1978, Krauthammer and Klerman [2] introduced the concept of secondary mania for mania caused by neurological, metabolic or toxic disorder. Mania can be a rare consequence of stroke, as there are few systematic studies of mania in acute stroke [3]. According to previous case reports, post-stroke mania has been related to predisposing genetic factor, subcortical brain atrophy, and damage to the right corticolimbic pathways [4]. Mania seems to be more frequent after right-sided lesions, but there are also reports of mania following left sided lesions [5]. The understanding of the disorder can be helped if strong data base is built as a result of reports and studies; hence we are reporting the case which highlights a patient who developed mania secondary to stroke.

Case Report

Mr. R a 65 year-old Hindu, married, male born to the lower socioeconomic class, presented to his general physician with an acute onset of weakness and numbness of the right side of the body. He was previously healthy until 6 months ago when he started to experience exertional dyspnea and chest pain. He was given symptomatic treatment by his general physician. On the second day, there were changes in his behavior. Patient became talkative with increased goal directed activities and started making elaborate plans such as opening a garment factory, buying new houses, cars since he believed he was very rich. His religiosity increased considerably. There was a decreased need for sleep. The patient reported to the outpatient psychiatry services of the Institute of Medical Sciences, Banaras Hindu University on the 5th day and subsequently he was admitted in the ward. Patient had past medical history of type II diabetes and hypertension. There was no significant family history of medical illness. The cognitive functions were not impaired.

On examination, he was found to be alert, conscious and oriented to time, place and person. His blood pressure was 160/100 and his pulse rate was 90 beats per minute. There were no audible murmurs. His lungs were clear. His abdominal examination was normal. Neurological examination revealed a full Glasgow Coma Scale score. There was no facial asymmetry or slurred speech. His cranial nerves were intact. Examination of the musculoskeletal system showed that power on the right side was reduced with increased tone. Tendon reflexes were brisk on the right side with up going plantars. Sensations were intact bilaterally. The fundoscopy was normal. Mental state examination revealed features of mania such as volubility, elation, grandiose

delusions, and decreased need for sleep. Computerized tomography of the brain was done immediately and it reported a left middle cerebral territory infarct with resolved hemorrhagic transformation (Figure 1). Troponin T test was negative and the thyroid function was normal. His lipids were raised. Echocardiogram (ECG) was normal. Patient was managed by the neurologist and psychiatrist. He was prescribed anti platelet agents and antihypertensive treatment. For the psychiatric symptoms he was started on a combination of quetiapine, 100 mg and 10 mg of zolpidem on a need basis. Patient started improving gradually and was discharged one week later.

