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NAVIGATING SECONDARY MANIA AFTER STROKE IN A 60 YEAR OLD

Dr. ABHINIT KUMAR
Professor, Department of Psychiatry, SMS&R, Sharda University.

Dr. KUNAL KUMAR
Head of Department of Psychiatry & Professor, SMS&R, Sharda University.

Dr. NIKHIL NAYAR
Assistant Professor, Department of Psychiatry, SMS&R, Sharda University.

Dr. SAUMYA MITTAL
Consultant Neurology, Sharda Hospital.

Dr. SIDDHARTH BHARGAVA
Post Graduate, Residents in Department of Psychiatry, SMS&R, Sharda University.

Dr. PANKAJ SHAH
Post Graduate, Residents in Department of Psychiatry, SMS&R, Sharda University.

Dr. PARIKA KOCHHAR *
Department of Psychiatry, Sharda Hospital, School of Medical Sciences and Research, Sharda University, Greater Noida, Uttar Pradesh, India. *Corresponding Author E-Mail: parikakochhar@gmail.com

Abstract

Mania is characterised by affective disturbances, elevated or irritable mood, increased rate or amount of speech, flightof ideas, grandiose ideation, lack of insight, behavioural disturbances characterised by over activity and social disinhibition. In 1978, Krauthammer and Klerman introduced the concept of secondary mania where mania is caused byneurological, metabolic or toxic disorders. Mania seems to be more frequent after right-sided lesions, but there are also reports of mania following left lesions. Here is a case of 60 year old male from Meerut, retired army professional, married who came with the chief complaints of aggressive, violent behaviour, increased talk, decreased sleep and increased activity since past 2 months, insidious in onset and continuously progressive in nature. On MSE he was unkempt, untidy, overfamiliar, irritable affect, increased volume of speech, grandiose delusion with impaired judgement and insight grade 1 showing past h/o hypertension, noncompliance with medication, h/o stroke 2 months back after which he had these symptoms. On MRI right sided old frontal lobe infarct present. Patient was admitted and started on Olanzapine and Quetiapine and showed improvement inbehavioural symptoms. Hospital stay was 1.5 weeks after which he was discharged on the same medication and is currently maintaining well.

INTRODUCTION

Mania is characterised by affective disturbances, elevated or irritable mood, increased rate or amount of speech, flightof ideas, grandiose ideation, lack of insight, behavioural disturbances, over activity and social disinhibition [1–5]. In 1978, Krauthammer and Klerman introduced the concept of secondary mania where mania is caused by neurological, metabolic or toxic disorders [6].

Mania is a rare consequence of stroke [4, 7–9], but there are only a few systematic studies of mania in acute stroke [10]. According to previous case reports, post-stroke mania has

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been related to: (1) predisposing genetic factor, (2) subcortical brain atrophy, and (3) damage to the right corticolimbic pathways [8–16]. Mania seems to be more frequent after right-sided lesions, but there are also reports of mania following left lesions [7,8,17]. The cerebral components of secondary mania and disinhibition syndromes are very similar and it is proposed that disinhibition syndromes, secondary hypomania and secondary mania with and without psychotic symptoms are simply a continuum of severity of mood disorder and secondary mania with psychotic symptoms may be an extreme form.

For the neurophysician, geriatrician, and the psychiatrist there is much to be attained by simplifying the issues and accepting the view that secondary mania is a discrete entity. This broadly includes elevated mood, flight of ideas, rapid pressured speech, impaired judgment, and decreased sleep amongst others. Satzer and colleagues stress that a high index of suspicion for lesional mania is necessary, and that clinicians should consider this diagnosis in cases with the following characteristics: focal or soft neurological signs; atypical manic features (visual or olfactory hallucinations, clouding of consciousness, disorientation, or memory impairment); initial presentation at an older age (≥40 years); uncommon illness course (single manic episode, unremitting or refractory mania). It was found a typical patient to be male, without a personal/family history of psychiatric disorder, with at least one vascular risk factor, without subcortical atrophy and with a right cerebral infarct the temporal relationship between stroke and mania ranged from immediately after stroke to up to 2 years thereafter [14]. Antipsychotics were used in cases of severe mania with psychotic symptoms, and throughout all these years atypical antipsychotics have been preferred because they have comparatively minor side effects.

Case:

Here is a case of a 60-year-old male from Meerut, retired army professional, married who was brought to the Emergency Room by his sons with the chief complaints of aggressive and violent behaviour, irritability, increased talk, decreased sleep, abusive language and increased activity since past 2 months which was sudden in onset and had been continuous in nature. The patient was unable to sit in one place and was agitated throughout the interview. The informants gave a history of him throwing and breaking things in the house and getting anger spells over trivial matters. He was also not taking care of himself like he used to and perceived himself as an extremely strong man who could do anything he wanted to. There is also a past history of hypertension and non compliance with medication. There was a past history of stroke which happened 2 months back, few days after which he began showing these behavioural symptoms.

Mental Status Examination:

Patient was unkempt, untidy, overfamiliar, good eye to eye contact, restless, had an irritable affect, with lability of mood, increased volume and rate of speech, flight of ideas, grandiose delusion with impaired judgement and insight grade 1.

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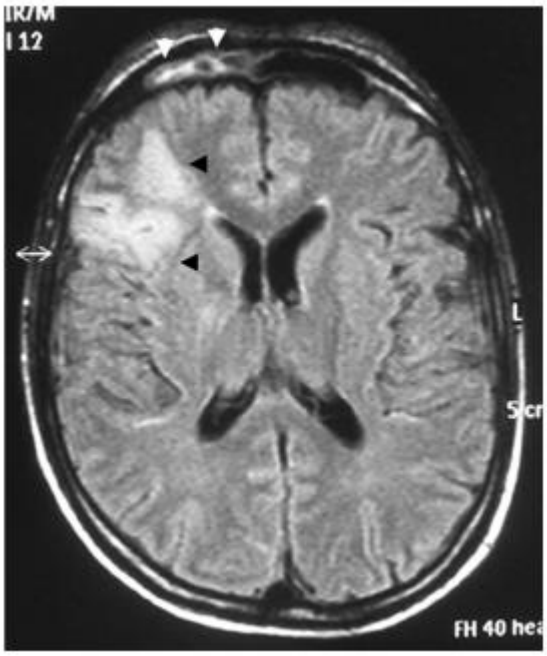
Investigations:

Patient was sent for an MRI which concluded the presence of a right sided old frontal lobe infarct. After this Neurological opinion was taken, which concluded that this is a case of Secondary Mania due to Frontal Lobe Infarct.



Patient was admitted in the Psychiatry Ward and underwent routine investigations including CBC, LFT, KFT, RBS, Tridot, Lipid Profile, Thyroid Profile, etc.

CONCLUSION

Patient was admitted in the Psychiatry Ward and started on Olanzapine and Quetiapine and showed improvement in behavioural symptoms within 1 week. Hospital stay was 1.5 weeks after which he was discharged on the same medication and is currently maintaining well. Manic symptoms were concomitant with the onset of the event. This case highlights the importance of ruling out organic causes in patients who present with behavioural changes and have psychiatric symptoms. A high index of suspicion is required in elderly people with comorbidities for early detection and prompt intervention of such disorders.

In conclusion, 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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