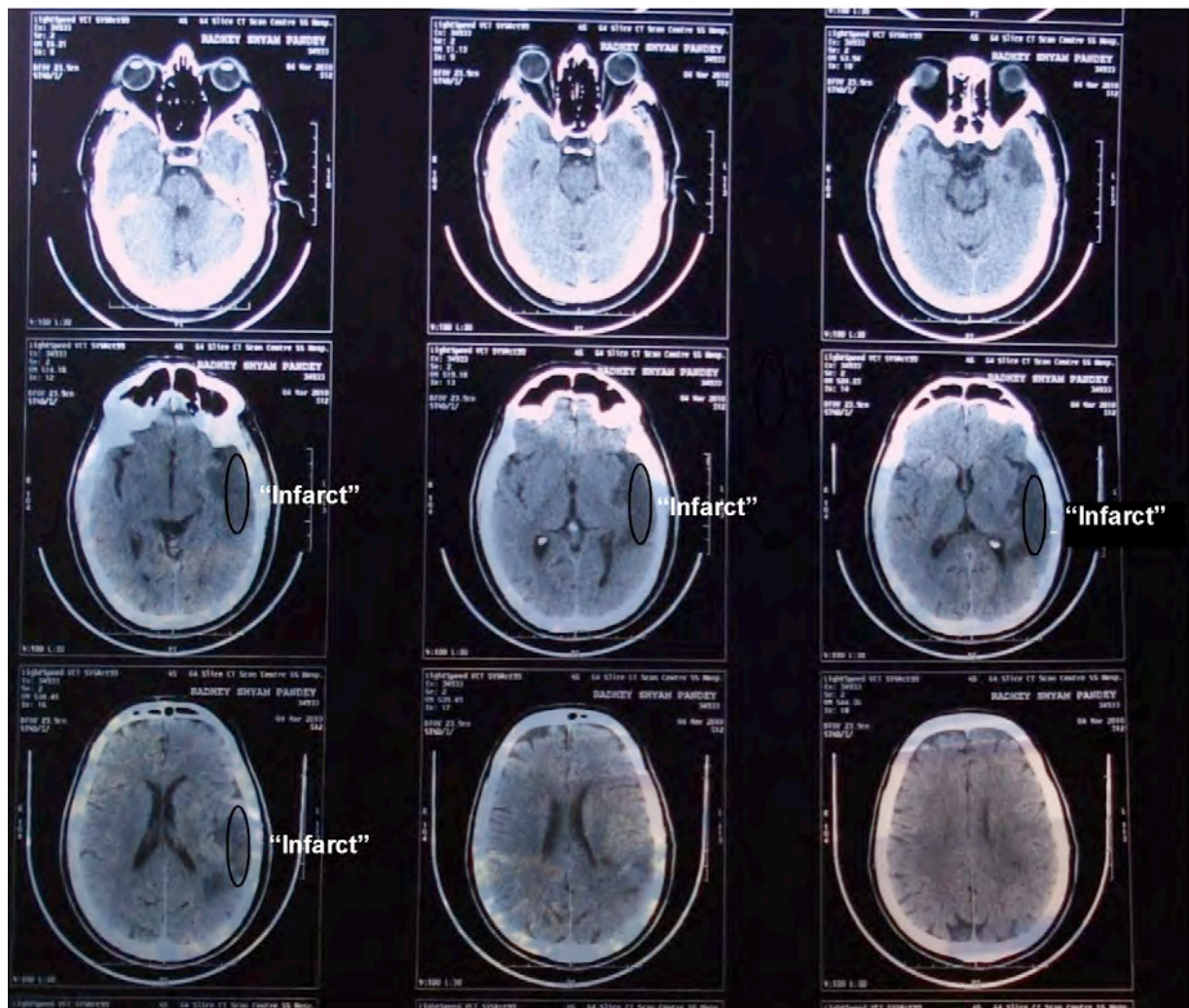


Figure 1. Computerized tomography Scan (Brain) showing infarct in the left hemisphere

Discussion

Our patient was male, without a personal/family history of psychiatric disorder, with at least one vascular risk factor, without subcortical atrophy and with a left cerebral infarct. Our results support previous research which showed a significant relationship between post-stroke mania and left hemispheric lesions causing a dysfunction in the ventral limbic circuit that involves the left orbitofrontal and basotemporal cortices, dorsomedial thalamic nucleus and head of the caudate nucleus and it is a central circuit in mood regulation and social behavior [4]. Secondary mania is often misdiagnosed as delirium in elderly patients. The course of organic mania is not clear and its prevalence and incidence are not known [6]. The temporal relationship between stroke and mania ranged from immediately after stroke to up to 2 years thereafter [7]. Patients with organic mania may have some cognitive dysfunction in contrast to patients with primary mania. The clinical profile of post-stroke mania is very similar to primary mania, characterized mainly by elevated mood/euphoria, pressured speech, flight of ideas, grandiosity and insomnia [4,5]. Establishing a causal relationship between stroke and mania has also been based on other factors than left sided lesions. The lack of a previous personal or family psychiatric affective disorder, the presence of vascular risk factors and a temporal relationship between the vascular event and the mood change in the absence of other potential precipitants of mania reinforce this etiological relationship [8]. Functional bipolar disorder usually manifests in a chronic and recurring pattern but in secondary mania, episodes may be acute in nature [8]. The likelihood that mania is secondary is greater when there is no prior personal or family history of bipolar disorder, when cognitive dysfunction or focal neurological signs are present, or when affective symptoms fail to respond to treatment [9]. Secondary mania has been attributed to various conditions, including drug use, CNS trauma, neoplasms, vascular and degenerative diseases, epilepsy, infections and metabolic conditions [10]. Post-stroke mania should be considered in any manic patient who presents concomitant neurological focal deficits and is older than expected for the onset of primary mania.

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